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Dr. A.L. Meenakshi Sundaram

I'm very happy to meet you all again through the second issue of JIDAT. I can't believe we are halfway through 2018. Once again I congratulate Dr. Musthafa for the second issue of JIDAT. I request all the members to make use of this journal for their publications.

First of all I want to extend my thanks to all the members for their cooperation, dedication and support.

It is a great pleasure to witness growth in the membership and all the members surely put a lot of work helping us grow.

Speaking of events, in the previous months we had many events and conferences of highly valuable and educational and there was an incredible amount of sharing and learning.

These events help to rejuvenate and revamp the association.

I want to call your attention for the forthcoming events:

- -6th Tamil Nadu State IDA Sports Meet on August 18 and 19, 2018 hosted by Marthandam Branch .
 - -33rd Annual Dental Conference on November 30, December 1 and 2, 2018.
 - -First ever Student Conference at Salem on November 9 and 10, 2018.

Before closing, I like to remind our special goals,

- -reach membership growth of 10000
- -reach 5 lakhs for Care and Concern.

Our members are strong, passionate and dedicated to our mission and look forward to continuing to make a difference together in 2018.

I'm humbled and honored to be able to serve you as the President of IDA, Tamil Nadu.

Thank you

Yours sincerely,

Dr. A.L. Meenakshi Sundaram
Hon-State President
DA Tamilnadu State Prenah



Dr. K.P. Senthamaraikannan

Dear Members,

Warm regards from Indian Dental Association Tamilnadu State branch.

It's great that our Editor has coming out with 2nd issue of JIDAT on quick successful time. I as state secretary appreciate for his dedicated work towards JIDAT.

JIDAT is dedicated to dental health and providing results of studies based on research into various facts of dentistry. It's extremely happy to see that dental professionals are showing greater involvement in JIDAT. There is nothing greater than generation of enthusiastic response among the reader that publishers should come out with qualitative movements in the publication consistent with the global changes in the dental science.

JIDAT is the forefront of dental research that is of immense value for students, academician and private practitioner.

My best wishes.

Thanking you,

Yours sincerely,

Dr. K.P. Senthamarai Kannan

K.P. Smiller

Hon-State Secretary IDA-Tamilnadu State Branch



Dr. H. Mohammed Musthafa

Warm wishes,

Happy to meet you all through this revamped and refurbished JIDAT.

My humble request to all the members is to equip yourself for the upcoming Clinical Establishment Act.

"Nothing is Stationary, except Changes"

There is no goback for the CEAct, as of now.

We, as law abiding citizens and responsible sculptors of this society, need to follow the rules enforced by the Government, with regard to CEAct.

Rules in C E Act needs to be amended further, for sure, and the Office of IDA Tamil nadu State is striving at its best, by all means for the same.

My suggestion to all the local office bearers is to acquaint yourself with the local government authorities for a better and placid sailing in the squall of C E Act.

Same way, My kind request to all the members is to make use of the Journal to evince your dexterity in the field of Dentistry which may familiarise you amongst the fellow Dental Fraternity as well as be helpful to fellow dentists' to pursue your footsteps for their successful Practice.

We at the Office of Editor of JIDAT take humongous efforts to bring out the issue on time with all your contributions.

United We Stand, Divided We Fall.

Jai IDA...!!!

Dr.H.Mohammed Musthafa,

& Dhamed Down

Editor in Chief,

JIDAT.



INFORMING, SERVING, EDUCATING!.

Dr. G. P. SurendranDental Practitioner, Coimbatore.

Indian Dental Association (IDA) is an authoritative, independent and recognised voice of dental professional in India. We are asking you to join us, and here is why should you become a member?

"Working together works". From the minute you join, you can be confident that IDA is looking after your best interests. The association works towards acknowledging the members needs and requirements, appreciating our skills, awarding our talent and helping us achieve greater heights professionally.

IDA enhances the reputation of our profession and accelerates our practise by IDA Membership certification.

Empowers Dentists- IDA ensures that the voice of dentists and dentistry is heard in public and government forums, with the strength of 75000+ members in protecting our rights.

Networking- IDA teams up with trade, media and related organisations to gain maximum benefits, which is profitable for fellow members.

Education- Being in IDA is a way of staying current with the profession. Through CDEs, Fellowship programmes and educational training centre (ETC) and CE online, IDA conducts many lectures, hands-on courses and certification programmes which gives great scope for up gradation and access to research & training. State and national level conferences are held regularly for cohesive learning.

Service to the Community- If you believe in the saying," Service to Mankind is service to God", you have great opportunities as an IDA member to serve the society through various projects created by the team.

- Corporate and School dental health screening programs,
- TII: Tobacco Intervention Initiative

- OCF: Oral Cancer Foundation
- CDF: Child Dental Foundation
- EDC: Emergency Dental Centres
- · HSF: Healing Smile Foundation, and
- IDRF: Indian Dental Research Foundation.

Legal Disputes- In case of medico-legal cases, IDA protects you, defends you and fights for you- if you are right. The power of information about law is the key to successful business.

Care and Concern Scheme- IDA has started the scheme for the families of deceased doctors wherein the family members will be provided with financial assistance. IDA is working out an exhaustive scheme for the benefit of family's at their loss.

Newsletter- To keep you abreast, IDA publishes newsletters with the information and technology trending in our field.

Channelize your inner leader- If you are a person who can take up responsibility, if you have administrative and leadership capabilities, you are most welcome to be a part of executive committee which will pave way further to the hierarchy of our association.

IDA Membership brings a whole wealth of benefits and any member of the IDA would unanimously agree that our organisation is all about improving oral health, quality of life and achieving `optimal oral health for all'. One would also say, we aim to represent the dental profession and support members in the provision of comprehensive and quality oral health care.

IDA can help you professionally, socially and of course as a trade union, wherever appropriate.

It is time to become a part of this prestigious association to create hope, possibilities and partnership for all individuals.

Cheers!



EVALUATION OF DIAGNOSTIC ACCURACY OF KI – 67 (IMMUNOCYTOCHEMISTRY) AND AGNOR IN DETECTING EARLY CHANGES IN SMOKERS AND TOBACCO CHEWERS.

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ABSTRACT

The study was conducted to validate the accuracy of Ki - 67 (ICC marker) in identifying the proliferative and malignant changes occurring in the exfoliated buccal cells while comparing it with the standard AgNOR staining technique. The study group comprised of normal, smokers and tobacco chewers with a total of 30 subjects. The solutions for AqNOR staining was prepared as prescribed by Bukhari et al (2007) Ki 67 was used for ICC procedure. The AgNOR number where more in smokers than in chewers. The AqNOR counts in chewers were also comparatively higher when compared to normal and close to smokers. The Immunocytochemistry (ICC) study, showed positive expression of Ki 67 in the nucleus of exfoliated cells which was significantly higher in chewers than smokers and normal.We conclude that even ICC (Ki 67) is even more sensitive than AgNOR and it could also be used as an adjunct to histopathological investigation. As literature states that proliferation is observable with AgNOR, this holds true for ICC technique with Ki 67also.

Keywords: AgNOR, Immunocytochemistry, Ki 67, Exfoliative cytology, Buccal smears.

Introduction

Cancer incidence in humans has gradually increased over the last century. Despite advances in diagnostic techniques and treatment protocol the disease is not under complete control. The World Health Organization (WHO) reported oral cancer as having one of the highest mortality ratios amongst all malignancies (Parkin et al., 2000). It ranks 12th among all cancers (Jemal et al., 2002). It is important to diagnose oral cancer in its early stages, since the management of small and localized tumors involves less morbidity and mortality than more advancedstage disease, where treatment must be more aggressive.

Exfoliative cytology is the examination of exfoliated cells from the epithelial surface generally from oral mucosa. Cells in the deeper regions adhere to each other strongly in normal physiologic condition but in case of malignancy they tend to loose these adhesion and are exfoliated alone or along the

cells of superficial layer.1Nucleus of a cell has an valuable role in the proliferation, regulation and protein synthesis.2 The nucleolar organizer regions (NORs) are the chromosomal loops of DNA involved in ribosomal synthesis. Some nucleolar proteins which are associated with these NORs stain with silver particles which are called AgNOR proteins. They correlate very well with cell proliferative activity and protein synthesis.3

Any type of screening test which works on biomarkers are amenable to automation, there by resulting cost savings and potential for applications in the developing world.4So the study aimed at performing AgNOR staining procedure from buccal smears collected from normal, smokers and tobacco chewer subjects and further validating it with Ki 67, a nuclear proliferative marker using Immunocytochemistry method.

Materials & Methods:

A total of 10 subjects were taken in each category of normal, smokers and tobacco chewers with an age range of 25 – 50 years, comprising a total of 30 subjects. The subjects were collected from those attending the Oral Pathology out patient department for routine dental check up and treatment. The smokers had the habit of smoking only using more than 5 cigarettes per day, the tobacco chewers had the habit of using smokeless form of tobacco for more than 5 years with a frequency of consuming it more than 4 times a day.

Patients who were included in the study subjects didn't had any oral lesions. The smears were taken from normal buccal mucosa of the subjects. Patients were asked to rinse with water and the area to be smeared was cleaned with gauze to remove excessive saliva and surface debris. The smears were collected by scraping wooden spatula along the buccal mucosa and smear was applied on the 2 glass slides and fixing one slide in alcohol for AgNOR staining according to Bukhari et al (2007) and other acetone fixing for Immunocytochemistry staining for half an hour.

Preparation of Working solutions:-3

Solution A – The solution was prepared by dissolving 500 mg gelatin powder in 25ml deionized water at 37 c and then 250 Ml formic acid is added. Continuous shaking of the glass ware for about 10 min at 37 c was sufficient to dissolve the gelatin and a clear solution is obtained.

Solution B – It consist of silver nitrate and deionized water. 50% w/v concentrated solution of silver nitrate in deionized water

The final working solution was prepared by

mixing one part of solution A with two parts of solution B and filtered using a filter paper into glass bottle and used immediately. Solution was prepared as an when required to avoid wastage and considering cost factor.

The slides are covered with the silver solution and kept in dark place for 20 – 30 minutes and dehydrate dwith alcohol (50%,70%,80%,96%,100%) for 5 min each and clarified with xylene for 5 min.5 The slides were dried in dark place and coverslip mounted with DPX.

