ISSN No. 0972-396X

KDJ

Kerala Dental Journal

Vol 33 | No. 3
July 2010
Quarterly Publication of Indian Dental Association, Kerala State Branch

Mucositis

Email: editorkdj@gmail.com
Web: www.idakerala.com

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Vol. 33 | No. 3 | July 2010

Contents

President's Message

Dr. Samuel K. Ninan

Kerala Medicare Service Persons and Institutions Bill - A need of the day

The entire medical fraternity of the state has protested recently because of the delay in implementing the above bill. Many of the members are not fully aware of the context in which the protest was exhibited. The number of hospital attacks is increasing day by day in the state and it has deteriorated the morale of doctors in handling risky cases. This has resulted in defensive treatment to safe guard the doctor which in turn has increased the number of investigations and eventually the cost of health care. Doctor should be able to deliver his services without fear. Eleven states including Andhra Pradesh, Tamilnadu, Karnataka, Maharashtra, Rajasthan, Delhi, Haryana, Orissa, Gujarath, Chhathisgad and West Bengal have already implemented a law to protect the health care institutions and the staff from attacks. The government of Kerala had appointed the Law Reforms Commission to prepare a draft bill and the commission was chaired by Hon. Justice V.R. Krishna Iyer. The commission had prepared the draft bill in 2008 and which was verified by all the concerned departments. But, due to some unexplainable reasons, the implementation of the bill did not take place.

A Joint Council was formed to take necessary steps for the implementation of the bill, under the leadership of I M A and other organizations. IDA has actively participated in the protest strike and three of us- Dr. Samuel K. Ninan, Dr. Shibu Rajagopal and Dr. K.N. Pratap Kumar observed hunger strike in front of the Secretariat on 27th June along with other leaders of IMA. A large number of our members visited and expressed the solidarity at the "sathyagraha pandal". I take this opportunity to express my gratitude to the members. We have temporarily with drawn the strike on 29th on account of the written assurance received from the government regarding the implementation of the Law. Let us hope that the collaboration with IMA will be maintained in the future on matters of common interest.

A Permanent International Study Centre will be established by IDA at IMA House, Kochi. This will be first of its kind in India which will benefit all the dental professionals of our country. We are planning to conduct long term courses with accommodation facilities. We are happy that the National Oral Health Programme and School Dental Health programme have been re implemented and the communications have already been sent.

Congratulations to Dr. Presanthila Janam, for being selected for the Best Doctor award of Kerala State.

Thanking You,

Jai IDA

Pathanamthitta,
01-07-2010.

Dr. Samuel K. Ninan

Usher in some changes, because that is the need of the time

Dr. K. Nandakumar

Government of India aims at reforming the entire medical education system in the country. A task force is formed by the Union Health Ministry and it has decided to scrap all regulatory bodies, including the Medical Council of India, Dental Council of India, Pharmacy Council and the Nursing Council. There will instead be a single regulatory body – National Council for Human Resources in Health – which will oversee seven departments related to medicine, dentistry, nursing, rehabilitation and physiotherapy, pharmacy, public health/hospital management and allied health sciences. The process now waits for a formal government approval. The Union health ministry has already drafted a bill titled **The National Council for Human Resources in Health** (Draft Bill, 2009). The proposed Council will not only perform the regulatory functions but also carry out assessment and accreditation of medical and health institutions across the country.

Sixty three years of independence has improved the health parameters of our country. The number of medical, dental and allied colleges has increased in unimaginable proportions but we cannot claim that our educational standards had a parallel growth. A leading national daily has once quoted a comment: "Medical education today is dictated by bank balance and caste. The existing councils, besides being unwieldy, have failed to provide a synergistic approach. There is an urgent need for innovation in health-related education. It is unfortunate that medical seats are auctioned in front of students today." Time has come for some radical steps to be taken by the government.

Councils played a pivotal role in the formative years but drastic reforms could not be brought in because of lack of empowerment. The present action envisions some drastic directional changes. The Journal takes note of the plan to introduce national exist test. Though all central and state universities shall conduct their own examinations and award degrees, the national council will conduct national-level exit examinations to standardise UG/PG medical and allied health courses. It will become mandatory for each graduate to qualify through this exit exam so that he can get registration to practice in this country. This screening examination shall also be mandatory for students who have successfully completed UG from a foreign institution that is not recognised in this country. Such an attempt to implement exit exams for law graduates is already in the process. I am sure that this attempt will not be endorsed whole heartedly but in the larger interest of the country, we have to welcome it. Ultimately the aim of dental education is to produce dentists to whom we can go confidently for our own treatment.

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Radiation mucositis and acute pseudomembraneous candidiasis

There are a few acute and chronic reactions in the oral cavity of patients receiving cancer chemotherapy and head and neck therapeutic radiation. Oral Mucositis and acute pseudomembraneous candidiasis are the most common acute problems seen in cancer therapy. Mucosal injury starts as erythema, develops into oral mucositis progresses to ulceration which gets secondarily infected and ends up a foul smelling necrotic ulcers. These mucosal injuries are undesirable, distressing, painful and expensive to treat. Side effects of cancer therapy and are disheartening for the patients and frustrating to the care givers. The 80% of patients receives radiotherapy for head and neck cancers develop oral mucositis. Rates of hospitalization due to mucositis is reported to be 16% in overall and 32% in Altered Fraction Radiotherapy patients.

Candidiasis is an inevitable lesion in the oral cavity, when there is a change in the oral environment. It is also seen in patients having general immuno suppression due to drugs, radiation and diseases like AIDS. The common subsites in the mouth lesions of acute pseudomembraneous candidiasis appear are palate, tongue, buccal vestibule and the alveolar mucosa. The basic lesion is a scrapable white patch and red mucosa with tiny bleeding point, below the scraped patch.

Often, the complications of cancer therapy are more distressing than the disease. Identifying these problems in patients receiving cancer therapy and treating them with appropriate state of the art treatment is a priority area in modern dental practice. Proper palliative care will improve drastically the quality of life of cancer patients. Training in palliative care is now available to dental surgeons in Calicut and Trivandrum.

Cover case: Dr. P.S. Satheesh Kumar
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Edited by: **Dr. K. Nandakumar,** Hon. Editor • Published By: **Dr. Shibu Rajagopal,** Hon Secretary • **For IDA, Kerala State Branch** • Production : **Suman Graphics,** sumangfx@gmail.com

Crash cart

* Venugopalan, ** Sajith Kumar, *** Shamsad Salim

Introduction

A crash cart is the trolley for storing lifesaving equipment and drugs in a hospital emergency room, intensive care unit, clinics and other areas. The cart is characterized by being easily movable and readily accessible into all sides of the cart for quickly viewing and removing equipment and drugs during a crisis situation.

The first cardiac crash cart was created at Bethany Medical Center in Kansas City, Kansas. The first crash cart was fabricated by one of the doctor's fathers. It contained an Ambu bag, defibrillator paddles, a bed board and endotracheal tubes.

A crash cart or code cart (crash trolley in UK medical jargon) is a set of trays/drawers/shelves on wheels used in hospital emergency rooms for transportation and dispensing of emergency medication/equipment at site of medical/surgical emergency for life support protocols like Advanced Cardiac Life Support/Advance Life Support (ACLS/ALS), Pediatric Advanced life Support [PALS] to potentially save someone's life.

Purposes

1. To enhance the Code Blue team's response to patients with emergency medical situations by providing immediate access to supplies and medications.
2. An emergency cart or crash cart is a cart that facilitates coordination of emergency equipment.
3. A specific crash cart type facilitates staff familiarity with equipment
4. It is help to ensure a properly stocked emergency cart will be readily available
5. It also ensures a properly functioning defibrillator will be readily available.
6. It helps to save the valuable time at the time of emergency.

Contents

The contents of a crash cart vary from hospital to hospital, but typically contain the tools and drugs needed to treat a person in or near cardiac arrest. These include but are not limited to:

- Monitor/defibrillators and suction devices
- Advanced Cardiac Life Support (ACLS) drugs such as Epinephrine, Atropine, Amiodarone, Lidocaine, Sodium bicarbonate, Dopamine, and Vasopressin
- First line drugs for treatment of common problems such as: Adenosine, Dextrose, Diazepam or Midazolam, Epinephrine, Naloxone, Nitroglycerin, and others
- Drugs for rapid sequence intubation: Succinylcholine or other paralytic agents like Pancoronoum , a sedative drugs such as Etomidate or Midazolam; endotracheal tubes and other intubating equipment
- Vascular access devices – Intra Venous(IV) Cannulae, Drip sets [Micro and Macro], IV fluids [Normal Saline(NS), Lactated Ringer(RL), Hydroxy ethyl starch
- Other drugs and equipment as chosen by the facility.

Types of crash cart

Based on age or developmental variations

- Adult Emergency Crash Cart
- Pediatric Emergency Crash Cart
- Newborn Intensive Care Crash Cart

General information

1. A Licensed Staff member as designated by the head of the department is responsible for checking the crash cart, oxygen cylinder levels, defibrillator, and documenting compliance on crash cart checklist.
2. Each emergency cart is equipped with a lock and kept locked unless in use.
3. If the lock is not intact, the cart is to be checked and unit personnel will replace any missing supplies.
4. Crash cart is checked every shift and recorded.
5. Defibrillator load checks will be performed every shift with the defibrillator plugged in and unplugged.
6. All carts will be opened and checked for contents once monthly and following each use. Sterile items will be checked for package integrity and expiration date. Items with expiration dates expiring within the month will be replaced. The medication drawer will not be opened if it is sealed and intact.
7. Laryngoscopes will be checked prior to placement on the cart and monthly.
8. Oxygen cylinders are replaced when the tank has emptied.
9. Drawers of crash carts are to be clearly labeled to identify contents.
10. Special procedure trays are kept on the bottom shelf.

Front view

Side view

Equipment (essential)

Drawers are organized and arranged from top to bottom in different order in different institutions but it contain medications, IV solutions and tubing, miscellaneous.

The medicines mainly used in crash cart is Adenosine, Amiodarone, Atropine,Dextrose, Dobutamine, Dopamine, Epinephrine, Etomidate, Flumazenil, Lidocaine, Magnesium, Naloxone, Norepinephrine, Procainamide, Sodium bicarbonate, Vasopressin, Verapamil etc. are the some medicines we can commonly see in crash cart

Along with this we can see other equipments to establish and maintain airway, breathing and circulations like ambu bag, nasal cannulae, oral airways, intubation trays (laryngoscope, straight and curved blade, 10cc syringe, lubricant),tongue blades, adhesive tape, exam gloves, suction catheters, endotracheal tubes, tracheostomy tubes, IV cannulae, disposable syringes, oxygen cylinder, defibrillator…etc.

Arrangement of medicines and equipment in crash cart is different from different institutions based on their policy. Different types of arrangements are following:

1) based on the airway, breathing, circulation and

2) based on the need and necessity of medicines and equipment.