For Immunocytochemistry procedure :-

The acetone fixed slides are subject to immuncytological procedures. The slides first hydrated with decreasing grades of alcohol (100%70%,50%) and water. Followed by 2 times PBS(Phosphate buffered saline) wash for 5 min each. Next step, is peroxide block for 5 min, protein block for 5 min, Ki 67 (primary block) for ½ hour followed by post primary for ½ hour, polymer link for 15 min and each step should be followed by PBS wash for 5 min x 2 times. DAB chromogen for 2 -3 min followed by running tap water wash and hematoxylin for 5 min, washing in water.

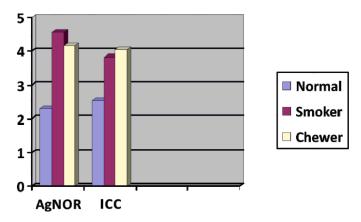
The slides are subjected to dehydration of alcohols (50%,70%,100%) for 5 minutes each and cleared with xylene for 5 minutes. Slides are dried and mount coverslip with DPX.

Results:

AgNOR dots were seen within the nuclei and viewed clearly as black dots. In Immunocytochemsitry slides, the nuclei showed brown dots which varies in intensity.

Subjects	AgNOR Staining (Mean)	Subjects	ICC Staining (Mean)
Normal	2.29	Normal	2.54
Smoker	4.58	Smoker	3.83
Chewer	4.17	Chewer	4.05

Table 1: Mean value of AgNOR Staining & ICC Staining.



Graph 1: Comparative mean between AgNOR and ICC staining

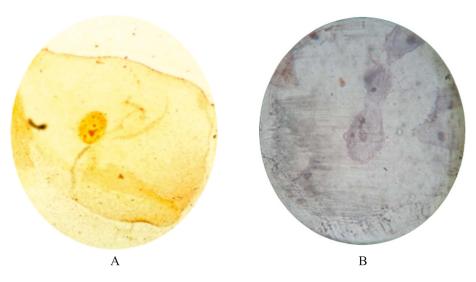


Fig: 1- Cytological smear stained with (A): AgNOR & (B): ICC (Ki 67)

Discussion:

Cancer especially oral squamous cell carcinoma is quite common in India due to adverse use of tobacco in smoking and smokeless form.6 Oral squamous cell carcinoma has a poor prognosis inspite of advances in therapy. Diagnosis at early stage and treating them are the main tool in improving patient survival rate. Generally scalpel biopsy is used for diagnosis which is invasive and morbidity, so they are employed in severe suspected lesions and not in all conditions.7

To control the rapidly developing situation technquies which are less expensive, non invasive, and well accepted by the patient needs to be developed and those which can be repeated frequently. It is an easy procedure that can be carried out at outdoor patient department to diagnose malignancy at early stage.6Exfoliative cytology is not a new science. In 1920, aspiration and exfoliative cytology were introduced. Johannes Muller (1801 - 1858), a pathologist in Berlin was first to show cancer cells in microscope on scrapings from the cut surface of surgically excised tumors.1The silver staining procedure which is used for identification of NORs has been frequently utilized in formalin fixed, paraffin embedded specimens but in this study we have used exfoiliative cytology. 9

Jahanshah Salehinejada et al has showed in their study that, in cytologic smears the analysis of AgNORs is more accurate as whole nucleus can be assessed as in tissue sections and has used AgNOR technique successfully in oral smears1. This present study carried out AgNOR staining in cytologic smear also successfully carried out ICC staining in oral cytologic smears.

The Ki-67 antibody was first developed by Gerdes and coworkers who demonstrated the antigen to be present in G1, S, G2 and M phases of continuously cycling cells, but absent in G0 cells. To best of our knowledge there are no previous publication comparing AgNOR and ICC (Ki 67) marker but was suggested by Jahanshah Salehinejada et al (2007). 9In deep invasive fronts of tumours, the expression of Ki 67 is higher than those at the centre or surface of mucosal cancers. It shows that actively proliferating cells are more in number at deep tumour margin. The cytological assessment has certain difficulties like obscuring blood products, necrotic debris and contamination by bacterial and other components.9

In this study we have considered 3 or more black dots in the nucleus to have more cellular and proliferative activity and followed the same criteria for ICC counting. The AgNOR number where more in smokers than in chewers as not ususally expected because the silver granules may be counted as silver dots and were considerably high than normal. The AgNOR counts in chewers were also comparatively higher when compared to normal and close to smokers.

Regarding ICC study, it showed positive expression of Ki 67 in the nucleus of exfoliated cells. It showed a significantly higher value in chewers than smokers and normal. This shows that this procedure is more sensitive in detecting the nuclear alterations and miscounting of silver granules as silver dots could be eliminated.

Conclusion

Based on this study, we conclude that even ICC (Ki 67) is even more sensitive than AgNOR

and it could also be used as an adjunct to Histopathological investigation. One of the main advantage in employing this technique is that counting will be accurate as we will be avoiding counting silver granules as silver dots in nucleus and it will detect even mild alterations in nucleus.

As literature states that proliferation is observable with AgNOR, this holds true for ICC technique with Ki 67also.

Further studies with larger population and certain other criteria for assessment could enable us to validate and improve the technique, so that it will help in formulating new diagnostic methodologies which are specific and can be performed at minimum laboratory set up.

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'COMPARISON OF TMA, STAINLESS STEEL AND TIMOLIUM FOR FRICTION, LOAD DEFLECTION AND SURFACE CHARACTERISTIC"

Dr. K. Abhirami MDS.,

Associate Professor, PSG Institute of Medical Science & Research, Coimbatore.

Abstract:

OBJECTIVE

The present study aims at characterizing and comparing the mechanical property and surface characteristic of two arch wire alloys in orthodontics, stainless steel and TMA with newly introduced Timolium.

METHOD

An INSTRON 2366 testing machine was used for frictional characteristic and three points bend testing. Scanning electron microscope was used for surface evaluation and x-ray fluorescence for elemental analysis of Timolium wire specimen.

RESULT

Stainless steel was the strongest archwire alloy, and less friction at the archwire-bracket interface. TMA wires exhibited better load deflection characteristics with less stiffness than the other two wires. The surface of TMA appeared rough and exhibited very high values for friction at the archwire-bracket interface. Timolium appeared to be an alpha-beta titanium alloy composed of titanium, aluminum, and vanadium and intermediate in nature for all the parameters evaluated.

CONCLUSION

Timolium with its smooth surface, reduced friction and better strength can be considered as an introductive breakthrough in clinical orthodontic practice.

KEYWORDS

Friction, load deflection, scanning electron microscope, stainless steel, TMA, Timolium.

Introduction:

Gold alloy was one of the earliest orthodontic wire used .The gold wires have decreased usage in orthodontics because of their low yield strength and increasing costs. In 1940s austenitic stainless steel with adequate spring back, good formability, and moderate cost began to displace gold as primary alloy for orthodontic wires. Elgiloy, a chrome cobalt nickel alloy with excellent corrosion resistance was used. Nickel titanium alloy was introduced in orthodontics several years ago and find its application where deflection and low forces are required. These wires are not amenable to joining operations. With the introduction of b-titanium alloy distinctive features of good spring back, low force delivery, good formability, weld ability, high corrosion resistance, and excellent bio compatibility were available for orthodontist. The disadvantages are surface roughness and high friction. In 1995 a new TMA featuring dramatically reduced friction for a superior sliding mechanics was introduced by Burstone, Timolium, a patented vanadium based high performance aerospace

alloy, has been designed to produce a much smoother surface texture than other titanium molybdenum wires(TMA). Timolium as claimed by the manufactures has almost the same frictional resistance and half the stiffness of stainless steel making it ideal choice for finishing, aligning as well as leveling and torqueing through all phases of treatment.

MATERIALS AND METHOD

The following materials were used in the study

- 0.016"x 0.022" stainless steel (ormco corporation Giendora CA) – 15 numbers.
- 2. 0.016"x0.022" TMA (ormco corporation Glendora CA) 15 numbers.
- 3. 0.016"x0.022" Timolium (TP labs, Indiana polis IN) 15 numbers.
- 0.018" Edgewise canine brackets (American ortho.)
 -45 numbers.
- 5. 0.010" ligature wires (ortho organizer).
- 6. Cold cure acrylic.
- 7. Stainless poles 5mm in diameter.
- 8. Instron machine with model number 2366.

METHOD

FRICTION ANALYSIS

In this study Tidy's (30) frictional test designs were used to stimulate canine retraction. The force acting on the surface of the tooth was simulated by single equivalent force acting at the center of resistance of the root. [27] . Four 0.018" slot edge wise bracket were fixed, at 8mm intervals with a 16mm space for a movable canine bracket at the center. A power arm of 10mm length from bracket slot was fixed at the base of each canine bracket. This distance was chosen according to Burstone finding relating to the location of center of resistance of canine. (3)

The wires stainless steel, titanium molibdinum and Timolium, were then evaluated for friction in the 0.018" slot edge wise brackets.

This jig assembly was mounted on to the lower iaw of instron machine. The canine bracket was then ligated to the test wires. The stainless steel ligatures 0.010" were initially fully tightened and then unwound by three turns; loose ligation was checked by rocking the brackets to confirm that there is play between both the spans of the bracket and the archwire. (21) the bracket was then moved over the archwire with the help of a 0.016" stainless steel wire which was suspended from a special jig which was attached to the upper jaw of the instron machine which was moved in the upward direction, at cross head speed of 5mm/min. In each test they were moved not less than 2.5mm across the central space and the load cell reading were recorded on the digital display.

The differences between load cell reading and load on the power arm thus represents the frictional resistance of the given material.

LOAD DEFLECTION ANALYSIS

The three point bending test was conducted as described by miura et al. (9)

A single bracket was attached on a steel pole,

acting like a dental unit placed on a movable stage, so that the bracket span could be set at 14mm. the test wire was held in place with a ligature wire in the slot with a known quantity of force. This jig assembly was mounted on to the lower jaw of the instron machine.

A steel pole of 5mm in diameter was placed in an acrylic block, which was mounted on to the upper jaw of the instron machine. The steel pole which was attached to the upper jaw of instron machine was moved down so that mid portion of the wire segment was deflected to 2mm at the speed of 0.1mm/min under the pressure from the metal pole. The load cell reading in digital display was noted at 0.5mm, 1mm, 1.5mm and 2mm deflection.

The above tests were divided in to three groups comprising of 15 wires each.

Group 1 - 0.016"x0.022" stainless steel wires.

Group 2 - 0.016"x0.022" TMA wires.