1) based on airway, breathing and circulation:-

- **Drawer 1- Medications-** Adenosine, Amiodarone, Epinephrin, Magnesium Sulfate, Atropin…etc

- **Drawer 2 - Breathing and Airway-** Ambu bag O2 Nasal cannulae, Oral airways, Intubation tray, suction catheters, Endotracheal tubes, Tracheostomy tubes, Inner cannulae,….etc

- **Drawer 3 – Circulation:** IV supplies,3-Way, Blood set, ABG kits, heparinized aspirators, Needles, Alcohol swabs, Syringes….etc

- **Drawer 4-Circulation: IV solutions and tubing-** RL, NS, D5W, IV Tubing, Macro & Micro drip, Extension tubing, Blood pump tubing, Arm boards: long & short…etc

- **Drawer 5- Cardiac, Chest Procedures-** ECG electrodes, Restraints, Sterile gloves, Masks with face shields or masks and eye protection, Scalpels with blades, Dressings, drain sponge, Betadine solution, Sutures, silk with needle, Cardiac needle, Sterile towels,3 - lumen Central Venous Pressure catheter kit, Chest tubes….etc

2) Based on the need and necessity of medicines and equipments:

1. Medicines: It is again classified based on the priority-first, Second and Third priority medicines.
2. Equipments -Like Ryle's tube, Macro set & micro set Blood set, I.V splint, Micro pore, Gloves, Kidney tray, ECG electrodes & jelly, Tourniquets & spirit swab
3. IV fluids- Like NS 9% 500 ml, NS 9% 100 ml, Isolyte P, Volven 500 ml
4. Open tray on the top-Disposable syringes, Extension no.10cm, 200cm, IV cannulae, Needles, Intra osseous needle, Defibrillator
5. Bottom of crash cart-Plastic apron, Intubation tray, Intubation pillow, ICD set, Percutaneous tracheostomy set

Commonly on the top of the Crash Cart we can place Defibrillator, Inventory Checklist/ Code Blue sheets. On the side of the Crash Cart we can find an Oxygen Cylinder and al so we can find a Cardiac Board.

In pediatric settings we are using different types of crash cart by using Broselow Cart- A color-coded pediatric Code Blue cart[Color coding is based on the *Broselow Pediatric Emergency]* Tape containing age specific supplies and medications for pediatric patients. The Broselow Cart is designed to provide appropriately sized equipment for a pediatric patient. Each drawer is color-coded and contains supplies based on the age and weight of the patient. The top drawer contains medications for all ages.

Broselow pediatric emergency tape

Now a day's different types of crash kits are also

MEDICINES USED IN CRASH CARTS

DRUG NAME	ADULT DOSE	PEDIATRIC DOSE	INDICATION	FREQUENCY
EPINEPHRINE	1mg IV or 2-5 mg IV via ETT	0.01mg/kg IV or IO or 0.1mg/kg via ETT	Any pulseless arrhythms	Every 3-5 min
VASOPRESSIN	40 units IV	Not indicated	VF, PulselessVT	Single dose, may be followed at 10 min by epinephrine
AMIODARONE	For VF or pulseless VT: 300mg IV push	For VF or pulseless VT: 5mg/kg IV push	VF, pulseless VT, VT with a pulse,SVT	May use 2nddose of 150mg for recurrent VF/VT.In children may be repeated in 5mg/kg doses to a total of 15mg/kg
LIDOCAINE	1-1.5 mg/kg IV push	Same	VF, pulseless VT, VT with a pulse	2nd & subsequent doses of 0.75mg/kg every 5 min to a total dose of 3 mg/kg
MAGNESIUM	1-2g IV slow push	25-50mg/kg IV slow push	Torsde de pointes, known hypomagnesemia	Single dose
PROCAINAMIDE	17 mg/kg IV slow bolus at maximum rate of 50mg/min	15 mg/kg IV load; 3-6 mg/kg over 5min,not to exceed 100mg/kg	VT with a pulse	Continue infusion(4mg/min) until QRS widening>50%,dysrhythmia terminated, onset of hypotension; or 17 mg/kg infused.
ATROPINE	PerfusingPatients: 0.5mg Iv push5min, to maximum of 3mg. Pulseless patients: 1mg IV push q 5 min, to maximum of 3mg	0.02mg/kg; minimum dose of 0.1 mg	Bradycardia, asystole.	May be repeated once upto maximum dose of 3mg
ADENOSINE	6 mg rapid IV push through proximal peripheral line; central line dose is one-half	0.1mg/kg rapid IV push;maximum dose, 6mg	SVT	If needed,2nd dose of 12 mg(pediatric, doble initial dose up to 12 mg); 3rd dose of 12-18 mg
DILITIAZEM	0.25mg/kg to a maximum dose of 20mg IV push over 2min	Same	SVT	2nd dose of 0.35mg/kg,maximum dose of 25mg, at 15min; after conversion, start dilitiazem drip at 5-15 mg/HHH
ESMOLOL	500Mcg/kg bolus over 1 min	100-500 Mcg/kg bolus over 1min	SVT	May give another bolus if desired effect is not achieved; start drip 50Mcg/kg/min
ATENOLOL	5mg IV over 5min	Not indicated	SVT,MI 50mg oral load	Repeat in 10 min, then give
METOPROLOL	5 mg IV push	Not indicated	SVT,MI	Repeat twice at 5min intervals, then give 50 mg oral load
DOPAMINE	5-20 mcg/kg/ min	Same	Hypotension	Low doses are predominantly beta;higher doses become predominantly alpha.
DOBUTAMINE	2-20 Mcg/kg/min	Same	Hypotension	Titrate to effect
NOREPINEPHRINE	Start at 8-12Mcg/min, then titrate to 2-4 Mcg/min for maintenance; maximum dose of 30Mcg/min if hypotension un responsive to lower doses	0.05-2mcg/kg/min	Hypotension	Titrate to effect
PHENYLEPHRINE	100-500Mcg bolus IV	0.1-0.5 Mcg/kg/min	Hypotension	Every 5min until desired effect, then continuous infusionof 40-180 Mcg/min

available it contain different emergency drugs, oxygen system, Ambu disposable resuscitator and manual suction unit. This types of crash kits help us to deliver ACLS measures outside the hospital settings also.

You can make it as your own!

Different crash carts explained above is commercially available and it is costly also. If you are ready, you can also make an emergency crash cart as your own. Buy a plastic double Decker basket with a lid and few pearl pet jars. Take pearl pet jars, put the medicines and label it (stick the label on the sides and top of the jar lid for identification) and keep it on the top of the double Decker basket. Keep the needed equipment and supplies in the bottom of the basket. Cover the basket with lid. Now your emergency crash cart is ready. You can prepare and use this crash cart in any settings with minimal cost.

Conclusion

Crash cart is a specially designed trolley, used for transporting and dispensing medicines and equipments at the emergency site for participating in life saving measures. Crash carts are located in areas of patient care in case of a life-threatening occurrence. Physicians, nurses, pharmacists, and respiratory therapists must become familiar with the contents of this cart. It contains necessary equipments to handle an emergency situation. A crash cart is enabling healthcare providers to manage medical emergencies easily and confidently.

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Bond strengths after bleaching 10% sodium ascorbate - an invitro study

* Satheesh Kumar S., ** Md Aslam Khan

Abstract

Vital tooth bleaching is a safe and well accepted procedure for esthetic considerations. Hydrogen peroxide used for bleaching affects the bond strength of composites to acid etched enamel when bonding is performed immediately after bleaching. This study was done to asses the clinical importance of antioxidants and time of application required to achieve reasonable bond strength immediately after bleaching.

Introduction

Bleaching systems have been received by public as a more conservative and economical method of improving the appearance of dentition. Hydrogen peroxide used as bleaching agent affects the bond strength of resin composites to acid etched enamel when bonding is performed immediately after bleaching treatment. The use of antioxidants before the bonding process could reverse the compromised bonding to bleached surface immediately. The clinical application time required for application of an antioxidant and its relationship to reverse bond strength was undertaken in this study.

Aim

The aim of our study was to evaluate the role of 10% sodim ascorbate on the reversal of bond strength in bleached enamel following different time periods of application.

Materials and methods

90 freshly extracted bovine incisors were obtained and stored in distilled water saturated with 0.05% thymol. The teeth were then cleaned and the roots were sectioned at the middle level with a slow speed diamond saw. The teeth were embedded in self- curing acrylic resin blocks 2x2 cm. The enamel surface was polished with wet 600- grit silicon carbide abrasive paper on a polishing machine for 60 seconds to create a flat enamel surface.[4]

The teeth were randomly divided into 6 groups of 15 teeth each

Group 1: Bleaching procedure was not performed but the surfaces were directly etched with 37% phosphoric acid, and bonded with composite resin (Esthet-X Dentsply, USA) as per manufacturer's recommendation. (positive control)

Group 2 : Samples after bleaching with 35% hydrogen peroxide for 30 minutes were bonded with composite resin without application of 10% sodium ascorbate (negative control)

Group 3 : After bleaching, 10% sodium ascorbate was applied for 5 minutes and then the samples were bonded with composite resin.

Group 4: After bleaching, 10% sodium ascorbate was applied for 10 minutes and the bleached samples were bonded with composite resin.

Group 5: After bleaching, 10% sodium ascorbate was applied for 20 minutes and the bleached samples were bonded with composite resin.

Group 6: After bleaching, 10% sodium ascorbate was applied for 30 minutes and the bleached samples were bonded with composite resin.

Method of bleaching

A commercially available concentration of 35% solution Hydrogen peroxide (Spectrum Chemicals, India) was used and 2 ml of 35% HP solution was applied to the surface of the sample for 30 minutes by repeating the process every 5 minutes.[2]

Application of antioxidant

10% sodium ascorbate (Sigma-Aldrich, USA) solution dripped on the sample following the bleaching process and agitated with a brush for 5, 10, 20 and 30 minutes for groups 3,4,5 and 6 respectively.[2]

Shear bond strengths of the samples were analyzed in a universal test machine across head speed of 0.5cm/mm. the data was directly converted into a computer and Mpa was calculated.[3]

Results

Figure 1 & table I summarize the shear bond strength tests of various groups.

Discussion

Vital tooth bleaching is a safe and well accepted procedure for the treatment of surface and intrinsic staining of teeth. Not only is there an esthetic gain for the patient at minimal risk and cost, but this procedure preserves tooth structure and simplifies restorative

The data was analyzed by ANOVA (one–way analysis of variance) and Duncans Multiple range test.

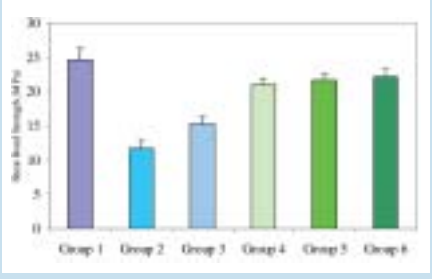

Fig.1 Shear bond strength (MPa) of different groups.

Group	Mean	± SD	F value	p value	
Group 1	24.567[a]	1.656	281.929	< 0.001	
Group 2	11.733[f]	1.894			
Group 3	15.387[e]	1.848			
Group 4	21.060[c]	0.837			
Group 5	21.669[ab]	0.815			
Group 6	22.173[b]	1.017			

Table I. *One way ANOVA of shear bond strength (MPa) comparing different groups*

procedures for the dentist as well.

Hydrogen peroxide is the most commonly used bleaching agent. It is a known fact that bleaching agents affect the bond strength of composites to acid etched enamel when bonding is performed immediately after bleaching. This decrease in bond strength is due to the inhibition of resin polymerisation caused by the presence of residual oxygen . (Rueggeberg 1990, Lai et al 2001).

Antioxidants such as alcohol, catalase, and ascorbic acid have been used as scavengers of these free oxygen radicals (Turkun et al 2004). Ascorbic acid and its sodium salt are the most commonly used antioxidants in restorative dentistry (Buettner1993, Gutteridge1994]. 10% sodium ascorbate (sodium salt of ascorbic acid) with a neutral pH (7.76) is commonly used to overcome the acidic effects of ascorbic acid on teeth. (Lai et al 2002)

It has been postulated that sodium ascorbate restores the altered redox potential of the oxidised bonding substrate and hence allows free radical polymerisation of the adhesive resin to proceed without premature termination (Lai et al 2002).

The results of our study are in accordance with many other studies in that application of 10% sodium ascorbate effectively reverses the compromised bond strength of bleached enamel.

In our study, Group 1 (positive control) showed the maximum shear bond strength values (24.5 MPa) and group 2 (negative control) showed the least (11.7 MPa).

Group 3 showed a slight increase in bond strength (15.3 MPa) compared to group 2 but not to a significant level. This may be due to more peroxide radicals remaining in enamel even after application of sodium ascorbate for 1/6th the bleaching time (5 mins)

Group 4, group 5 and group 6 showed the mean shear bond strength values of 21 MPa, 21.6 MPa and 22.1 MPa respectively.

All the three groups showed bond strength values to clinically acceptable levels with mild increase in bond strength values for Group 5 and Group 6.

Our study confirmed that the reversal of bond strength to bleached enamel was time dependant and that the application of sodium ascorbate for 1/3rd the bleaching time (10 mins) is needed to achieve appreciable bond strength .

Conclusion

Application of 10% sodium ascorbate effectively reverses the compromised bond strength to bleached enamel.

Reversal of bond strength to bleached enamel was time dependant and that 10 minutes (1/3 rd bleaching time) of 10% sodium ascorbate is needed to return the bond strength values nearer to original level.

Additional studies are needed to test higher concentrations of antioxidant agents reducing the time needed to efficient neutralizing action.

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Physical properties of non precious alloys used for dental castings

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Abstract

Physical and mechanical properties of three commercially available brands of non-precious dental casting alloys were determined and related to their compositional and microstructural characteristics. The tests were based on ADA specification No.14. Compositions of the alloys were determined and quantitative and scanning electron microscopic analysis of corrosion characteristics was also studied. Of the three alloys which were tested, two satisfies most of the ADA requirements. The property of one alloy was inferior, that it cannot be used for long span restorations. The study shows that the alloys which had taken up superior surface finish were subjected to less corrosion.

Introduction

The use of metals in dentistry dates back to 400 B.C[1]. Even though gold alloys can be used satisfactorily, its increased cost and search for better mechanical properties, led to the development of non precious alloys. Along with the increased number of the newly introduced alloys, classification systems, conflicting claims of safety, efficacy and economic feasibility and lack of documented long term evidences of clinical reliability, have created a confusing situation, and that made the position of the clinician difficuilt in making a prudent selection. Elwood Haynes in 1910 produced a hard corrosion resistant alloy, using chromium, cobalt, molybdenum and tungsten. This alloy called "stellite" was first introduced in dentistry by Erdle and Prange in 1929(2). Asgar etal[3] in 1970, formulated two sets of alloys with Cr-Co and Ni, and they were compared for the physical properties with the A.D.A specification No:14. A study was conducted by Civjan et al[4] to determine the mechanical properties of two base-metal alloys and to relate these properties to composition and microstructure. Sarkar[5] had conducted a scanning electron microscopic and X-ray microanalysis of in vitro corrosion of a copper-zinc alloy. A comparison of the corrosion behaviour of base metal and semiprecious alloys with noble alloys, in an artificial saliva, was made by Ewars and Thomber[6]. Even though metals are getting replaced in the restorative field, affordability ensures their useful presence for a few more decades and hence the evaluation of properties is relevant.The present study was designed accordingly. The main objective of this study was to evaluate and compare the physical and mechanical properties, microstructure and corrosion characteristics, of three non-precious alloys; as per the American Dental Association specification No. 14.

Materials and methods

Three brands of base metal dental casting alloys viz *Brand A* - Sunilium, (Sankin Industry Co. Ltd Japan), *Brand B* - C.B .80 (Sankin Industry Co. Ltd Japan) and *Brand C* - Remanium (Dentauram Germany) were selected for comparing the physical and mechanical properties, composition, and corrosion characteristics. They were given codes as A, B and C respectively. The properties studied and the methods used for it are as follows.

A. Physical Properties

Preparations of test specimen

The dumbbell shaped specimens were prepared in accordance with the A.D.A specification No. 14[7]. In a split brass mould, molten wax was injected and wax pattern of the specimens were made. Casting of the pattern was done in accordance with the manufacturers instructions. (Fig 1)

Determination of mechanical properties

The diameter of the tensile specimens were determined using a micrometer. The gauge length between two marks were measured. The specimens were mounted in a tensile testing machine (Instron Universal tensile testing machine). The load was applied until the specimen was broken. A load deflection graph was drawn by the machine. The broken pieces of specimen were held in approximation and the gauge length was again measured. The increase in gauge length was found out. The load and the increase in length corresponding to the load were obtained from the deflection graph. The area of cross section was calculated from the diameter using the formula, $A=pd^2/4$. Stress and strain at various points were

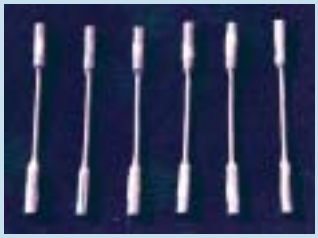

Fig.1 Specimen for Tensile Test

Fig.2 Microstructure of brand A x 64

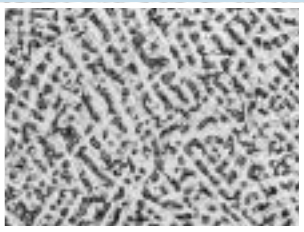

Fig.3 Microstructure of brand B x 64

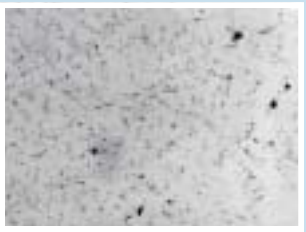

Fig.4 Microstructure of brand C x 64

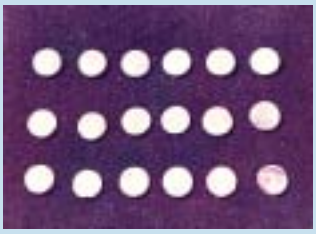

Fig. 5 Specimen for Corrosion Test

Fig.6 Corrosion Pattern of Brand A after one month x 1800

Fig.7 Corrosion Pattern of Brand B after one month x 1800

Fig.8 Corrosion Pattern of Brand C after one month x 1800

determined using the following formula, and the stress-strain graph was drawn.

$$\text{Stress} = \frac{\text{load (Newton)}}{\text{area of cross section (mm}^2\text{)}}$$

$$\text{Strain} = \frac{\text{Increase in length (cm)}}{\text{Original length (cm).}}$$

Ultimate tensile strength

It was determined by dividing the maximum load applied to fracture the specimen by the original area of cross section, before application of load.

Yield Strength

Since the elastic limit is difficult to determine experimentally, the yield strength was used to describe the elastic - plastic transition. It was calculated by dividing the load corresponding to 0.2% offset strain, by the original area of cross section, before application of load.

Modulus of elasticity

It was determined from the stress-strain curve by calculating the ratio of stress to strain.

Elongation

The ratio of the increase in length after fracture, to the original gauge length, expressed in percent, is called the percentage elongation.

Hardness

Hardness of the specimen was determined with the use of Brinell-cum Vickers hardness testing machine.

Specific gravity

The tensile test specimen of each alloy was weighed in a balance. It was weighed again, by immersing in distilled water in a beaker. The loss of weight in water was determined. The specific gravity was calculated by dividing the weight of the specimen by loss of weight in water.

Burnishability

Burnishability of the alloys were calculated using the formula proposed by Moon & Modjeski[8]

$$\text{Burnishability} = \frac{\text{Brinell hardness number}}{\text{Percentage of elongation}}$$

B. Microstructure

The specimens for evaluation of microstructure were etched with concentrated hydrochloric acid, sulfuric acid and nitric acid. The surfaces were observed with a Leitz metallophan microscope at a magnification of 64x and 160x; and the photographs were taken. (Fig 2, 3 and4)

C. Composition

Disc shaped specimens were highly polished. The constituents of the alloy were determined by X-ray fluoroscence spectroscopy and Lecocarbon analyser. Quantitative analysis of the alloys were done by comparing them with standard alloys, whose composition was known.

D. Corrosion characteristics

Highly polished specimen discs (Fig 5) were accurately weighed. The surface were examined with scanning electron microscope (JEOL JSM-35c) under magnification of 180x and 1800x. The specimen discs were stored in artificial[9] saliva. They were taken out and viewed under scanning electron microscope at intervals

Table I. *Physical and Mechanical properties*

Brand	Ultimate Tensile strength MPa	Yield strength MPa	Strain between stress of 350 &4200 kg/cm^2 (cm/ cm)	Modules of Elasticity (MPa)10^3	Elongation %	Hardness VHN	Specific Gravity	Burnish-ability
A	860.40	604.35	0.0054	76.46	7.87	267	8.51	38.053
B	480.82	437.38	0.0072	61.38	4.13	225	7.52	54.695
C	821.43	650.07	0.0058	73.20	5.45	379	8.53	69.947
ADA Specification	618	-	0.0025	-	1.5	300	-	-
Type IV hardened Gold alloy	618-827	493	-	90	7	264	15.2	37.7
Vitallium	870	710	-	223.5	1.6	432	-	-

of 2 weeks and 4 weeks (Fig 6, 7 and 8). The degrees of tarnishing were observed and graded as 0-nil, 1-mild, 2- moderate and 3- severe. The surfaces of specimen were brushed and buffed to remove the corrosion product. Washed, dried and weighed accurately. The loss of weight of the specimen was found out.

Results

Each experiment was repeated six times. The results were subjected to statistical analysis using student t test. The results are given in Table – I and II

Discussion

Ultimate tensile strength

The required minimum value for ultimate tensile strength is 618 MPa. Among the alloys in the present study, only brand A and C could meet this requirement. For B, the value is less because it is a nickel based alloy and does not contain cobalt. Though these alloys do not come up to the level of Vitallium, they have comparable UTS values, to the type IV hardened gold alloy. Brand B, which has got low UTS value, can fail easily on loading, especially when a long span bridge is designed

Yield Strength

The yield strength at 0.2% offset was maximum for C and then A and B. The value for B was very much lower than that of A and C. The result shows that the brand A and C can be used for fabrication of partial dentures. Adjustment of the clasp will be easier with these alloys. Partial dentures made of brand B will undergo permanent deviation very easily. Hence use of this alloy is to be restricted to the fabrication of crown and short span bridges. The A.D.A. specification No. 14 for Cr-Co alloy does not mention a specific value for yield strength. The yield strength is gauged by measuring the strain between stresses of 350 and 4200kg/cm^2. The maximum allowable value for this is 0.0025cm/cm. Since the value for all the alloys are more than this, none of them satisfies the ADA requirements.

Modulus of elasticity

Since the value for Brand A and C are more than that of B, the appliance made of them will be stiffer. If brand B is used, the structure would be unaesthetically bulkier

Ductility and Elongation

Ductility of a material is the property related to burnishability of the margin of casting. Clasps can be adjusted and margins of crowns or inlays can be burnished if they are prepared from alloys with high values for elongation. Table I shows that all brands have percentage of elongation more than ADA specification (1.5%). Hence the partial denture clasps made of them do not easily fracture in service.

Hardness

Ease of finishing the surface of the structure and the resistance to scratching depends on its hardness. While brand C has higher value than ADA specification, A and B have got inferior values. But their values are comparable to the hardness of type IV gold alloys.

Specific gravity

Since the specific gravity is less, appliances made of these alloys will have lesser weight when compared to gold alloys with specific gravity 15.2gm/ cm^3. High density has an advantage in casting procedures. Hence these alloys with low density require special care in producing precision castings.