Group 3-0.016"x0.022" Timolium TM wires.

STATISTICAL ANALYSIS

Mean and standard deviation were estimated from the samples for each study group. The groups were subjected to one –way ANOVA to test the level of significance among the test groups. Multiple range tests by Tusky –HSD procedure was employed to identify the significant group if the p –value in the one- way ANOVA was significant.

In the present study, p<0.05 was considered as the level of significance.

SURFACE CHARASTERISTIC

In surface, characteristic study each wire was studied before and after sliding through the bracket with the help of a scanning electron microscope a 1cm specimen of each alloy wire was mounted and these wires were then scanned and viewed on the monitor at different magnification and representative micrographs (750 x) of the alloys were obtained.

RESULT

The result of the frictional resistance of the three wires which were analyzed in the study using instron testing machine are tabulated as follows (Table-1); and is graphically plotted in (chart-1)

Table-1
Frictional resistance between stainless steel,
TMA, Timonium.

Group	Number of sample	Mean ± (grams)
1	15	203.2 ± 24.2
II .	15	301.3 ± 27.5
III	15	222.5 ± 32.9

The above values were subjected to statistical analysis and tabulated as below (Table-2)

Table-2
ONE-WAY ANOVA AND MRT

	ANOVA	Multiple range test	
		Table-2	
GROUP	P-Value	ONE-WAY ANOVA AND MRT	
		Significant group at 5%level	
1		TMA VS STAINLESS STEEL	
11		TMA VS TIMOLIUM	
III	<0.001/(sig)		

- ANOVA test revealed a statistically significant difference among the three groups of the wires analyzed.
- Multiple range tests revealed a statistically significant difference between group-I and group-II and group-III.

The results of the three groups were analyzed for load deflection is tabulated as follows (Table 3); and are graphically represented in (chart 2).

Table-3

Load deflection between stainless steel, TMA and Timolium.

Group	Number of samples	Mean ± S. D (grams)
1	15	1576.3 ± 46.3
11	15	1189.3 ± 19.2
III	15	1319.6 ± 281

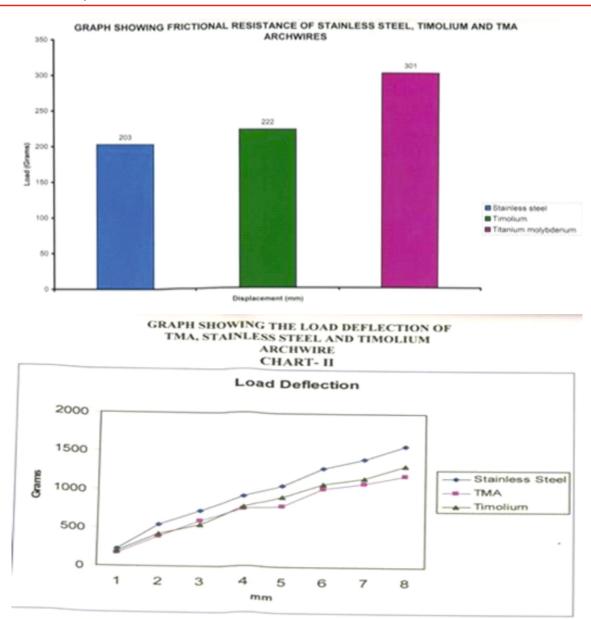
The above values were then subjected to statistical analysis and tabulated as below (Table 4)

Table-4
ONE-WAY ANOVA AND MRT

Group	ANOVA	Multiple range test
Group	P-Value	Significant group at 5%level
I		STAINLESS STEEL VS TMA
II		STAINLESS STEEL VS TIMOLIUM
III	<0.0001/(Sig)	TIMOLIUM VS TMA

- ANOVA test revealed a statistically significant difference among the three groups of wires analyzed.
- Multiple range tests revealed that there was a significant difference between Timolium and TMA wires

All the three groups of wires which were



evaluated for the surface characteristic using scanning electron microscopy both before and after sliding of wire in the bracket, reveals that in

STAINLESSSTEEL (FIGURE -1)

BEFORE SLIDING OF WIRE

(a)
20kU ×758 78nm 8808 27 25 SEI

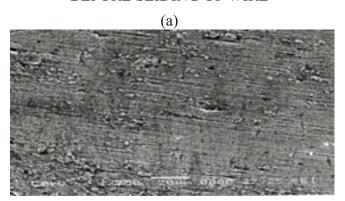
AFTER SLIDING OF WIRE

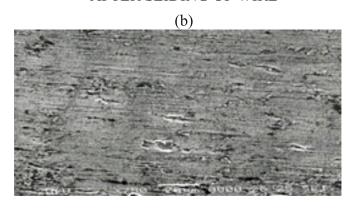
(b)

TIMOLIUM (FIGURE-2)

BEFORE SLIDING OF WIRE

AFTER SLIDING OF WIRE

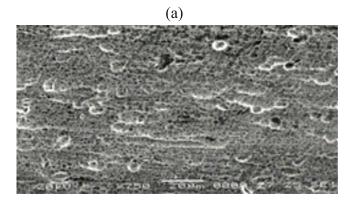


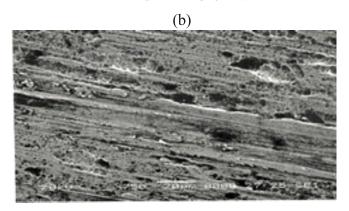


TMA (FIGURE-3)

BEFORE SLIDING OF WIRE

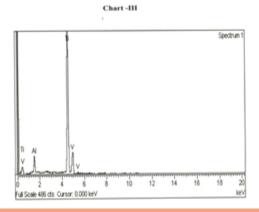
AFTER SLIDING OF WIRE





The composition of the timolium wires in (Table-5) and graphically represented in (Chart- 3.) Table – 5

Element	Weight%	Atomic%
TiK	91.70	87.97
ALK	5.67	9.65
VK	2.63	2.37
Totals	100.00	



DISCUSSION

Characterization of arch wire alloy forms an initial step towards understanding arch wire behavior in biomechanical requirement of the clinical situation from the plethora of the material available.

Frictional force has long been an important consideration in orthodontic mechanotherapy. It is a well-known fact that any force needed to retract teeth must overcome friction. (13)

Friction is a function of the relative roughness of two surfaces in contact [23].

Both static and kinetic sliding friction arises in any orthodontic situation of intended displacement, overtime, of an arch wire through a bracket (or) a bracket along an arch wire. (1)

Profit (2000) (26) reported that 50 per cent of the force necessary to initiate tooth movement required to overcome the regarding force generated between brackets, arch wires and ligatures. This implies that only 50 percent of the force applied reaches the tooth and its supporting tissues. The absolute value for the optimum force required to produce "biological tooth movement" is extremely difficult to quantity and it has been demonstrated that increasing the force increases the rate of orthodontic tooth movement up to a point (Andreasn and Quevedo,(1970)(1); and Nikolai, (1980) (8); Garner et al; (1986) (10), Beyond this point tooth movement fails thus, the concept of optimal force. (8)

Kinetic friction was measured rather than static friction because of the variable nature of the frictional process. The result shows that the frictional resistance of Timolium lies between stainless steel and TMA with range being close to that of stainless steel.

The present study clearly indicates a greater friction when TMA wires are used, with

a mean value of 301.3 (\pm) 27.5 grams. The least friction arch wire with stainless steel, with a mean value of 203.2 (\pm) 32.9 grams.

The result of the study conducted by Cash et al., (2004)(5) is similar to the present study.

The study conducted by Garner et al., (1986)(10), Dresche et al., (1989)(7), Kusy and Whitley, Kapila et al., (1990)(14), Angolkar et al., (1990),(2) is similar to the present study stating that beta titanium arch wires developed frictional forces greater than stainless steel.

Surface evaluation of an arch wire alloy is important because of its influence on working characteristic as well as working potential. (18) Scanning electron microscopic evaluation of surface characteristics revealed a smooth surface with horizontal lines for Timolium arch wires as in accordance to, pradeep babu[30] . Stainless Steel exhibited vertically oriented cracks, and TMA, a very rough surface as reported extensively in the literature [4, 3, 6].

A modified version of the three-point test by Miura et al(9) was performed to evaluate the load deflection properties, the most important parameter determining the biological nature of tooth movement.(4) The result indicated beta titanium with a mean of $1189.3~(\pm)~19.2~grams$. Stainless steel was more rigid among three arch wire alloy with very high loading value and less spring back properties with a mean of $1576.3~(\pm)~46.3~grams$. Timolium was intermediate in nature $1319.6~(\pm)~281~grams$.

According to cristiane [33], Oltjen et al., (1970)[22], vijayalakshmi RD [34] Timolium possesses comparatively low stiffness, better strength and behave as an intermediate between stainless steel and TMA.

Elemental analysis of Timolium with the help of EDS revealed the alloy, with

combination of alpha and beta phase of titanium alloy, exhibits an unusual combination of strength and surface smoothness.

CONCLUSION

In the study performed shows that in frictional studies, Timolium has almost has the same surface friction as stainless steel, unlike TMA (Titanium molybdenum) which have much higher frictional characteristics. This reduced frictional property of the Timolium helps in efficient canine retraction.

Timolium has the half the stiffness midway between TMA and stainless Steel making it ideal choice for finishing, aligning as well as leveling and torqueing throughout all phases of treatment.

Timolium bends easily in "T" boots and" loops" because it has a higher stiffness than TMA and fewer surface defects.

Timolium with its smooth surface, reduced friction and better strength can be considered as an introductive breakthrough in clinical orthodontic practice.

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DRUG INDUCED ORAL ERYTHEMA MULTIFORME: A CASE REPORT

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ABSTRACT

Erythema multiforme is an acute, self limited and sometimes recurring skin condition that is considered to be a type IV hypersensitivity reaction associated with herpes infection, medication and other various triggers. Here we report a case of Drug Induced Erythema multiforme in a 56 year old woman who developed oral lesions after taking antiepileptic medication.

Key words: oral erythema multiforme, drug reaction, oral mucosa, ulcerations.