Burnishability

Burnishability values of the alloys studied were found to be greater than that of type IV gold alloys

Microstructure

Microstructure is the basic parameter, that controls the properties of a material. According to Asgar microstructure of Cr-Co alloys consists of a matrix,

Table II. *Composition (%)*

Brand	Cr	Ni	Co	Mo	Ca	Mn	Fe	C	Ti
A	13.63	56.02	17.45	12.35	-	-	0.21	0.30	0.04
B	15.65	72.20	-	0.65	3.65	7.2	0.4	0.25	-
C	30.45	-	61.82	5.8	-	1.2	0.28	0.45	-

composed of solid solution of various metals like Cobalt, Chromium, Molybdenum etc. and "islands" of interdendritic carbides which acts as cores. The type and arrangement of carbides vary depending with composition and manipulative condition. When viewed under microscope, the grain boundaries were seen as black lines. The carbides solidify last and they appear at the grain boundaries. The carbide cores provide slip interference, and hence strength is increased and ductility is decreased.

In the present study, microstructure of brand A (Fig. 3) showed the presence of carbide precipitate, in the dendritic solid solution matrix. Microstructure of brand B (Fig 4) showed dark eutectoid areas which were lamellar in nature. In the microstructure of brand C (Fig 5) the solid solution matrix formed by various metals showed a dendritic structure and islands of inter dendritic carbides. The microstructure of alloys A, B and C were similar to that of typical microstructure of chromium-cobalt alloy given by Asgar and Peyton.[11]

Corrosion characteristics

In the present study, one month after immersion in artificial saliva,the surfaces of specimens have become slightly dull. Quantitative analysis showed that brand C corroded most, while brand B corroded the least; but this difference was not statistically significant. The scan pictures support the gravimetric evaluation of corrosion of the alloy. This can be read against the fact that brand B had low hardness and had taken up superior surface finish.

Composition

The analysis for composition of the alloy shows that A and B are nickel based alloys. The high value for strength and modulus of elasticity of brand C is due to the presence of cobalt (61.82%)

Summary and conclusions

Physical and mechanical properties, corrosion characteristics and composition of three commercially available brands of non-precious dental casting alloys , viz. Sunilium, CB-80 and Remanium, were determined and related to their microstructural characteristics. Sunilium, and CB-80 are nickel based alloys, while Remanium is cobalt based one.

Only Sunilium and Remanium had the minimum required tensile strength. It is less for CB-80 because it is a nickel based alloy. The yield strength is maximum for Remanium followed by Sunilium. For CB-80, the value was much inferior.

While the modulus of elasticity of Sunilium and Remanium were nearly equal, that of CB-80 was less The percentage of elongation of all the three alloys were more than the specified minimum value 1.5. Remanium was the hardest alloy in the experimental group and that satisfied the ADA requirements. Sunilium and CB-80 had inferior values.

Density of all the three alloys were nearly equal. The burnishability is maximum for Remanium and minimum for Sunilium. Of the three alloys, Remanium showed the maximum values of physical properties.

Sunilium and Remanium can be used for the fabrication of long span bridges and partial dentures. Use of CB-80 should be restricted to the fabrication of inlays, crown and short span bridges.

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Case report

Oral mucous membrane pemphigoid

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Abstract

Mucous membrane pemphigoid (MMP) is a rare group of chronic autoimmune disorders. These disorders are characterized by vesiculobullous lesions that primarily affect various mucous membranes of the body. Early recognition of this disorder and treatment may decrease disease-related complications. The choice of agents for treatment of MMP is based upon the sites of involvement, clinical severity and disease progression. This article reports a case of MMP in a 62-year-old female patient who presented to our dental clinic with multiple ulcers in the mouth which was treated successfully with topical and systemic corticosteroids.

Introduction

Mucous Membrane Pemphigoid (MMP) is a rare group of chronic autoimmune disorders characterized by blister producing lesions that primarily affects various mucous membranes of the body. The condition belongs to a group of mucocutaneous autoimmune blistering disorders often collectively referred to as subepithelial bullous dermatoses (SEBDs). It is most likely to occur in the oral cavity and eyes although other membranes of the body may also be affected.[1] Disease onset is usually between 40 and 70 years and oral lesions are seen as the initial manifestation of the disease in about two thirds of the cases. There is no racial or ethnic predilection although most studies have demonstrated a female-male ratio of approximately 2:1.[2]

Chronic oral soreness is common and can be worse with the intake of spicy food.[2] The clinical appearance is one of gingival erythema and loss of stippling, extending apically from the gingival margins to the alveolar mucosa. The desquamation may vary from mild, insignificant patches to widespread erythema.[3,4] The typical lesion is a small or large, clear –fluid blister, which breaks fairly rapidly in the mouth leading to pseudomembrane-covered, irregularly shaped erosions. These erosions have a yellowish slough and are surrounded by an inflammatory halo. Oral scarring is rare. Some of the patients may also demonstrate a positive Nikolsky´s sign. Usually patients with oral involvement will lack major skin involvement, but the eyelids and genital mucosa are quite susceptible to the blistering phenomenon.[4]

Case report

A 62 year old female patient reported to outpatient section of Department of Oral Medicine and Radiology with the complaint of ulcers in the mouth since 3 months. Patient gave a history of vesicles which ruptured to form ulcers. Patient had pain and difficulty in eating food. She had no significant past medical history. She was moderately built and nourished. Intra oral examination showed diffuse erythematous and erosive areas covered with pseudomembranous slough on the upper and lower labial mucosa, right and left buccal mucosa, upper and lower gingiva and soft palate. Vesicles were present on left buccal mucosa, upper labial mucosa and soft palate (Fig 1, 2, 3, 4, 5, 6). Nikolsky's sign was positive. Oral hygiene was poor and multiple root stumps were present. A provisional diagnosis of vesiculobullous lesion was made. Patient was advised total extraction. A biopsy was taken and sent for histopathological examination. The report was suggestive of non-specific ulcer. A second biopsy was taken and sent for direct immunofluroscent staining and a diagnosis of mucous membrane pemphigoid was given. Initially she was given topical steroid but there was no significant improvement so she was given systemic steroid prednisolone 20mg which was gradually tapered and the erosive areas healed within four months but patient developed edema of face (Fig 7). Erythematous areas were persisting on both buccal mucosa and soft palate hence she was given topical corticosteroid (Triamcinolone ointment) and there was no exacerbation or recurrence (Fig 8, 9, 10, 11). The patient is under follow up.

Discussion

In 1879, Von Graefe was the first to describe the clinical signs, symptoms, and consequences of cicatricial pemphigoid. In the past, MMP was known by several terms, including "benign mucous membrane pemphigoid," "cicatricial pemphigoid" and "ocular or oral–gingival pemphigoid." However, in reporting the results of the First International Consensus on Mucous Membrane Pemphigoid, Chan and others recommended the term "mucous membrane pemphigoid".[1] MMP is a group of uncommon chronic blistering diseases found in 2–5 people per 100,000 population a year. The disease is seen twice as often in women, primarily those middle aged and older.[6] Our patient was a female aged 62 years.

It may affect a number of mucous membranes such as the oral and nasal mucosa, pharynx, anus, genital

Fig. 1 Extraoral photograph

Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

Fig. 2, 3, 4, 5, 6 Intraoral photographs showing vesicles and erosive areas

mucosa, esophagus and trachea. The skin may be affected, but is always a minor component. Patients with ocular involvement may present with pain or the sensation of grittiness in the eye and conjunctivitis. Erosions may be seen on the conjunctival surface. Involvement of the oropharynx may present with hoarseness or dysphagia.[1]

The oral mucosa is often the initial site of lesions in many variants of MMP.[7] Patients often have pain, dysphagia, or peeling of the mucosa. Orally, it primarily affects the gingiva but may involve any area. in our patient there was involvement of labial and buccal mucosa, gingiva and soft palate. Vesicles or bullae may occur anywhere on the oral mucosa and there can be a positive Nikolsky sign as in pemphigus.[8] The blisters rupture and leave irregularly shaped erosions with a yellowish slough and surrounding inflammatory halo that are persistent and rarely scar.[8] Our patient presented with multiple erosions and few vesicles in the mouth.

The pathogenesis of MMP can best be understood by considering the normal epithelial–connective tissue interface. Autoantibodies (IgG or IgA or both) attack 1 or more antigen sites in the molecules connecting the epithelium to the connective tissue and prevent the linkage of molecules in the hemidesmosomes and BMZ. Consequently, the epithelium is poorly anchored to the connective tissue and separates, allowing a subepithelial blister to form.[6] The most frequently targeted autoantigen in MMP is Bullous Pemphigoid Antigen 2 (BPAg2 or BP180); however antibodies to BPAg 1, laminin-5 and á6â4 integrin have also been identified.[9] The diagnosis of MMP is mainly based on history, clinical examination and biopsy of the lesions. When performing a biopsy, it is best to include a vesicle and a perilesional tissue not the erosion itself.[8]

Histologically MMP is characterised by junctional separation at the level of the basement membrane, that gives rise to a sub-basilar split with a chronic inflammatory infiltrate in the lamina propria that contains eosinophils, lymphocytes, and neutrophils.[4] Direct immunofluorescence shows deposits, usually of IgG and C3, in a homogeneous linear manner in the basement membrane zone along the epitheliomesenchymal junction as was seen in our case. Indirect immunofluorescence and immunoblot assays can be used to detect circulating antibodies. Indirect immunofluorescence using salt-split mucosa provides a more sensitive assay.[8]

Treatment of MMP is based on the site involved and severity of symptoms.[1,2] Patients with mild localized lesions may often benefit from topical steroids such as beclomethasone dipropionate, betamethasone, clobetasol propionate, fluocinomide. Patients with more extensive lesions can be prescribed systemic steroids like prednisolone.[1] Our patient was treated with systemic and topical steroids. Other treatment regimens, which were effective in certain resistant cases, were immunosuppressive agents such as azathioprine, cyclophosphamide, cyclosporine and dapsone. Sulphononamides and tetracyclines can also be implemented.[10]

Conclusion

The diagnosis of MMP remains a challenge as patients may approach a variety of specialists depending upon the initial symptoms. Routine histopathology and immunofluorescent study is mandatory to confirm the

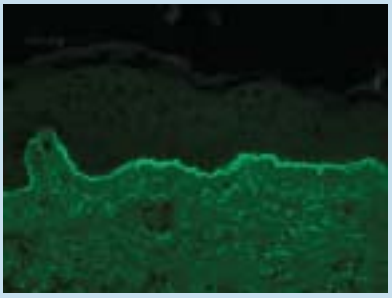

Fig. 7 Direct immunoflurosence showing antibody deposition around basement membrane zone.

Fig. 8 Extraoral photograph during treatment showing edema of face

Fig. 9

Fig.10

Fig.11

Fig.12

Fig. 9, 10, 11, 12 Post treatment Intraoral photographs

diagnosis. As oral manifestations are common in MMP and cause significant morbidity dentists should be able to recognize and manage it, or refer the patients when needed.

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Dr. Ipe Varghese taken charge as Contoller of Examinations

Dr. V. Ipe Varghese, Principal, Govt. Dental College, Kozhikode has taken as charge of the Controller of Examinations of the newly formed Medical university in Kerala.

Dr. Ipe Varghese has got a teaching experience of 30 years of which he was the Principal of the Govt. Dental College, Kozhikode for the past 4 years.

He was the National President of the India association of the Oral Pathologists and the Secretary of the Kerala State IDA and the charter Secretary of IDA Malabar Branch.

IDA wishes him all the best.

Case report

Denture - induced extensive fibrous inflammatory hyperplasia (Epulis fissuratum)

* Omal P.M., ** Mathew Sam

Abstract

Inflammatory fibrous hyperplasia (Epulis fissuratum) is an oral mucosal disease caused by ill-fitting denture wearing. Epulis fissuratum is relatively common and can be treated with surgical excision, conservative methods or both. This is a report of a case of Epulis fissuratum in a female patient which was treated surgically and prosthetically rehabilitated.

Introduction

Epulis fissuratum[1,2,3,5] (Denture induced inflammatory fibrous hyperplasia) is an term related to ill fitting denture wearing. This condition is of great concern to the patient as it leads to pain and discomfort while denture wearing. With proper treatment this condition can be corrected and patient's functional aspect of wearing complete denture can be restored.

Case Report

A 49 year old female patient reported to the Department of Oral Medicine and Radiodiagnosis, Pushpagiri college of dental sciences, Tiruvalla, Kerala, India with a complaint of discomfort while wearing complete denture since 3 months. 3 months back patient started to notice a small soft tissue growth over the denture bearing area later she gradually developed difficulty in using the denture and chewing food. Initially she consulted a private dental surgeon in Tiruvalla and later she was referred to Pushpagiri College of dental sciences, Tiruvalla for further evaluation and treatment. Her medical and family histories were non contributory. On general examination patient had an average built with normal gait (fig 1). Extra oral examination revealed no abnormalities. Intra oral examination revealed multiple folds of hyper plastic tissue in the maxillary alveolar vestibule extending from 16 to 26 region crossing the midline (fig 2) with flanges of the maxillary denture fitting conveniently into the tissues between the folds (fig 3). Size of the lesion measured around 1cm in diameter. On palpation it was firm and non tender. Lower arch patient had an RPD in the anterior segment with few posterior teeth remaining on either sides.

On the basis of clinical history and examination carried out a provisional diagnosis of Epulis fissuratum involving the maxillary edentulous arch was made.After routine blood investigations which were within the normal limits, a surgical excision of the fibrous tissue was planned and performed in the department of Oral and Maxillofacial Surgery. Surgical excision was done in 3 phases. During the 1st phase the redundant tissue from the left maxillary segment was excised under LA (fig 4). To maintain the depth of the sulcus, an Interpositional sterile rubber sheet was placed and fixed with interrupted sutures after dressing with Metronidazole and Chlorhexidine gel (fig 5). The surgical site was left to heal secondarily. A week later 2nd phase surgical excison was carried out in the right maxillary segment with the same surgical procedure (fig 6,7). After a span of 2 weeks, the 3rd phase was carried out in the maxillary anterior segment with a similar surgical procedure employed for the previous phases (fig 8). In all the 3 phases postoperative healing was uneventful.1month later patient was referred to department of prosthodontics for fabrication of a new upper complete denture. 3 months post complete denture follow up was uneventful with no recurrence of lesion seen in the surgically excised sites (fig 9, 10).

Discussion

Epulis fissuratum[1,2] is a common sequela of wearing ill fitting dentures, characterized by hyperplasia of the mucosa due to contact with denture border.Various synonymous[1,3] for this condition include- Denture induced Inflammatory fibrous hyperplasia, Redundant tissue, Denture injury tumor, Denture Epulis. Epulis fissuratum has a strong female[3,4,6] predominance over males and is seen in age group of 30-60 years[4]. Majority of cases, lesions are seen in the maxilla than in mandible[4]. Anterior portion of jaws[3] is affected much more often than posterior area. The lesions may be single or numerous composed of flaps of hyper plastic tissue. Clinically it is characterized by the development of elongated rolls of tissue in the mucolabial or mucobuccal fold area into which the irritating denture flange conveniently fits. Basic etiology of this condition is chronic injury from thin, over extended denture flanges of unstable dentures[1]. Inflammation though variable is seen in the bottom of deep fissures. In some cases ulceration is not uncommon. In our present case, there were inflammations but no ulcerations seen. Treatment of epulis fissuratum depends on the severity of condition at the time of diagnosis. It can be treated with surgical excision, conservative methods or a combination of both[5]. In most cases, inflammatory fibrous hyperplasia are surgically excised either by conventional methods or by C02 laser[7,8] and either a new denture constructed or old denture rebased to

Fig. 1 Facial photograph of the patient

Fig. 2 Multiple folds of hyper plastic tissue in the maxillary alveolar vestibule

Fig. 3 Flanges of the Maxillary complete denture fitting into the tissues between the folds

Fig. 4 Surgical site of left maxillary segment after excision of the redundant tissue

Fig. 5 Interpositional sterile rubber sheet is fixed and sutured during the 1st phase

Fig. 6 Surgical site of Right maxillary segment after excision of the redundant tissue

Fig. 7 Interpositional sterile rubber sheet is fixed and sutured during the 2nd phase

Fig. 8 Surgical excision carried out in the Maxillary anterior segment in the 3rd phase

Fig. 9 3 months post follow up photo of the surgically excised site showing uneventful healing

Fig. 10 3 months post follow up photo after placement of maxillary complete denture

provide adequate retention. The advantage of CO2 laser surgery on oral tissues is the good response to healing and no sutures or packing of any kind are necessary. After surgical excision of the tissue and replacement of the denture, lesions are unlikely to recur[2]. In our present case, hyperplastic tissues in the maxilla were surgically excised and no recurrence was seen after a thorough follow up of 3 months.

Conclusion

Epulis fissuratum is relatively easy to diagnose and treat. It is the dentist duty to reassure the patient about the harmless nature of the condition as Epulis fissuratum always leaves behind in some patients a phobia that it might turn into malignancy.

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Oral candidiasis and major apthous ulcers in a HIV patient

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Abstract

Oral lesions occur commonly in HIV infection. A comprehensive oral examination may not only suggest HIV disease but may also be useful in monitoring the disease progression and initiation of HAART therapy. Oral lesions initially develop when the CD4 count falls below 400cells/mm^3. Herein we report a case of oral candidiasis and major apthous ulcer which lead us to the diagnosis of HIV status in that individual.

Introduction

Oral HIV lesions are common and due to the high propensity of recurrence, prompt treatment and care are warranted to reduce morbidity and mortality. Around 80-90% of HIV positive individuals develop oral manifestations that include oral candidiasis (OC), gingivitis, periodontitis, mucosal pigmentations, ulcers and oral hairy leukoplakia (OHL)[1]. Oral candidiasis and major apthous ulcers are the most common manifestations and occur in patients whose CD4 count falls below 400 cells/mm^3 and thus serve as an indicator of low CD4 count even in the absence of CD4 levels.[2] Joint occurrence of two or more oral diseases in a HIV patient may impact their transmission, clinical presentation, management approaches, and treatment efficacy.

Two types of candidal infection that are commonly encountered in HIV seropositive patients are pseudomembranous and erythematous candidiasis. These infections can affect over 90% of patients at any stage during their illness. Occurrence of OC and OHL singly or together in HIV-1–infected persons may reflect an impaired local oral mucosal and/or salivary defense in addition to a compromised systemic immune system. Risk factors for OC or OHL alone may vary from those of joint occurrence of OC and OHL[3]. OC suggests immunological decline, and may be an initial sign of HIV infection or progression to AIDS. OC has been found to be associated with low CD4 count of 200 cells/mm^3 [4]. Apthous ulcers in HIV patients usually occur when their CD4 count falls below 100 cells/mm^3 and this is a marker to indicate that the patient is under severe immunosuppression.

Case report

A 40 year old female reported with a complaint of pain and burning sensation of the oral mucosa and difficulty in swallowing since 2 months. She was apparently normal 4 months back when she noticed gradual weight loss with fatigue and muscular weakness. She reported to have lost 12 kgs over a period of 4 months. A month back she developed an ulcer on the tongue and multiple curd like patches on the dorsum and lateral border of the tongue. Her medical history revealed that she was a known hypertensive, on medication since 3 years. Her family history revealed that her husband had expired 4 years back due to jaundice and acute hepatic failure. She has two children who are apparently healthy.

General physical examination revealed that she was poorly built and nourished (Fig 1). Her height was 151cm and weighed about 40kgs. Patient was febrile with a body temperature of 101.4^{0}F and B.P was 140/100mm Hg. Extraoral examination revealed dryness of the lips with cracking and peeling of the lips and ulceration at the angle of the mouth presenting as angular cheilitis (Fig 2). Intra oral examination revealed poor oral hygiene and multiple carious and missing teeth. Multiple white creamy curd like patches were noticed over the dorsum and ventral surface of the tongue, soft palate, pterygomandibular raphe which revealed erythematous areas on scrapping (Fig 3). A single large roughly oval ulcer measuring about 1 X 1.5 cm in size was present in the ventral surface of the tongue with indurated margins and covered by necrotic slough (Fig 4). The ulcer was tender on palpation with an indurated base. Multiple small ulcers of less than 2 X2mm in diameter were present adjacent to the large ulcer on the ventral surface of the tongue. A provisional diagnosis of pseudomembranous oropharyngeal candidiasis and major apthous ulcer of the tongue was given.

Patient was subjected to routine blood examination, random blood sugar, chest radiograph, sputum culture, ELISA (HIV).

A final diagnosis of HIV associated pseudomembranous oral candidiasis and major apthae of the tongue was given. Patient was advised nystatin suspension 500,000U to swish and swallow 4 times a day for 14 days, topical triamcinolone acetonide gel to be applied over the ulcer and chlorhexidine 0.12% mouthwash. Patient was advised for counseling and commencement of HAART therapy. On follow up, 3 months after the initiation of HAART along with topical antifungal and steroid therapy the oral lesions had resolved.

Fig 1 Debilitated appearance of the patient.

Fig 2 Cracking and peeling of lips with bilateral angular cheilitis.

Fig 3 Multiple creamy white patches on dorsum of tongue.

Fig 4 Major apthous ulcer at the base of the tongue.

Fig 5 Multiple small ulcers on the ventral surface of the tongue

Table I. *Hematological investigations of the patient*

CBC	*Reported value*	*Reference intervals*
Haemoglobin	11.2gm/dL	12-16gm/Dl
Total WBC count	3100	4000-11000/μL
Neutrophils	80	40-74%
Lymphocytes	14	20-40%
Eosinophils	05	1-7%
Basophils	00	-
Monocytes	01	1-9%
ESR	62mm/hr*	10-20mm/hr
HIV I&II(ELISA and SPOT method)	Positive	-
CD4 cell count	150cells/mm^3	>400 cells/ mm^3

Discussion

Oral candidiasis and oral hairy leukoplakia are common among HIV positive individuals[4]. Examining the risk of jointly occurring oral diseases in HIV-infected persons may provide some insights into the immunological capacity of the oral cavity, and help our understanding of comorbidity interactions with each other and with the oral defense system.[5] The impact of HIV infection on mucosal Langerhans' and CD4 cell populations locally in mucosa is likely central to the pathogenesis of mucosal candidiasis in HIV-infected patients. OC has been shown to be a reliable predictor of HIV infection[1]. Lower CD4 count is an important risk factor for joint occurrence of oral candidiasis and major apthous ulcers. In our case HIV was suspected because of the presence of oral candidiasis, apthous ulcers and drastic weight loss of 12kgs over a short period of 4 months. Hence the patient was subjected to ELISA, CD4 count and total blood picture. Since her CD4 count was 150cells/mm^3 which is very low, HAART was initiated. OC, apthous ulcers and OHL have been used as indicators for initiation of anti-retro viral therapy and to assess the progression.[5] Oral lesions are a component of the different HIV/AIDS classification schemes including the WHO revised classification and CDC AIDS classification.[6] In fact the WHO system emphasizes the use of oral candidiasis and OHL as signs of AIDS in places where HIV serology or CD4 counts may not be available and other causes of immunosuppression are not present.[4] Oral lesions that may appear and resolve, either spontaneously or with specific treatment, decrease the opportunity for professional observation of each clinical occurrence.[1] So careful observation and management should be done when suspecting an individual for AIDS based on such oral lesions.