INTRODUCTION

Erythema multiforme a self limited acute inflammatory disorder affecting skin, muous membrane or both was first identified by Bulkley and Bateman in 1817. In 1846, a first case was reported in America as "Herpes Iris." Later, in 1866 Von Hebra, described this condition under the term "erythema exsudativum multiforme". Erythema multiforme represents a spectrum of disease ranging from localized rash with minimal mucosal involvement to a more severe generalized rash with limited desquamation and involvement of mucous membranes with blister formation.1 The reaction comprises of polymorphous eruption of macules, papules, with a characteristic "target" lesions that are symmetrically distributed with a proclivity for the distal extremities. Other than EM minor and major, oral erythema multiforme (EM) is considered as a third category. The features include lip and oral ulcerations without any skin target lesions. No specific

laboratory tests are indicated to make the diagnosis of Erythema multiforme, which should be arrived at clinically. Immunofluorescence and histopathological examination may be used to confirm the diagnosis of Erythema multiforme and to rule out the differential diagnosis.

This article reports cases of drug induced oral EM highlighting its importance of differentiating this disorder.

CASE PRESENTATION:

A 56 -year-old female patient reported to our department of Oral medicine and

Radio diagnosis with a complaint of extensive oral ulcerations and hemorrhagic crusts on the lips due to which she was unable to take solid food and was on liquid diet since a week. Patient gave a history of seizures 15 days back for which she took Epitoin tablet 100 mg twice daily. Subsequently she developed multiple vesicles in the oral mucosa which eventually ruptured and transformed into extensive irregular ulceration.



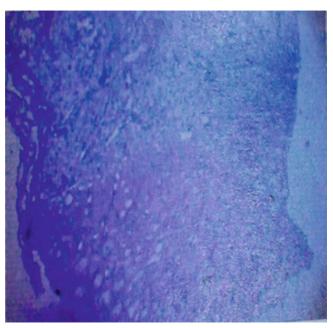
On extra oral examination, both upper and lower lips showed extensive, fissured irregular ulcerations, with blood encrustation. Bilateral submandibular lymph nodes were tender and palpable.

Intraoral examination showed multiple irregular ulcerations with yellowish base surrounded by erythematous borders on lip and palate. There was no extension into the pharynx.



The sudden onset, drug history, extensive ulcerations of the oral cavity cracking and fissuring of lips with bloody crustations lead to the provisional diagnosis of oral erythema multiforme. A incisional biopsy followed by immunofluoresence study was done after revealing no abnormality in blood investigation

Histopathological examination of the specimen showed surface squamous epithelium showing acanthosis, necrosis of deeper keratinocytes and vacuolation of basal layer. Sub epithelial layer shows edema, increased vascularity and infilteration by lymphocytes and few polymorphs. Direct immunofluorescence study shows no deposition of Ig G,IgA,IgM and fibrin.



The clinical and histopathological findings were considered diagnostic for Oral Erythema multiforme.

The patient was advised to alter the drug after consulting her physician and was treated with systemic corticosteroids, (prednisolone 20 mg BD for 3 days followed by tapering the dose for 10 days), mild analgesics, and topical application of

lignocaine gel to facilitate oral fluid intake. Patient responded to the treatment well and healing occurred within 10 days.

Review photos

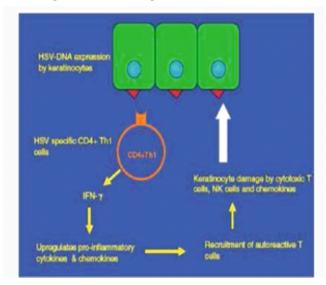




DISCUSSION

Erythema multiforme a self limited acute inflammatory disorder affecting skin, muous membrane or both was first identified Bulkley and Bateman in 1817. In 1846, a first case was reported in America as "Herpes Iris." Later, in 1866 Von Hebra, described this under the term "erythema condition exsudativum multiforme" .2 According to him the patients with this condition should have raised edematous cutaneous papules. or acrally dispersed typical target lesions. In 1968 Kenneth described an inflammatory oral disorder typial of EM but without any skin involvement.3

The disorder results from T-cell-mediated immune reaction to the triggering agent, leading to cytotoxic immunological attack on keratinocytes expressing non-self antigens, leading to subepithelial and intraepithelial vesiculation forming widespread blisters, erosions and ulcerations. In druginduced EM, the drug metabolites induce the disease. Tumor necrosis factor alpha (TNF-A) is responsible for keratinocyte apoptosis causing tissue damage.1



The sites commonly involved were cheeks, tongue and lips. Large irregular ulcers

bordered with necrotic tags are usually seen . When lips are involved bloody encrustation were seen frequently.4 Patients initially may experience burning and itching sensation at the site followed by eruption of sharply demarcated numerous macules progressing to papules with crustation occurring in the center of the lesions.1 The characteristic "target" or "iris" lesion has a regular round shape with three concentric zones: a central dusky or darker red area, a paler pink or edematous zone, and a peripheral red ring.5

The differential diagnosis of oral EM includes autoimmune vesciculobullous lesions such as bullous pemphigoid or pemphigus vulgaris and other patterns of drug reactions and ulcerative lesion like herpes6.

Keratinized mucosa are most prompt for herpetic lesions and also the ulcers are smaller having regular borders .7But in our case irregular ulcers are seen in the lining nonkeratinized mucosa which is not a feature of herpes infection.

The onset is acute and does not show any desquamative gingivitis is not a feature in oral EM unlike pemphigus vulgaris.

The features of anaphylactic stomatitis include urticarial skin reactions which is not seen in oral EM

Erythema multiforme major is more aggressive form involving multiple mucosa with typical target skin lesions; while lesions of EM minor are single mucosal ulcerations and typical target lesions of skin. The oral ulcers are large with necrotic tissue tags.8 Bloody encrustation of lips is commonly seen. The third category of EM is known as oral EM having lesions confined only to oral mucosa and lips without cutaneous involvement.9

The drugs triggering EM lesions are co-trimoxazole, long acting sulfa drugs especially phenytoin, sulphonamides, carbamazepine and nonsteroidal anti-inflammatory drugs such as diclofenac, salicylates and ibuprofen.10

Management of oral EM involves identification of triggering agentand treatment of lesions palliatively with analgesics and antibiotics.11 Mild case respond to topical steroids, while for severe cases systemic corticosteroids are recommended.

CONCLUSION

Oral EM, is a rare variant of Erythema multiforme often triggered by HSV infections and rarely by adverse drug reactions. Even though initial attacks are confined only to oral mucosa, subsequent attacks can produce more aggressive forms involving skin. Hence, early clinical recognition of this disease remains essential to promptly initiate appropriate treatment.

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THE VERSATILITY OF SUBMENTAL INTUBATION SIMPLE ROUTE OF INTUBATION IN COMPLEX MAXILLOFACIAL TRAUMA

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Abstract

Achieving a secure airway is of utmost importance in patients under general anesthesia. The two most common methods of airway management (intubation) in such patients include oro-tracheal and naso-tracheal intubation. However, both may be contraindicated in complex maxillofacial trauma, like panfacial fractures requiring surgical access to oral and nasal cavity in the same surgery. Traditionally, the only other alternative available in such situation is tracheostomy, which is associated with a high risk of iatrogenic complications. This report presents our experience of airway management using submental intubation in complex maxillofacial

Key words: Submental intubation, maxillofacial trauma, airway management.

Introduction

Achieving a secure airway is of utmost importance in patients under general anesthesia. The two most common methods of airway management (intubation) in such patients include orotracheal and naso-tracheal intubation. However, both may be contraindicated in maxillofacial trauma, like panfacial fractures requiring surgical access to both the oral and nasal cavity at the same time.

Hence, management of airways in the presence of midface or pan-facial injuries with mandibular involvement requires special consideration. Tracheostomy remains an excellent procedure for establishing a definitive surgical airway. This procedure may involve a significant risk of iatrogenic complications, such as tracheal stenosis, internal emphysema, damage to the laryngeal nerves, tracheosophageal fistula and scarring.

Indications for alternative route of intubation

For fractures that do not involve the occlusion such

as nasal, zygoma, nasoorbito-ethmoidal (NOE), frontal and orbital blow out fractures, oral intubation is indicated. For fractures that involve the occlusion such as mandibular and Le Forte fractures, oral intubation inhibits appropriate resolution of occlusion. In these situations, nasotracheal intubation is indicated.

However, under certain circumstances, such as persistent cerebrospinal fluid leakage, pan-facial fractures, stenosis of the nasal airway by deviated nasal septum, hyperopic turbinate, and nasal polyps, an alternate method of intubation is sought.

In some cases, tube exchange is possible, while tracheostomy is the preferred option in other situations. Retromolar intubation or dividing the surgery in to two separate procedures (nasal and oral intubation) are other options.

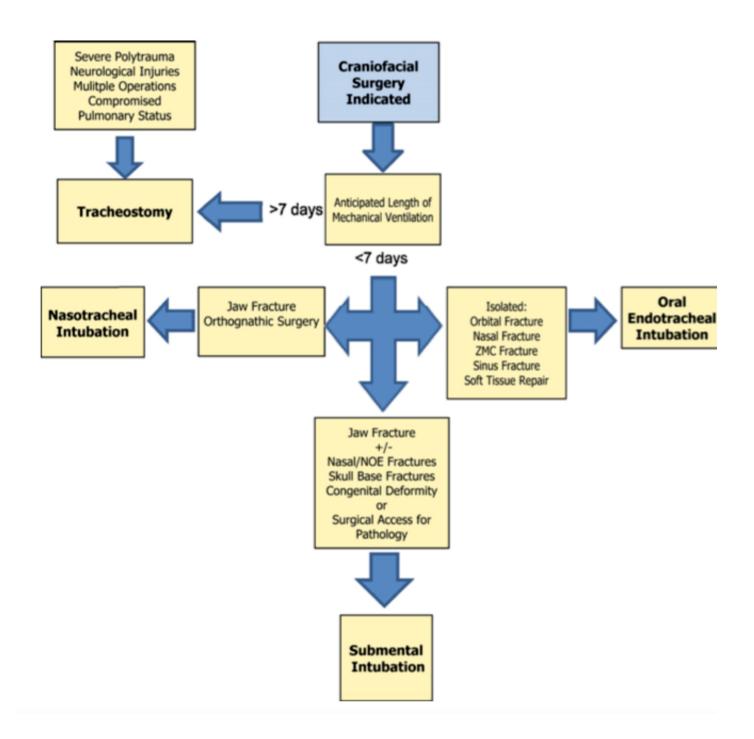
Submental intubation

An alternative to the classic methods is the submental route for tracheal intubation. Introduced by Hernandez Altemir in 1986,

this method has gained wide acceptance, and with some modifications, is an excellent alternative to tracheostomy. The technique consists of diverting the proximal end of an orotracheal tube through the floor of the mouth

and submental region. This allows free intraoperative access to the dental occlusion and nasal pyramid without endangering patients with skull base trauma, and at the same time avoids transtracheal dissection.

Maxillofacial Airway Algorithm



Case Presentation

A 24-year-old male patient presented to VMC ER following Road Traffic Accident. Following detailed clinical examination and CT evaluation, he was diagnosed with bilateral Leforte II fractures with derangement of occlusion; and was planned for open reduction and internal fixation under GA. The involvement of naso-orbital-ethmoidal complex, mobile midface and deranged occlusion, warranted the need for submental intubation.

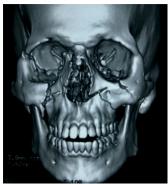




Figure 1 & 2: Fractures involving bilateral midface region at Le Forte II levels

Technique of submental intubation:

After establishing orotracheal intubation, a 2 cm skin incision is made on the median region of the submental area, directly adjacent to the lower border of the mandible. Following blunt dissection of the platysma and mylohyoid muscles, and maintaining close continuity to the lingual cortex of the mandible, a tunnel is created by placing an incision on the mucosal layer of floor of the mouth, in front of the sublingual caruncle.4 It is important to establish adequate width of the submental access to allow free passage of endotracheal tube. (Figures 3 & 4)



Figure 3: Skin Incision in submental area



Figure 4: Blunt dissection to the floor of mouth

After the surgical access was made, the pilot balloon with inflating tube was first passed through the tunnel with the forceps. ET tube was held firmly in position by an assistant while maneuvering the tube from oral to submental position. The tube was then disconnected from the ventilator and the universal connector briefly, and the tube brought out through the submental tunnel. Finally, the tube was positioned, the ventilation restored after checking bilateral air entry. Sutures were used to fix the tube in position.2,4,5 After the surgery, the submental intubation was converted to an orotracheal intubation by replacing the tube in the mouth and extubated in the conventional manner. The skin was sutured with 5-0 prolene and mucosal layer over the floor of the mouth with 3-0 vycril (Figures 5 & 6).



Figure 5: Submental Position



Figure 6: Reconverted to oral route

Discussion

Submental intubation allows for mobilization of the dental occlusion, and those of orotracheal intubation, and allows access to frontonasal fractures. It also avoids the risk of iatrogenic meningitis or trauma of the anterior skull base after nasotracheal intubation, as well as complications of tracheostomy.5,6

Nonetheless, tracheostomy is indicated in patients who present with a neurologic deficit or thoracic trauma and need more than 7-14 days of post operative ventilatory support or in patients with multi-trauma who require long periods of assisted ventilation.1,2,5

Although the incision for gaining submental access can be placed laterally/paramedially, midline access is most versatile because there are only a few anatomic structures present, and there is a minimum risk of nerve or vascular damage. Secondly, the scar is less visible behind the symphyseal region.3,6 Conclusion

Submental intubation should be chosen whenever possible in cases of purely maxillofacial trauma. It demands certain surgical skill, but it is simple, safe, and quick to execute. It also allows for operative control of the dental occlusion and the concomitant surgery of the nasal pyramid in major maxillofacial traumas and avoids iatrogenic placement of tube in skull base fractures.

Finally, it presents a low incidence of operative and postoperative complications and eliminates the risks and side effects of tracheostomy.

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LAB ON A CHIP "MINIATURIZED LABORATORY"

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Abstract

Since long oral cancer is considered as disfiguring and deadly disease of the oral cavity. Though there is increase in therapeutic modalities there is poor prognosis because of lack of early diagnosis and detection. For the timely care and prognosis, a mass screening procedure rapid diagnostic test is required. Lab on a chip is a promising replacement of all the laboratory procedures into a miniaturized chip with all levels of identification of cancer biomarkers from gene profiling to proteins in cancer cells.

Introduction:

Even though there is great progress in the diagnosis and treatment of many diseases, malignancy has been the most common cause of death worldwide. Accuracy in early screening and diagnosis is the critical part of the day to day new invention in the field of medicine and diagnostics. The whole world is putting their hands together in the research of the informative cancer biomarker detection in the saliva and to simplify the cumbersome lab procedures into a pocket card that is lab on a chip also called as micro total analysis system μ TAS.1

History:

It was in 2002 National Institute of Dental Craniofacial Research (NICDR) initiated the research effort on salivary diagnostics. NIDCR has funded the micro electro mechanical system for the salivary diagnostics. The important research group at the university of California Los Angles (UCLA) in developing the point of care micro-fluidic system for oral cancer and breast cancer , metabolic diseases (e.g.) diabetes.2

Discussion:

Micro-fluidics technology also called as lab on a chip or μ TAS micro total analysis system, is defined as the adaptation, miniaturization, integration and automation of analytical laboratory procedures into a single device or chip .Used in the analysis of disease diagnostics, control drug delivery, drug discovery, air and water quality control and monitoring.4 The sample introduced can be small biopsy or blood saliva etc. We will discuss about oral cancer diagnosis. 1

Typically micro-fluidic technology enables the activation of fluids and manipulation of the bioparticles (eg DNA, RNA, proteins and cell) at microscale. 1

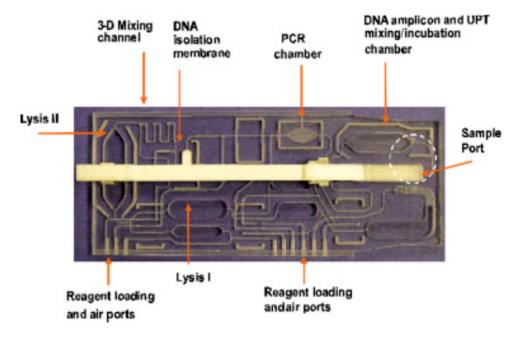
Microfluidic culture based studies gave way for integrated genomic, protemic and cytomic analysis to identify different novel biomarker potentially involved in tumerogenesis. 1

Types of microfluidic based analysis

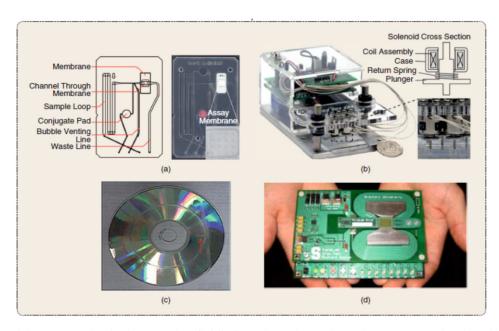
- 1. Microfluicidic –based gene analysis
- 2. Micro fluidic –based protein analysis
- 3. Microfluicidic –based cell analysis

There are two types of devices

- Stationary configuration, in which conventional PCR sample is held in a micro chamber and temperature of the chamber is cycled
- 2. Flow through configuration in which the sample flows through different thermal zones responsible for distinctive processes such as denaturation, annealing and extention.



Example of a microfluidic lab-on-a-chip. The saliva sample is introduced into the sample port. The saliva sample is lysed with enzymes, detergent, and chaotropic salts in a 2-step, 2-chamber lysis process. The nucleic acids are isolated from the lysate by solid-phase extraction using a porous silica membrane as a nucleic acid binding phase. Purified nucleic acids eluted from the silica membrane are amplified by PCR using specific primers. The PCR amplicons are labeled with up-converting phosphor particles and conjugated to antigens, then run on a blotted nitrocellulose strip, where they are captured by immobilized antibodies and detected by a laser scanner.



Examples of promising LOC technologies. (a) Microfluidic flow-through membrane immunoassay developed in the Yager laboratory achieves rapid and sensitive detection using dry reagents stored on the disposable card. (b) The Sia laboratory has demonstrated higher-level integration that is completely battery powered. (c) A CD-based approach for cell detection from the Liu laboratory reduces the requirements for pumps and valves. (d) The Wang laboratory has developed a wash-free multiplexed immunoassay based on magnetic nanotechnology. (All reproduced with permission from the Royal Society of Chemistry.)

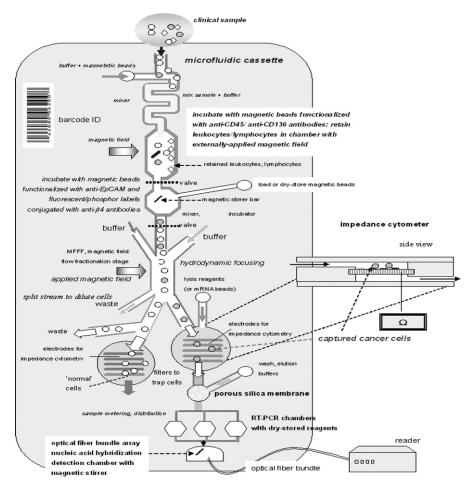
Microfluidic gene analysis or gene profiling: Genetic changes in cancer cells had lead to altered gene expression patterns which gives different biomarkers that can be used for detection of cancer cells. Gene expression profiling has shown many things in regards to progression of diseases, intra and extra vascular invasion and tumor development also. Whereas somatic cells show various heterogeneity in its mutation so gene profiling them is a challenging one.

Capillary array electrophoresis (CAE) is the basic DNA sequencing method involved. The genetic changes in cancer cells have lead to altered gene expression patterns that can be identified before the expression of phenotype. The gene expression in OSCC tumor progression are P53,cyclin D, epidermal growth factor receptor (EGFR) gene. There is

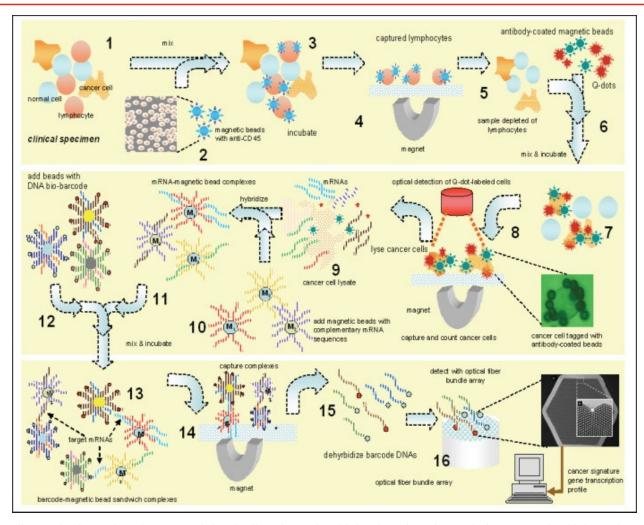
no single gene expression profiling for OSCC . They have found that 25 genes play a role in OSCC and 25 gene predicting Lab on a chip device is necessary to test a cancer patient saliva.