Oral ulcers resembling recurrent apthous ulcers (RAUs) in HIV infected persons are reported with increasing frequency[7]. HIV associated major apthous ulcers are larger than 10mm in diameter, well circumscribed, round, deep or shallow with indurated margins and may be covered with a grey pseudomembrane. They are usually painful, persist for several weeks and heal with scarring.[8] Apthous ulcers usually appear when the CD4 count falls below 100 cells/mm^3 indicating severe immunosuppression. Cell mediated immunologic dysfunction including impairment of macrophage and adhesion molecular functions have been suggested to play a role in the genesis of apthous ulcers. These ulcers represent an

exaggerated immune reaction in the oral mucosa or they represent a localized autoimmune reaction or develop in response to an unknown antigen or HIV per se, plays a direct role in the pathogenesis of these ulcers is still under debate.[9]

Management of oral candidiasis should be with topical antifungal drugs, if gastrointestinal candidiasis is suspected systemic antifungal drugs are advised. Minor apthous ulcers are treated with topical low potency steroids to avoid further immunosuppression and in case of major apthae, systemic or intralesional steroids are advised. Oral antibacterial mouth rinses and topical anesthetic gels are routinely prescribed to alleviate the burning sensation and infection. Initiation of HAART will help in reduction of severity of oral lesions considerably.

Conclusion

Oral manifestations not only help in early diagnosis but also in monitoring the progression to AIDS. Hence, as oral health professionals, we play an important role in identifying the oral lesions, supplement early diagnosis and aid in improving the quality of life among these individuals.

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High grade variant of mucoepidermoid carcinoma of the buccal minor salivary glands

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Abstract

Tumors of the salivary glands constitute a heterogenous group of lesions of great morphologic variation. Mucoepidermoid carcinoma comprises 4 - 9% of all salivary gland tumors of which 85% are in the minor salivary glands. The palate is the most frequent site for minor salivary gland tumors in 42% to 54% of cases. The lips are the second most common location (21% to 25%) followed by buccal mucosa (11% to 15%). Here we present a case report of mucoepidermoid carcinoma of the buccal minor salivary glands.

Introduction

Salivary gland carcinomas are rare and belong to a clinically diverse group of neoplasms.[1] The myriad of histologic types of minor salivary gland tumors makes this the most heterogenous group of neoplasms of the upper aerodigestive tract[2]. Mucoepidermoid carcinoma is the most common malignant tumor of salivary glands with a widely diverse biologic behaviour that is correlated with the histological grade of the tumor.[3] We are hereby presenting a case report of a high grade variant of mucoepidermoid carcinoma involving the minor salivary glands of the buccal mucosa.

Case report

A 66 year old male presented with a complaint of tooth pieces in the lower left posterior region of the jaw of 3 years duration. The patient had failed to notice a swelling present over the left cheek region. On further questioning the patient gave a history of the swelling being present for a period of 1 month duration with no obvious increase in size. Patient had no associated symptoms. The patient's past medical, dental and drug history did not reveal any information.

On head and neck examination, pallor was noticed. A diffuse swelling was noticed in the lower left cheek region measuring approximately 3x4cm in size (Fig. 1) which was non tender and fixed to the underlying tissues. Right single submandibular lymph node and left single superficial cervical lymph node were enlarged. Mouth opening was limited to two finger breadth.

Intra oral examination revealed a diffuse swelling 3x5 cm in size involving the left buccal mucosa extending superiorly 0.5cm above the occlusal plane, inferiorly involving the lower left buccal vestibule, anteriorly extending from the left angle of the mouth to posteriorly up to 0.5cm from the retro molar region; overlying mucosa showed grayish black pigmentation and appeared wrinkled at some areas (Fig. 2). The swelling was tender and soft to firm in consistency.

A differential diagnosis of a benign salivary gland tumor or a benign non odontogenic tumor in relation to the left cheek region was considered for the swelling.

The blood picture revealed haemoglobin to be 9.3g%, total count of 3,800/cmm, ESR 42 mm/hr and RBC count 3.4 million/dl.

A panoramic radiograph revealed a well defined C shaped radiolucent defect underneath the coronoid process 1.5 cm in size with no internal calcifications or trabeculations which could have been caused by the lesion (Fig. 3).

An ultrasound revealed multilobulated hypoechoic soft tissue lesion in the muscular plane anterior to the masseter muscle, with evidence of multiple calcific specks in it (Fig. 4). The lesion was seen to erode the anterior cortical surface of the mandibular bone. On Color Doppler evidence of increased vascularity within the lesion was seen. There was also evidence of lymph node enlargement in left side of neck at level II, III and IV and bilateral submandibular and submental region; suggesting a possibility of a minor malignant salivary gland tumor.

Ultrasound guided fine needle aspiration cytology gave an impression of mucoepidermoid carcinoma of a high grade variety.

CT revealed a soft tissue swelling with no bony destruction over the left cheek with bilateral submandibular lymphadenopathy measuring 1.8cm. The parotid and submandibular salivary glands were normal (Fig. 5).

Following which, wide excision of the lesion with primary closure and supraomohyoid neck dissection was done under GA. Lip splitting incision was placed followed by dissection of the primary lesion with 1 cm margin. An extra oral submandibular incision was placed with two finger breadth beneath the lower border of the mandible. Subplatysmal flap was raised superiorly up to the lower border of the mandible. Submandibular salivary gland and the submandibular group of lymph

Fig.1 Extra oral view of the swelling

Fig. 2 Intra oral view of the lesion

Fig. 3 Panoramic view showing the C shaped radiolucent defect

nodes were removed.

Histopathology revealed many islands and sheets of tumor cells showing a central area of necrosis. Cellular and nuclear pleomorphisms with numerous mitotic figures were found. Many salivary gland acini large blood vessels, nerve bundles and muscle bundles were seen in proximity to the tumor islands thereby giving an impression of high grade mucoepidermoid carcinoma (Fig. 6). The sections from the submandibular salivary gland and lymph nodes were free from malignancy.

The patient was further referred for post surgical radiotherapy and has been on a continuous periodic recall schedule for the past 1 year.

Discussion

Tumors of the salivary glands constitute a heterogenous group of lesions of great morphologic variation. Eversole has proposed a histogenetic classification of salivary gland tumors implicating two cell types as possible progenitors: the intercalated duct cell and excretory duct reserve cell. Mucoepidermoid carcinoma is a malignant epithelial tumor first studied and described as a separate entity by Stewart, Foote and Becker in 1945. The various factors implicated in the causation of this salivary gland tumor are therapeutic radiation, lipoidal instillation and presence of other foreign material. Mucoepidermoid carcinoma is believed to arise from reserve cells in the interlobular and intralobular segments of the salivary duct system.[4]

Tumors of the minor salivary glands constitute <25% of all salivary neoplasms.[5] Mucoepidermoid carcinoma comprises 4 – 9% of all salivary gland tumors of which 11% are in the major salivary glands and 85% in the minor salivary glands.

The tumor occurs fairly evenly over a wide age range, extending from the 2nd to 7th decades of life. The palate is the most frequent site for minor salivary gland tumors in 42% to 54% of cases. The lips are the second most common location (21% to 25%) followed by buccal mucosa (11% to 15%), retromolar region, tongue and floor of mouth (1% to 5%). 38% to 50% of tumors of the palate and buccal mucosa sites are malignant.[6]

Our case involved the buccal minor salivary glands and was treated as a malignancy.

Minor salivary gland tumors appear typically as asymptomatic swellings the reported duration being one week to 10 years and they can range in size from 0.3 to 4 cm.[6]

Our patient was unaware of the swelling and was completely asymptomatic though in a span of one month the lesion had acquired a size of 3x5cm.

The mucoepidermoid carcinoma is not encapsulated but tends to infiltrate the surrounding tissues, metastasizes to regional lymph nodes. Distant metastases to lung, bone, brain and subcutaneous tissue are also common.[6]

High grade lesions appear as homogenous indistinct infiltrating margins on CT studies with low to intermediate signal intensities on T1 and T2 weighted images. Despite being a high grade in our case, the lesion did not involve any bone destruction.[7]

The standard treatment for mucoepidermoid carcinoma is surgical resection. Minor salivary gland tumors require composite resection with transoral or transfacial approach.[11] Neck dissection is carried out when there is clinical evidence of regional metastases, high TNM staging, high histologic grading and proximity of the tumor to regional lymph nodes.[8,9] Aggressive adjunctive radiotherapy is considered in cases of high risk recurrences and bad prognosis. Histologic high grade mucoepidermoid carcinoma needs adjunctive chemotherapy to prevent local recurrence and distant metastases.[1]

Chemotherapeutic agents are mainly tested as palliation treatment after surgical or radiotherapy failures. Therapeutic effectiveness of single agent Cisplatin in a phase II study of patients with advanced salivary gland carcinoma showed a response rate of 18%. This response though had duration of 5-9 months and an

Fig.4 Ultrasound revealing multilobulated hypoechoic soft tissue lesion

Fig. 5 CT revealing a soft tissue swelling with no bony destruction

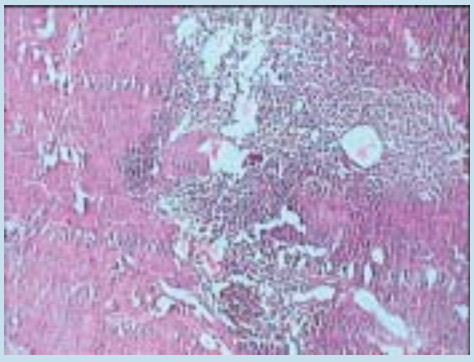

Fig. 6 Histopathology revealing islands and sheets of tumor cells

overall survival of 14 months. A phase II study of Taxane (Paclitaxel) given every 21 days resulted in 8 partial responses among 31 patients with mucoepidermoid xarcinoma.[1]

In minor salivary gland tumors, 5 year disease free survival rate was 35.3% for high grade and 80% for low grade tumors.[10] The outlook for high grade tumors is guarded. The overall recurrence rate of mucoepidermoid carcinoma is about 25% with low grade lesions exhibiting a rate of 10% and high grade variety showing a steep increment to 74%.[7]

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Best Doctor State Award

Best Doctor State Award of the Government to Dr. Presanthila Janam, Prof. and HOD of Periodontics, Govt. Dental College Trivandrum. She received the award from the Hon Minister Sri Elamaram Karim at a function in Trivandrum on 1st July, 2010.

Case Report

Bilateral cervico-facial actinomycosis

* Selvan K., ** Valsa Thomas, *** Sharafuddeen K.P., *** Nileena R. Kumar

Abstract

Actinomycosis is an infectious disease caused by a gram positive bacterium, *Actinomyces israeli*. Approximately 60% of all actinomycotic infections occur in the cervicofacial area, and there is a history of tooth extraction or jaw fracture in about 15-20% of cases. Submandibular region is the most frequent site of involvement in classic human actinomycosis, spread by direct tissue extension. One of the characteristics of actinomycosis is the lack of immediate tissue reaction after implantation of the organisms. This case reports a bilateral cervicofacial actinomycosis with typical features.

Introduction

Actinomycosis, which was first described by Israel in 1878[1], is a suppurative and granulomatous chronic infectious disease, that usually spreads into adjacent soft tissues without regard for tissue planes or lymphatic drainage; it may also be associated with a draining sinus tract.

Actinomyces are Gram-positive, non-acid fast, anaerobic or microaerophilic filamentous branched bacteria, living as commensal organisms in the human oral cavity and respiratory and digestive tracts, becoming invasive when, through a mucosal lesion, they gain access to the subcutaneous tissue. Thus dental caries, dental manipulations and maxillofacial traumas are the most triggering events.

Before the widespread availability of penicillin antibiotics, actinomycosis was a relatively frequent and often fatal disease. Harvey et al reported a 62% mortality rate prior to the antibiotic era[2]. Cervicofacial, thoracic, abdominal and pelvic actinomycosis have been reported. Most frequent clinical form is cervicofacial actinomycosis (40-55%)[3]. It is a challenging clinical diagnostic dilemma because of its variable presentations in the head and neck. A high index of suspicion and biopsy are often necessary to make the diagnosis, especially because it can mimic primary carcinoma[4].