Along with DNA analysis free floating RNA or cell free RNA is also tried in cancer detection, the procedure in the Lab on a chip device is via , capturing and isolation of cancer cells from normal cell with magnetic beads coated with antibodies to these cancer cells, then lysis, nucleic acid isolation via solid phase extraction or hybridization ,PCR, amplification labeling and detection.4

mRNA based detection of exfoliative cells in saliva analysis is done where mRNA can be isolated amplified using reverse transcription -PCR(RT-PCR) and gene expression can be profiled.4



Plan view schematic of a comprehensive cancer diagnostics lab-on-a-chip integrating microfluidic components for lymphocyte depletion, cancer cell isolation and lysis, mRNA isolation, multiplex amplification, and detection of a panel of mRNA.



Cancer diagnostics format based on magnetic bead cell sorting and multiplex detection of a panel of mRNAs using biobarcodes. The cell-laden sample containing normal cells, cancer cells, and lymphocytes is introduced into the microfluidic cassette [1]. The cell suspension is mixed [2] and incubated [3] with magnetic beads functionalized with anti-CD45 antibody that specifically binds to lymphocytes. An external magnetic field isolates the lymphocytes-magnetic bead complexes from the solution [4]. The supernatant, depleted of lymphocytes [5] is mixed and incubated [6] with magnetic beads functionalized with antibodies to membrane glycoproteins specific to cancer cells (such as EpCAM and HSP47) and with quantum dots conjugated with the same antibodies as the magnetic beads or different antibodies specific to membrane proteins of cancer cells. The cancer cell-bead-quantum dot complexes are isolated from the solution with the aid of an external magnetic field [8]. The compartment is washed to remove any unbound quantum dots. An estimate of the number of cancer cells is obtained with a CCD camera. The immobilized cancer cells are lysed [9], and then mixed and incubated with magnetic beads functionalized with oligonucleotides complementary to a pre-selected set of mRNAs and to housekeeping genes [10]. The magnetic beads with captured mRNAs are isolated by application of an external magnetic field and thoroughly washed [11]. The captured mRNAs are hybridized with nanoparticles with oligonucleotides complementary to the specific cancer cell mRNA and the barcode DNAs [12] to form barcode-magnetic bead-selected mRNA sandwiches [13]. The magnetic beads are immobilized and the solution is washed [14]. The barcode DNAs are removed from the beads [15] and detected by fluorescence using fiber optic array [16]. The measured profile is compared with archived gene transcription profiles to determine the cancer type and stage. The detection method described here combines the bio-barcode format.

microfluidic -based protein analysis: There is a difference in gene expression in cancer cells along with instability, protein cancer biomarkers are direct expression of oncogenes. By determine the protein expression we can monitor treatment prognosis, microfluidics based immunohistochemistry is use for protein detection.1

Antibodies against cancer specific membrane protein conjugated with labels such as fluorescent antibodies and quantum dots or magnets can tag the cancer cells. They have found two membrane glycoprotein expressions in cancer and precancer dysplastic cells.

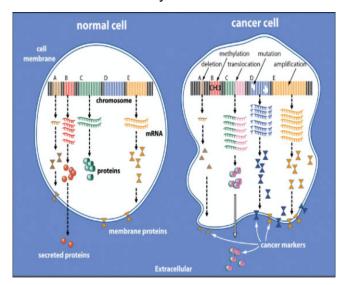
The Membrane Glycoproteins

1. Hsp 47- colligin

Ep CAM- epithelial trans membrane glycoprotein

Hsp 47 -endoplasmic reticulum resident protein. Ep CAM expressed in several epithelial cancers. Found high in tongue cancer. Studied by Chaudal et al.

Cancer cytology analysis with micro fluidic system



Potential cancer biomarkers exemplified by genetic changes in the chromosomal DNA are illustrated in the cancer cell. Typical changes in the host DNA such as point mutations, deletions, translocations, amplifications, and methylations alter mRNA transcripts from these affected genes. These affected mRNA transcripts could be lost, mutated, or increased. As a result of the mRNA changes, cellular protein products from these affected genes are similarly altered. The altered proteins in the cancer cell are expressed intracellularly, on the cell surface, or secreted into the extracellular space at higher or lower levels compared to normal cells. Exploitation of specific changes that occur in the cancer cell's RNA or protein provides convenient targets to enrich the cancer cells from normal cells and other cell types.

Cellular analysis of cancer gives an assessment of single cell or cell to cell communication in the microenvironment.

Simple form of micro fluidic diagnostics devices are pregnancy test and saliva based HIV test commercially available ad ora sure test. 6

Latest advancement in oral cancer diagnosis called OFNACET Oral Cancer Nanosensor Test by university of California Los Angeles Collaborative Oral fluid diagnostic research laboratory led by David Wong developed point of care to detect oral cancer from saliva

Specific patterns of gene profiling in oral cancer four specific pattern of mRNA appears in saliva of patients with OSCC

- IL-8
- Ornithine decatbonylase
- Spermidine acetyltransferase
- IL-1B



Oral Fluid NanoSensor Test, a handheld, automated, easy-to-use integrated system that will enable simultaneous an rapid detection of multiple salivary protein and nucleic acid targets.

This is hand held automated easy to use integrated system which enables simultaneous and rapid detection of multiple salivary protein and nucleic acid targets.

Advantages:

- 1. Small sample volume
- 2. Automated operation

- 3. Short time for processing
- 4. Reproducible and consistent
- 5. Reduces reagent consumption
- 6. Reduced exposure to hazardous materials /infections agents
- 7. Minimal risk of sample contamination and convenient disposal
- 8. Eliminate human errors
- 9. Easy to use

Conclusion:

The Recent diagnostic efforts in all fluids is developing tremendously and becoming a boon to the practitioners from laboratory diagnostic procedure which might take two days before is ready result in hard in 60 min. This Lab-on-a chip technology has made a significant impact on oral cancer screening and diagnostic and general healthcare.

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A REVIEW OF WEGENER'S GRANULOMATOSIS – A RARE GRANULOMATOUS DISEASE

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Abstract

Wegeners Granulomatosis is a rare multisystem autoimmune disease characterized by necrotizing granulomatous inflammation of the upper and lower respiratory tract, glomerulonephritis and vasculitis. The aetiology of WG remains unknown although a number of exogenous factors have been suggested to be of aetiological relevance. Most clinical characteristics of this disease are nonspecific, making the clinical diagnosis challenging. Histopathological examination of lesional tissue is not pathognomonic, but it remains an essential investigation to confirm the presence of disease and exclude other disorders. The present paper reviews the peculier aspects of this rare granulomatous disease with respect to diagnosis, laboratory features and treatment.

KEYWORDS:

Antineutrophil Cytoplasmic Antibodies, Autoimmune Disease, Gingival Hyperplasia, Granuloma,

INTRODUCTION:

Wegener's granulomatosis (WG) is a unique systemic inflammatory disease characterized by necrotizing granulomatous vasculitis of the upper and lower respiratory tract, pauciimmune segmental necrotizing glomerulonephritis, and small vessel vasculitis.1 WG was first reported by Friedreich Wegener in 1936, incorporating both clinical and histopathological criteria to describe what he believed represented a unique and distinctive syndrome.2 The cause of Wegener's granulomatosis remains obscure, although progress has been made on understanding the interrelation between granulocytes, endothelium. and antineutrophil cytoplasmic antibodies. Environmental factors, such as silicates and nasal carriage with Staphylococcus aureus, might trigger the onset of the disease.The most common oral lesion is hyperplastic gingivitis (Strawberry Gingivitis) which is red to purple with many petechiae that may remain localized in the oral cavity for unusually

long periods of time before multiorgan involvement occurs.3,4

The diagnosis of Wegener's granulomatosis can be dillcult, but is greatly helped by measurement of antineutrophil cytoplasmic antibodies with cytoplasmic staining (C-ANCA). For a proper diagnosis, histological evidence of granulomatous inflammation or small-vessel vasculitis, or both, in the appropriate clinical setting is needed, although a positive result for the C-ANCA test alone is not diagnostic. Management with appropriate therapy produces good response in most cases, with occasional relapses.5 The present paper reviews the peculier aspects of this rare granulomatous disease with respect to diagnosis, laboratory features and treatment.

HISTORY OF FRIEDRICH WEGENER IN WEGENER'S GRANULOMATOSIS:

Friedrich Wegener was born on April 4, 1907, in Varel, a small town in northwestern Germany .Wegener began his medical studies in Munich in 1927, and completed his undergraduate training at the University of Kiel in 1932.6 By that time, Wegener had developed an interest in pathology, because of elective attachments with Karl August Borrmann (1870–1943), who is remembered for his classification of gastric carcinoma.

In June, 1934, Wegener did a post-mortem examination on a 38-year-old man who had died from uraemia after prolonged febrile illness. At autopsy, a saddle nose deformity was noted, and there was inflammation of nasal mucosa and cartilage with destruction of the nasal septum. Middle ear, larynx, pharynx, and trachea were similarly allected. Histological examination revealed granulomatous necrotising inflammation. The kidneys were large and swollen and showed histological evidence of necrotising glomerulonephritis.7

Wegener recognized the importance of his findings and it urged him to study the disorder in detail. Later he also encountered with many same disorder. Finally he said, "This disease was on the verge of being discovered. Somebody had to do it." 8

In 1936, Wegener examined his cases in great detail, excluded an infectious cause, and presented his findings at the meeting of the German Pathological Society in Breslau.In 1967, Wegener published an extensive review on his study cases as Wegener's granulomatosis and also he witnessed the discovery of antineutrophil cytoplasmic antibodies as a marker of disease.9,10

AETIOLOGICAL FACTORS:

The specific aetiological factor for WG remains still unknown. Many clinical and experimental data suggested that microbial exogenous factors may highly prone to disease expression.

 Exposure to infectious agents such as Staphylococcus aureus, Mycobacterium avium- intra cellular or Parvovirus B19 and Fungi causes non specific activation of the

- immune system , resulting in elevation of cytokine levels in the presence of ANCA and leading to cell destruction. 11-14
- Environmental factors have also been implicated as a potential triggering factor for WG.It has been reported that ANCA associated Glomeruonephritis and Vasculitis can be associated with occupational exposure to crystalline silica or hydrocarbon, inhaled fumes and particulars – but the evidence is conflicting.15
- In WG inflammatory response is highly elucidated by pathergic reaction to certain foreign agent in which specific autoimmune response may occur.

CLINICAL PRESENTATION:

Wegener's granulomatosis can affect a wide spectrum of systems, and causes diseases involving Ocular, Cardiac, Aural, Cutaneous, Neural and vascular system. The quintessential features are seen in upper respiratory system, Lungs and kidney.

UPPER RESPIRATORY SYSTEM:

Upper respiratory tract disease occurs in 95% of patients with WG .16 The Sinusitis, solitary and most common initial presentation seen in 73% of patients, although may be unrecognized by clinicians for several months until other manifestations of WG arise.17 Patients may also complain of nasal obstruction, and crusting, foul-smelling rhinorrhea, purulent nasal discharge, epistaxis, hyposomia (due to mucosal swelling) and epiphora (caused by involvement of both the naso-lacrimal duct and the lacrimal sac) .18

PULMONARY AIRWAY:

Necrotizing granulomatous pulmonary inflammation may give rise to a variety of symptoms such as cough (which is usually unproductive), pyrexia, haemoptysis, dyspnea, thoracic pain and post- obstructive infection. Nodular(70%) and cavitary

disease(35–50%)of patients with WG of lung involvement is usually sub-pleural.19,20 RENAL DISEASE:

The renal disease is usually initially asymptomatic, although with time it can leads to potential complications. In WG, ≤20% to 80% of patients at the time of diagnosis is asymptomatic. During follow up 80% to 94% of patients invariably develop renal involvement, characteristic by the presence of focal, segmental, crescentic and necrotizing glomeruonephritis. The glomerulonephritis can lead to rupture of Bowman's capsule. 17,21 ORAL MANIFESTATION:

Oral lesions are reported to be occur in 6-13% of patients.17 The gingivae, particularly the upper anterior region are the usual oral site of involvement of WG. A strawberry- like

gingivitis is suggested to be a one of the characteristic sign of WG. It is thought to be an early manifestation, if present, is characteristic sign of WG. This manifest as enlarged, interdental papillae with red to purple in colour, have petechiae on its surface with granular appearance. Other intraoral sites that are rarely affected include the tongue, palate and lips. Palatal mucosal ulceration and inflammatory destruction is uncommon, but can arise as a down ward extension of WG from the nose and nasal septum. 22-24

DIAGNOSTIC CRITERIA:

In 1990, American College of Rheumatology proposed diagnostic criteria for diagnosis of WG, which requires atleast two features of following four criteria .25

Criteria	Description		
Oral ulcer or Nasal discharge	Development of painful or painless oral ulcers or Purulent or bloody nasal discharge		
Abnormal ches radiograph	Chest radiograph showing the presence of nodules, ?xed in?ltrates or cavities		
Nephritic urinar sediment	Microhaematuria (= 5 red blood cells per high power ?eld) or red cell casts in urine		
Biopsy	Histological change showing granulomatous In?ammation within the wall of an artery or in the perivascular or extravascular area.		

DIAGNOSTIC METHODS:

BIOCHEMICAL INVESTIGATION:

If WG was suspected from clinical history, systemic and oral examination, then biochemical investigations should be done to detect the clinical course of this disease. It should include complete blood count, ESR, C-Reactive Protein, Serum Creatinine, Blood Urea Nitrogen levels, 24hr Proteinuria, Urinalysis and ANCA serology test. Besides this, special stains are also required for detection of microbial organisms to rule out systemic infections.

ANCA SEROLOGY TEST:

The current recommendation for a mandatory ANCA testing for WG is essential when there is a strong clinical evidence of signs and symptoms. The association between WG and ANCA was first confirmed by Vanderwoude et al in 1985.26Initial screening of all sera by Indirect Immunofluorescent on ethanol fixed Neutrophils should be done to discriminate 2 main pattern of ANCA: Cytoplasmicpattern(C-ANCA) and a Perinuclear pattern(P-ANCA).27Myeloperoxidase and Proteinase 3 are the major target antigen for P-ANCA and C-ANCA which is present in the granules of neutrophils and lysosomes of Monocytes .C-ANCA is considered to be a sensitive and specific marker for multisystem WG and may be helpful in tracking disease activity and possible relapse. The detection of ANCA levels also plays an important role in the monitoring of patients response to treatment.28

RADIOGRAPHIC INVESTIGATION:

Chest radiographs and Computerized Tomography scan are mainly required to detect the specific characterized features of WG in Pulmonary system. The most common feature of pulmonary involvement is the radiological presence of single or multiple (usually less than 10) cavitary nodules of 5 to 100 mm diameter at cortical and sub-pleural sites.29

PATHOLOGY:

Biopsy is mandatory to confirm the disease, to rule out and differentiate it from other granulomatous diseases. Wegeners Granulomatosis has three specific pathological features and it serves as an element in diagnostic criteria: Necrosis, Granulomatous Inflammation, Multinucleated

Giant cells and Vasculitis.30 The granulomatous in?ammation is characterized by collections of loose macrophages, multinucleated giant cells, acute or chronic in?ammatory cells, and the cellular composition of the granulomatous lesions of WG are composed of CD4+ T-cells, CD8+ T-cells, histiocytes, CD20+ B-lymphocytes, neutrophil granulocytes, CD68+ macrophages and CD68+ multinucleated giant cells that envelop the central area of necrosis.31.32

The central necrosis can be distinct and shows a serpiginous pattern of necrosis, Polymorphonuclear leucocytes and epithelioid histiocytes which may get arranged around the necrotic foci, occasionally. And the vasculitis of WG typically show ?brinoid necrosis affecting the walls of small to medium- sized arteries and veins, the affected vessel wall has acute/or chronic in?ammatory in?Itrate and occasionally accompanied by granulomatous in?ammation within the vessel wall. At the same time, most of the case reports with Gingival biopsy of WG shows pseudoepitheliomatous hyperplasia, Polymorph microabscess and giant cells .30,33...

TREATMENT

The choice of therapeutic agents for WG depends on severity of disease. If correct therapeutic decision is taken, most of the patient responds immediately to treatment within a week. Therapy is mainly aimed at inducing remission with oral prednisolone 1mg/kg and cyclophosphamide 2-3 mg /kg. Once remission is achieved prednisolone is usually tapered gradually to alternate days at 3 months and then discontinued, whereas cyclophosphamide is continued for atleast a

year after remission induction.34 Resolution of oral lesions, clearing of pulmonary infiltrates with evidence of stable scarring and no further evidence of active renal sediment signifies complete remission. However, remission in some cases may soon be followed by relapse which usually coincides with tapering of immunosuppressive therapy. At present combination of Azathioprine and low dose prednisolone are mainly used as maintenance therapy. Because of high morbidity associated with standard therapy, intermittent intravenous treatment with cyclophosphamide has been introduced with the intention of reducing treatment related morbidity. 35,36,37.

CONCLUSION

Wegener's granulomatosis is a multisystem disorder associated with significant morbidity and mortality. ANCA and other immunological analysis are relevant to its diagnosis but histopathological confirmation is vital. The oral health care provider's role is vital to the diagnosis of Wegener's Granulomatosis as "Strawberry Gingivitis" could be an early presenting symptom. Appropriate referral and relevant management of the oral lesions would be under the purview of the oral physician thereby ensuring early diagnosis and better outcome.

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MANAGEMENT OF PALATOGINGIVAL GROOVE ASSOCIATED WITH LOCALIZED PERIODONTITIS - A CASE REPORT

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Abstract

Palatogingival groove is a developmental anomaly which has been implicated as an initiating factor in localized gingivitis and periodontitis. These grooves which facilitate plaque growth can present as a challenge to the operator in diagnosis and treatment planning. This article describes the management of shallow palatogingival grooves present in the maxillary incisors. In the present case, a timely diagnosis was made and treated surgically with odontoplasty and sealing of the grooves with BiodentineTM and Mineral Trioxide Aggregate (MTA). On re-examination of the patient after 6 months, patient had good oral hygiene and no signs of disease progression.

Keywords: Palatogingival groove, odontoplasty, Biodentine, Mineral Trioxide Aggregate Glomerulonephritis

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The tooth is a specialized part of the human body, understanding the development of which is enigmatic and still challenging.1 Anatomical malformations of tooth often causes clinical problems and one such variant is the palatogingival groove(PGG).

The palatogingival groove is a developmental anomaly of the maxillary incisor teeth which has been reported to be associated with severe localized periodontal disease.2-5 Palatogingival groove also referred to as palato radicular groove (PRG), disto lingual groove, corono radicular groove or radiculo lingual groove is described in literature as a developmental malformation that exists on the palatal aspect of the incisor teeth and runs towards the mesial, distal or midpalatal root regions.3,7 It has a reported incidence ranging from 2.8 to 18%.6

In most cases, the course of the grooves is straight. According to their localization, they are differentiated as distal, mesial and central patterns, with the distal portion dominating, as it occurs in approximately 70 % of cases.8 The grooves also

vary in depth. Deep grooves with direct communications with the pulp are seldom reported. With increasing depth of the goove, the thickness of the root cementum increases.9

The negative effect of palatogingival groove is related to their plaque accumulating effect. The groove may facilitate plaque growth by providing surface areas sheltered from cleaning efforts as well as from host defense mechanisms.

Prichard2 was the first to state that lingual grooves on maxillary incisor teeth are a predisposing factor to localized severe periodontal destruction.Lee et al3 reported on 13 patients who were seen with localized periodontal lesions associated with these anomalies. Simon et al4 described unsuccessful attempts to treat the periodontal defects associated with palatogingival grooves and felt that extraction of the involved tooth was the treatment of choice.

Various materials have been used for sealing the PRGs.In the present case a tricalcium-based cement called BiodentineTM was used for sealing the palatogingival grooves in two teeth and Mineral trioxide aggregate (MTA) was used in the other tooth.

Biodentine, a tricalcium silicate-based material popularly known as "dentin replacement and repair material" was introduced commercially in 2009. It is recommended for use as both an endodontic repair material and a dentinsubstitute under resin composite restorations. It contains tricalcium silicate, dicalcium silicate, calcium carbonate and oxide, iron oxide, and zirconium oxide as its powder components; and calcium chloride and a water soluble polymer as its liquid components.