Case report

A 35 year old male patient presented with complaint of multiple bilateral swellings with history of pus discharge in face, neck and forehead since 5 months (Fig 1, 2, 3). Patient had pain since 4 months and intermittent fever since 5 months. Intraorally, mouth opening slightly reduced and multiple draining sinus distal to 38 in retromolar trigone (Fig 4). History of extraction of 37, 47 five months back. His medical, family and personal history was non-contributory. Extraorally, multiple nodules over forehead, submandibular, ramus and angle region. Left submandibular lymph node, 1x1cm in size with soft to firm in consistency. Radiographic examination shows missing 37, 47 (Fig 5). Incisional biopsy was done. Histopathological examination suggestive of actinomycosis (Fig 6, 7).

Discussion

Cervicofacial actinomycosis is the most common form of this rare disease[3]. In cervicofacial actinomycosis, the sites most commonly involved include the submandibular space, cheek, parotid gland, teeth, tongue, nasal cavity, gingiva, hypopharinx, frontal and lymph nodes of the neck[4]. There is a slight male prevalence in young adults, varies the age group of 20-60 years, the highest incidence, in female patients, being found between 11-40 years and in male patients, aged 21-50years[5]. Most cases of cervical actinomycosis are odontogenic origin and occurs predominantly inimmunocompetant individuals[4]. Actinomyces are usually low pathogenicity and cause disease only in the setting of previous tissue injuries like complication of a maxillofacial trauma, after surgical procedures and/or dental manipulations, in patient presenting poor oral hygiene or caries, after recent extraction of a mandibular molar. Actinomyces are bacteria that cannot penetrate healthy tissue and mucosal break-down is a prerequisite for infection[6].

Actinomycosis is an endogenous infection, there is no person to person transmission[6]. Malnutrition, radiation therapy, alcoholic, debilitating states such as diabetes, malignancy and immunosuppression, are considered predisposing factors. The common initial signs and symptoms such as fever, sudden onset of cervicofacial pain, swelling, erythema, oedema and suppuration may be absent[7]. Some authors have described unusual cases of cervicofacial actinomycosis presenting as acute upper airway obstruction and demanding emergency tracheostomy[8]. The infection most commonly presents as a chronic mass, as a suppurative or indurative mass with discharging sinuses, as intraoral lesions, combined with a parotid gland cystadenolymphoma[8], as a chronic, often floating mass frequently located at the border of the mandible with or withought cervical lymphadenopathy. According to some authors, the characteristic sulphur granules, in the



Fig. 1, 2, 3 Extra oral views

Fig. 4 Intra oral view

Fig. 5 Radiograph

Fig. 6 Sulphur granule

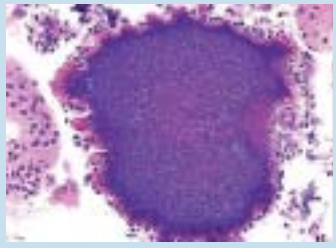

Fig. 7 Histopathology

specimens, are present in only 35-55%cases.In these cases, the diagnosis is definitive[6]. Imaging technique such as CT and MRI usually yield non-specific findings, contributing only to define radiological features of mass and its involvement in adjacent soft tissues. Isolation of the organism and its identification take from 2-3weeks (it grows slowly). Cultures should be placed immediately in anaerobic conditions and incubated for approximately 4-8hrs[3]. FNAC is the method of choice in the diagnosis of cervical actinomycosis[9]. It is an easy, safe, rapid and highly effective method.

Penicillin is the drug of choice for prolonged treatment, usually administered for 2-12months[10]. Tetracycline and erythromycin are employed in patients allergic to penicillin[6], or after intravenous penicillin, for oral use. In the acute phase of treatment, penicillin can be replaced by third generation cephalosporins which are also effective if co-infection with other bacteria, not responding to penicillin causes persistence of symptoms due to actinomyces[7]. A steroid (for example, methyle prednisolone) is effective in eliminating the residual inflammatory granulomatous reaction[7]. Surgical treatment is often indicated for curettage of bone, resection of necrotic tissue masses[10]. Surgery plays an important roll both in the diagnosis and treatment of actinomycosis. Surgery is necessary if CT and US cannot exclude a neoplasm[4]. When the cervico-facial infection is circumscribed, of small size and not associated with draining fistulas, a medical approach is preferred to a surgical procedure[6].

Conclusion

Cervicofacial actinomycosis, although relatively rare, can present a diagnostic dilemma for the clinician because of its capability to masquerades other infectious and neoplastic disease processes. The diagnosis can be confirmed by biopsy and appropriate cultures. These lesions respond well to surgery and extended courses of therapy, with high-dose penicillin remaining the treatment of choice.

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Case report

Regenerative treatment of an immature traumatized teeth with apical periodontitis

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Abstract

Treatment of the immature nonvital teeth includes apexification and calcium hydroxide, surgical endodontics or single visit mineral trioxide aggregate plug and obturation. A new technique of revascularisation has recently been introduced involves disinfecting the root canal,providing a matrix of blood clot into which cells could grow,sealing the coronal access.It results in complete resolution of clinical signs and symptoms,thickening of apical dentinal walls and continues root development

Introduction

The traumatic injury of an immature permanent tooth can lead to the loss of pulp vitality and arrested root development. The consequences of interrupted development include a poor crown-root ratio, a root with very thin walls, an increased risk of fracture, and an apex that is open. The traditional endodontic management of such cases typically includes debriding the root canal, disinfecting the space, and final obturation of the system preceded either by an apexification procedure or by developing an apical barrier by using materials such as mineral trioxide aggregate (MTA)[1-4].

The apexification clinical procedure, which consists of applying calcium hydroxide as an intracanal medication to induce an apical closure over time, has a certain predictability of success[1-3]. Its disadvantages are the necessity of multiple visits during a relatively long period of time (an average of 12 months) and the fact that there is no expectation that the root canal walls will be strengthened[3-5].

An alternative to traditional apexification is to place an artificial barrier at the apex to prevent the extrusion of filling materials during obturation. The material of choice is MTA for its sealing ability and its biocompatibility[4]. This latest technique is convenient because it is faster than the traditional apexification. The case can be finalized within 2 appointments, and a hard tissue barrier eventually forms against the MTA[4]. However, even this alternative approach has the same disadvantage of a tooth with thin dentinal walls and no further root development.

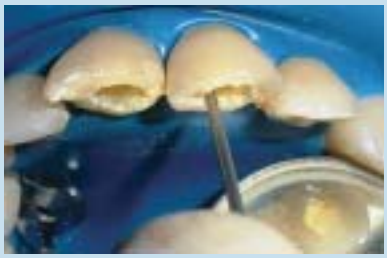
The ideal treatment to obtain further root development and thickening of dentinal walls in an immature tooth with apical periodontitis would be to stimulate the regeneration of a functional pulp-dentin complex[6-8]. It has been proposed that reimplantation of the tooth with an open apex permits coronal proliferation of tissue, leading to replacement of the necrotized pulp and subsequent continued development of the root. Although the histologic identity of this pulp-like tissue is generally unknown, radiographic presentation often includes progressive thickening of the dentinal walls and apical closure. Because the term *regeneration* is based on clinical and radiographic outcomes and not histologic or biochemically based assessments, one can only make a clinically functional interpretation of the healing process. It is not known whether a complete pulp-dentin complex has been regenerated. However, once the regeneration protocol is completed, these teeth can continue to develop, with the radiographic presentation of full tooth development and the clinical presentation of an asymptomatic functional tooth.

The typical revascularization protocol advocates that the immature tooth, diagnosed with apical periodontitis[7], should be accessed and irrigated with either 5% NaOCl +3% H_2O_2[7] or 5.25% NaOCl[8] and PeridexTM (Procter & Gamble, Cincinnati, OH)[6]. An antimicrobial agent (either an antibiotic such as metronidazole+ ciprofloxacin[7] or ciprofloxacin+ metronidazole+ minocycline[6] or Ca $(OH)_2$[8] should be then applied into the root canal system, and the access cavity is sealed. After an average of 3 weeks, in the absence of symptoms, the tooth is re-entered, the tissue is irritated until bleeding is started and a blood clot produced, and then MTA is placed over the blood clot[6], and the access is sealed. Within the next 2 years a gradual increase in root development can be observed.[6-8]

Case report

A 18-year-old boy was referred to the Department of Conservative Dentistry of Govt Dental College, Calicut for evaluation of upper front teeth. The boy had reported slight discomfort region for 1 month, but he was asymptomatic during the examination visit. He had a history of trauma 9 years ago. On clinical examination, Ellis class 1V fracture of 11, 21, 22 was noted with discolouration of these teeth. A sinus tract was not present. Diagnostic testing on cold and electric pulp testing showed no response. Percussion test


Pre operative


Triple antibiotic paste


Sealed with cavit


Reopened after 11 days and paste removed with naocl


Bleeding induced


MTA placed

revealed mild pain. The periodontal probings were within normal limits for the tooth. Periradicular radiographic examination revealed that tooth 11, 21, 22 had an incomplete apex and a periradicular radiolucency.

Rubber dam isolation was done and access cavity was made. The root canal was irrigated with 5.25% NaOCl and replaced every 5 minutes for a total of 30 minutes. A mixture of ciprofloxacin, metronidazole, and minocycline paste was placed into the root canal with a lentulo spiral, and the access cavity was closed with Cavit. The patient returned 11 days later. The patient was asymptomatic.

The root canal was slowly flushed with 10 mL of 5.25% NaOCl and continuously irrigated with the same solution for 15 minutes. The root canal was dried with paper points. Once the canal became dry revascularisation can be done with a sterile 23 guage needle or sterile endodontic file. A stopper can be placed at 2mm beyond the working length in the needle or file and with sharp strokes it can be pushed beyond the confines of the canal into periapical tissue to intensionally induce bleeding into the canal. When frank bleeding is evident at the cervical region a tight cotton pellet can be inserted to a depth of 3-4 mm into the canal for 10 minutes to allow the formation of clot in the apical 2/3 of the canal. After that the access opening can be restored with mineral trioxide aggregate (MTA) (Dentsply Tulsa Dental, Tulsa, OK) followed by intermediate restorative material (IRM) (Caulk Dentsply, Milford, DE). A radiograph taken 3, 6, 9 months after MTA placement revealed a slight increase of the thickness of the root canal wall and continued development of the apex. The IRM was replaced with a bonded resin restoration.

Discussion

The traditional approach for treating cases of a necrotic immature permanent tooth would have been either apexification with $Ca(OH)_2$ or MTA[3-4], but it does not lead to a further thickening of dentinal walls or an increased root length. A more conservative approach would be to perform a regenerative procedure by not instrumenting the root canal and instead using only copious irrigation and application of antimicrobial agents to preserve any remaining vital pulp tissue in the apical region. The outcome of the conservative treatment depends on the type and duration of the infection, state of the pulp at the time treatment is started, host and the size of the open apex.[8,9]

Mechanism of regeneration

1. Few vital pulp cells remain at the apical end of the root canal and might proliferate into the newly formed matrix and differentiate into odontoblasts under the organizing influence of cells of HERS[10]. The newly formed odontoblasts can lay down atubular dentin at the apical end, causing apexogenesis (elongation of root), as well as on lateral aspects of dentinal walls of the root canal (reinforcing and strengthening the root).

2. The multipotent dental pulp stem cells are present in permanent teeth and might be present in abundance in immature teeth[11]. These cells from the apical end might be seeded onto the existing dentinal walls and might differentiate into odontoblasts and deposit tertiary or atubular dentin.