MTA is a powder that consists of fine trioxides (tricalcium oxide, silicate oxide, bismuth oxide) and other hydrophilic particles (tricalcium silicate and tricalcium aluminate) responsible for the chemical and physical properties of this aggregate which set in the presence of moisture. Hydration of the powder results in the formation of a colloidal gel. The gel solidifies to a hard solid in approximately 3 to 4

hours. This cement is different from other materials because of its biocompatibility, antibacterial properties, marginal adaptation and sealing properties and its hydrophilic nature.

It is important to note that it is the ability to adequately treat the periodontal defect that ultimately determines the prognosis of these teeth.14

ETIOLOGY

The etiology of PGGs is unknown. Similar to an invagination this seems to be a peculiarity of tooth development accompanied by a further anomaly. Black was the first to describe PGGs as a malformation during embryo development in 1908.10

Atkinson11summarised that the reason for its malformation is that there is no enough space during tooth development in the maxilla, resulting in folding in the area of the Hertwig's epithelial root sheath. In the opinion of Goon et al12 this could be also an attempt at a root partition. According to recent studies, PRGs may be caused due to genetic changes.9

CLASSIFICATION

MILD	Gentle depressions of the coronal enamel, which terminate
	at or immediately after crossing the cemento-enamel
	junction
MODERATE	Continue to extend some distance apically along the root
	surface in the form of a shallow or fissured defect
COMPLEX	Deeply invaginated defects that involve the entire length of
	the root or that separate an accessory root from the main root
	trunk

According to Yong - Chun Gu13

TYPE 1	The groove is short (not beyond the coronal third of the
	root)
TYPE 2	The groove is long (beyond the coronal third of the root)
	but shallow, corresponding to a normal or simple root canal
TYPE 3	The groove is long (beyond the coronal third of the root)
TILS	and deep, corresponding to a complex root canal system

CASE REPORT

A 22-year- old female patient reported to the department of Periodontology, Best Dental Science College and Hospital, Madurai with the chief complaint of pain, intermittent discharge of pus from the upper front tooth region and bad smell for the past 2 days.

There was no history of trauma and the medical history was non-contributory. Dental history revealed that the patient was wearing removable orthodontic appliance for the correction of proclined upper incisors for the past 2 days. On intraoral examination, oral hygiene was satisfactory. On manual probing with William's periodontal probe, there was draining periodontal pocket in the distopalatal aspect (5mm) of 12, mesiopalatal aspect (7mm) of 11 and mesiopalatal aspect (6mm) of 21 and the teeth indicated concavity crossing CEJ extending to the root in the form of a groove (Goon et al- mild; Youn- Chun Gu type1). These grooves on all the upper maxillary incisors were shallow and continue to extend for some distanceapically but not beyond coronal third of the root. These palatoradicular grooves which lead to plaque accumulation and act as a channel for the microbial deposits to carry subgingivally became evident to be the reason for the pathosis to occur. Mobility of tooth was within physiological limits. Thermal and electrical

pulp testing showed normal response. Thus the endodontic treatment was not indicated. The pain initiated from the day she has started using the removable orthodontic appliance which was suspected to be the aggravating factor for the existing periodontal problem.

TREATMENT

Suggested treatment modalities were curettage of the affected tissues, elimination of the groove by grinding (saucerization), or by sealing with a variety of filling materials.14 The patient was instructed not to use the removable orthodontic appliance.Phase I therapy (scaling and root planning) was completed and medications (Amoxicillin 500mg TD and Metronidazole 400 mg BD) were given.

Phase II therapy was carried in two appointments. The first appointment involved the surgical management of 11 and 12 sealing the PRG with biodentin and the second appointment involved the surgical management of 21 with saucerization of the groove and sealing the PRG with respect to 22 with MTA.

SURGICAL PROCEDURE

Anesthesia was achieved after administering 2% lignocaine with 1:80,000 adrenaline. Sulcular incision was given and full thickness flap was elevated on the palatal aspect from

mesial to 13 to mesial to 21. Thorough debridement around the groove was performed by meticulous scaling and root planing. Granulation tissue was debrided using Gracey curettes number 1/2 (Hu-Friedy Manufacturing Co., Chicago, IL).

Next, the groove was shaped with high-speed diamond bur under continuous air-water spray and blended smoothly with the adjoining surface to receive the restorative material. Biodentine™ (Septodont, St. Maur-des-Fosses, France) was mixed according to the manufacturer's instructions and applied into the defect after proper control of bleeding. The material was allowed to initial set for about 9 min. During the setting phase, the tissues were kept hydrated using moist gauze piece. The flap was approximated and sutured using 3-0 silk suture. Analgesics and antibiotics were prescribed, and the patient was given regular oral hygiene instructions including chlorhexidine (0.2%) mouth rinse for 2 weeks.

After 7 days suture removal was done and irrigated with saline. The other palatogingival groove in relation to 22 was sealed with MTA (Mineral trioxide aggregate) in the same way by elevating full thickness periosteal flap. Odontoplasty was done in 21 in which the groove was shallow.

DISCUSSION

Depending on the morphology of the palatogingival groove, localized periodontitis may develop accompanied by pathosis. This fissure like channel is a locus of plaque and calculus accumulation, which acts as a secondary local etiologic factor encouraging the development of periodontitis. An incorrect or delayed diagnosis decreases the prognosis and could result in the extraction of the tooth.

Several different approaches have been proposed for management of palatogingival groove. In most cases where the groove is shallow and not extended apical to CEJ, odontoplasty is sufficient to eliminate the groove by so called 'saucerization' or flattening the groove. This involves the grinding of the root surface, sometimes quite extensively which results in loss of tooth substance and exposure of cut dentin. One limitation of this technique is its impracticality in deep grooves that communicate with the root canal, and as concluded by Meister, "this treatment only can be successful if there is not a continuous opening along the length of the radicular lingual groove between the pulp canal and the periodontal tissues".

However when the groove is more advanced with associated extensive periodontal destruction, the management becomes more complex. The reported treatment procedures in which the groove is extended too far apically include filling of the groove with amalgam17, or calcium sulphate18 or GIC19, or the intentional replantation after root planing and the insertion of emdogain20. In the last decades, with extensive knowledge of guided tissue regeneration, mechanical barriers have been used to halt epithelium downgrowth along the root surface, allowing periodontal ligament, cementum and bone to regenerate along periodontally diseased roots.

In some cases the periodontal pathosis may get driven more apically to involve periapical tissue leading to endodontic-periodontal lesion. In such cases, the treatment plan comprised of oral prophylaxis followed by endodontic management primarily and then periodontal pocket elimination and groove repair.14

In this case, the tooth was vital and had no bony defects associated with it. Localized flap surgery & restoration of defect with filling materials were done in 11 and 12 in which the grooves were quite deep and odontoplasty was done in the groove which is shallow in 22.

MTA was initially introduced as a root-end filling material for surgical endodontic

procedures. Since then, its clinical applications have broadened to include perforation repair, pulp capping, pulpotomy and apexification. During these procedures, the dental filling materials usually come into contact with the underlying tissues. The bond strength of most dental materials is significantly reduced by moisture contamination from the tissue, whereas MTA requires the presence of water for setting. Therefore, set MTA can acquire its optimal strength and produce excellent sealability in the presence of moisture21.

MTA offers a biologically active substrate for bone cells and permits cementoblast attachment, growth and the production of mineralized matrix gene and osteocalcin expression.22 Balto demonstrated that human periodontal ligament fibroblasts were well attached and grew on MTA.23 It has an antibacterial effect, biocompatible, optimal strength and excellent sealability in the presence of moisture, ability to form cementum layer. Because MTA has got all excellent properties in the field of regeneration for both hard and soft tissues, itwas used to seal the groove.

In a study by Zhou et al it was concluded that Biodentine caused gingival fibroblast reaction similar to that by MTA and can be safely used in procedures requiring close approximation with the periodontal tissues.24 It has proven bioactive properties, known to promote hard tissue regeneration and is biocompatible. Compared to glass ionomer cement, this material is more approving when adhesion and growth of fibroblasts is concerned. The ability to form hydroxyapatite crystals at the surface especially when formed at the dentin material interface is known to improve its sealing ability. Sealing with Biodentine was better than MTA due to its better handling characteristics and short setting time whereas when using MTA in sealing of radicular groove it was difficult to control moisture during the setting of MTA causing degradation and poor marginal seal. In addition to documented uses of biodentine in diverse clinical applications like retrograde filling material, perforation repair, pulp capping and pulpotomy; our case shows successful application of Biodentine in management of complicated palatogingival groove compared to MTA.A 6 month follow up revealed reduction in pocket depth of 3mm and no signs of disease progression. In this case if the diagnosis was missed at an early stage, the tooth would have progressed for non-vitality and a combined approach by endodontist and periodontist would be needed..

CONCLUSION

This paper emphazises the early diagnosis of the silent killer PGG and its appropriate management to save the teeth from progressing to hopeless prognosis.





FIGURE 1A, 1B. Removable orthodontic appliance worn by the patient.



FIGURE 2.Patient was having traumatic occlusion with deep bite

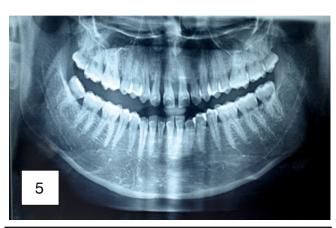


FIGURE 5.Orthopantomography of the patient showing horizontal bone loss in the maxillary anterior region.



FIGURE 3. Palatogingival groove noticed in 11, 12 (pocket depth of 7mm in 11)

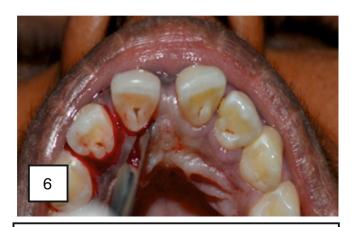


FIGURE 6.Intrasulcular incision was given at the palatal aspect



FIGURE 4.Palatogingival groove noticed in 21 (pocket depth of 7mm in 21)



FIGURE 7. Palatal flap was elevated in relation in to 11, 12



FIGURE 8.Groove blended smoothly with adjoining surface with high-speed diamond bur to receive the restorative marterial



FIGURE 11.PRGs sealed with Biodentine in 11,12



FIGURE 9.Palatogingival groove noticed in 11,12



FIGURE 12.Flap closed with simple interrupted suture with 3-0 silk suture.



FIGURE 10.PRG sealing with Biodentine in 11



FIGURE 13. Palatogingival groove in 22 was sealed with MTA. Palatogingival groove in 21 was managed by saucerization.

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