3. The stem cells in the periodontal ligament[12,13] can proliferate, grow into the apical end and within the root canal and deposit hard tissue both at the apical end and on the lateral root walls. The evidence in support of this hypothesis is presented by documentation of

IRM placed | Pre operative | 3 months | 6 months

cementum and Sharpey's fibers in the newly formed tissues.

4. Presence of stem cells in the apical papilla or in bone marrow.

Instrumentation beyond the confines of the root canal to induce bleeding can also transplant mesenchymal stem cells from the bone into the canal lumen and these cells have extensive proliferating capacity.[14,15]

5. The blood clot itself, being a rich source of growth factors such as platelet-derived growth factor, vascular endothelial growth factor (VEGF), platelet-derived epithelial growth factor and tissue growth factor, could play an important role in regeneration. They could stimulate differentiation, growth and maturation of fibroblasts, odontoblasts, cementoblasts from the immature, undifferentiated mesenchymal cells in the newly formed tissue matrix.[16]

The limitations of revascularization are-

- Long-term clinical results are as yet not available.
- It is possible that the entire canal might be calcified, compromising esthetics and potentially increasing the difficulty in future endodontic procedures if required.
- In case post and core are the final restorative treatment plan, revascularization is not the right treatment option because the vital tissue in apical two thirds of the canal cannot be violated for post placement.

Conclusion

Revascularization induced apexogenesis offers several advantages over other established treatment protocols for the management of immature, infected nonvital teeth. However, longer case series with longer follow-up period is required to establish it as the standard protocol for management of such teeth.

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Multiple roots in premolars - a brief review

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Abstract

Premolars are transitional teeth, with the first premolar reflecting the transition from the canine, and the second premolar showing the change toward the molars. They morphologically and functionally lie between canines and molars. Differential growth of the epithelial diaphragm in multirooted tooth causes the division of trunk into two or more roots. A knowledge of variation in the tooth morphology, especially root is essential for the clinician in detecting and treating dental pathologies. A pretreatment radiographic evaluation helps in detecting these variations or anomalies especially prior to extractions and endodontic therapy.

In this article we present a few cases of mandibular second premolars with two roots and maxillary first premolars with three roots, and a brief review of literature has been attempted.

Introduction

Premolars, positioned anterior to molars are called bicuspids in the sense that they have two well developed cusps. The term 'bicuspids' hold true only in the case of maxillary premolars where there are two well developed functional cusps, the buccal and palatal cusps, and not for mandibular premolars, as the first mandibular premolar does not have a well developed lingual cusp, and in case of second premolar, there are two or three cusps varieties. Usually maxillary first premolars have two roots and root canals. Maxillary second premolars, though less frequently, can have two roots and two pulp canals. But the mandibular premolars usually have only one root. Various reports show that mandibular first premolars can present with bifurcated roots, or less frequently two roots. But the incidence of bifurcated root or two roots in mandibular second premolars are very rare.

In this study five hundred extracted premolars each collected from different dental clinics/ hospitals in Kerala were evaluated for supernumerary roots. This article present a few cases of mandibular second premolars with two roots and maxillary first premolars with three roots.

Discussion

Premolars, positioned anterior to molars are called bicuspids in the sense that they have two well developed cusps.They assist canines and molars in function. The term 'bicuspids' hold true only in the case of maxillary premolars where there are two well developed functional cusps, the buccal and palatal cusps. The term bicuspids cannot be applied for mandibular premolars, as the first mandibular premolar does not have a well developed lingual cusp, and in case of second premolar, there are two or three cusps varieties.

The two mandibular premolars do not resemble each other nearly as much as do their maxillary counterparts. They are transitional teeth, with the first premolar reflecting the transition from the canine, and the second premolar showing the change toward the molars. The first premolar has a diminutive lingual cusp which is normally non-functional, so that its morphology and role in mastication parallel those of the canine. The second premolar often has two lingual cusps, and more closely approximates a small molar in structure and function. The slope of the marginal ridges is also transitional. The first premolar exhibits a slope which is more similar to that of anterior teeth, and the second premolar displays a more horizontal angulation like other posterior teeth. Usually maxillary first premolars have two roots and root canals. Maxillary second premolars, though less frequently, can have two roots and pulp canals. But the mandibular premolars usually have only one root. Various reports show that mandibular first premolars can present with bifurcated roots, or less frequently two roots. But the incidence of bifurcated root or two roots in mandibular second premolars are very very rare.

The advances in the dental research have paved light to the molecular aspects of odontogenesis and many genes responsible for the variation from normal or anomalies are now well documented.

A brief review of literature supports the above statement that the incidence of two well formed mesial and distal roots in mandibular second premolars are very rare, especially in Indian population. Anomalies are rare, although a root bifurcation into buccal and lingual branches is sometimes seen.

Differential growth of the epithelial diaphragm in multirooted tooth causes the division of trunk into two or more roots. During general growth of enamel organ the expansion of its cervical opening occurs in such a way that long tongue like extensions of the horizontal diaphragm develop. Two such extensions are found in the two rooted Mandibular second premolar.. Before division of the root trunk occurs, the free ends of these horizontal epithelial flaps grow toward each other and fuse. The single cervical opening of the coronal enamel organ is then divided into two openings.

The premolars appeared to have either a single root or two roots , but three-rooted maxillary first premolars

Fig 1. Mandibular II premolar

Fig 2. Mandibular II premolars with two

Fig 3. Mandibular II premolar

Fig 4. Maxillary I premolar with well developed three roots

Fig 5. Maxillary I premolar with three roots

A. Premolars with supernumerary roots

with mesiobuccal, distobuccal and palatal roots were detected in our study. The premolars were classified as (A) single rooted, (B) two-rooted with a Tomes' root form, or (C) two-rooted with fully developed mesial (or mesiobuccal) and distal roots, (D) three rooted. A Tomes' root refers to a condition where the mesial root surface is deeply grooved and a mesiobuccal and a distolingual root is formed with various degrees of separation (Wood et al., 1988; Turner et al., 1991). The Tomes' root is part of a morphogenetic continuum from a single- to a double-rooted tooth (Wood et al., 1988).

A recent analysis of the morphology of mandibular premolars in man and in Plio-Pleistocene hominids (Wood et al., 1988) showed that two-rooted forms have been common in certain ancestral species of modern man. It seems that all species in the genus Australopithecus have had two-rooted mandibular premolars with a high frequency of Tomes' root in Australopithecus africanus and Australopithecus robustus. Since the root morphology of the mandibular premolars is among the criteria which are used in the classification of fossil humans (Wood et al., 1988), it is worth noting that two-rooted forms seem to occur frequently in individuals of modern Homo sapiens with a 45,X or 45,X/46,XX chromosome constitution. This suggests that some caution must be exercised in the use of classifications relying on single morphological features such as root form.

It has been suggested that an abnormal amount of chromatin may disturb the growth of the transverse processes of the root sheath and delay formation of root furcation (Jaspers and Witkop, 1980; Witkop et al., 1988). This proposal is based on the reduced mitotic activity and high frequency of taurodontism found in Down's syndrome with an extra chromosome 21 (Jaspers, 1981).

Mosaicism refers to a chromosome abnormality where the cells of an individual show two or more different karyotypes (Therman, 1980). Chromosomally aberrant cell lines result usually from a mitotic nondisjunction and subsequent loss of chromosomes. Mosaics involving an abnormal number of the X chromosome are the most common. Low-grade mosaicism may not affect the phenotype, but with an increasing proportion of the abnormal cell line, phenotypic effects begin to appear (Therman, 1980). It was recently found that two-rooted mandibular premolars occur frequently in 45,X females (Varrela, 1990). A more detailed analysis of the morphology of two-rooted premolars in 45,X females revealed that teeth with a mesiobuccal and a distolingual form as well as teeth with fully developed mesial and distal roots were present (Varrela, 1992). The development of two-rooted forms in 45,X individuals suggests that the lack of the second sex chromosome is associated with molarization of the mandibular premolar roots.

A knowledge of variation in the tooth morphology, especially root is essential for the clinician in detecting and treating dental pathologies. A pretreatment radiographic evaluation helps in detecting these variations or anomalies especially prior to extractions and endodontic therapy.

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Pediatric mandibular fractures: Children are not just tiny adults

* Sheela Sreedharan, ** Kavita Kumar

Abstract

Management of mandibular fractures in children is age dependent. Conventional methods of open reduction and fixation are better avoided in children as they hinder the growth of the mandible resulting in various facial deformities and TMJ dysfunctions. Hence they should be managed as non invasively as possible. Various methods of conservation management are now available for management of pediatric mandibular fractures. These should be substantiated with good long term follow up to avoid complications associated with mismanaged mandibular fractures. This article reviews the various considerations in pediatric mandibular fractures and throws light on its management.

Introduction

All dentists who deal with children will sometimes encounter cases of acute orofacial injuries in their practice. Of the various orofacial injuries, fractures form a very significant part. While considering fractures of the orofacial complex, nasal bone is the most frequently fractured bone. This is followed by fracture of the mandible.

The principles involved in the treatment of mandibular fractures are the same whatever be the age of the patient. However in the treatment of children the techniques used are necessarily modified with certain anatomical, physiological and psychological factors in mind. The small size of the jaw, existing active bony growth centres, and the contained overwhelmingly crowded primary teeth with permanent tooth buds located in great proximity to the mandibular and mental nerves, all significantly increase therapy related risks of pediatric mandibular fractures. Intact active mandibular growth centres are important for preserving mandibular function which have a significant influence on future facial development. Thus restoration o mandibular continuity after fracture is not only important for immediate function but also for future growth of the craniofacial complex.

Accordingly the goal of the treatment is to restore the underlying bony architecture to its preinjury position in a stable fashion as non invasively as possible with minimal residual and functional impairment.

Incidence

Fractures of the facial bones in children are relatively infrequent. There is a fairly general agreement that only about 5% of facial fractures occur in children and below the age of five years the incidence is significantly less. Fractures of the nasal bone and of the mandible account for the great majority of facial fractures in children. Of the various sites condyle is the most frequently fractured. This is followed by symphysis, angle and body of the mandible. Fractures of the body and angle are initially infrequent but then increase with age.[1]

Important considerations in pediatric mandibular fractures

In order to account for the disparity of incidence in facial fractures between children and adults a number of factors have to be considered.

First of all, parental supervision during the preschool years, reduces the risk of serious injury. Although falls are frequent, they are usually from a small height. The second consideration is the difference in the size of the cranium and facial skeleton in adults and children. In young children (less than 5 years) the face is in a more retruded position relative to the skull (fig 1). Therefore there is a lower incidence of midface and mandibular fractures and a higher incidence of cranial injuries. With increasing age and facial growth, in a downward and forward direction, the midface and mandible become more prominent (fig 2). In addition to this the incomplete dentition provides strength to the maxilla and mandible, as the presence of tooth buds within the jaw increases the stability and elasticity of the bone. Also the abundance of cartilage and cancellous bone, low mineralization and underdeveloped cortex, along with the more flexible suture lines and indistinct corticomedullary junction confer greater elasticity and flexibility to the pediatric facial skeleton. The thick layer of adipose tissue that overlies much of the pediatric facial skeleton, and the fat pads that surround the upper and lower jaws, also help to protect these bones.

The common causes of mandibular fractures in children are road traffic accidents, and falls. Less common causes are sporting activities and play.[2]

Diagnosis

The clinical signs and symptoms of a fractured mandible in a child are same as that in an adult: pain, swelling, trismus, derangement of occlusion , sublingual hematoma, step deformity, deviation, loss of sensation due to nerve damage, bleeding, ecchymosis, tenderness, movement restriction, open bite and trismus.

Through examination though difficult, must be done in all cases of pediatric maxillofacial trauma. Swelling

Fig 1 Pediatric skull with retruded jaws relative to skull

Fig 2. Adult skull with prominent jaws relative to skull.

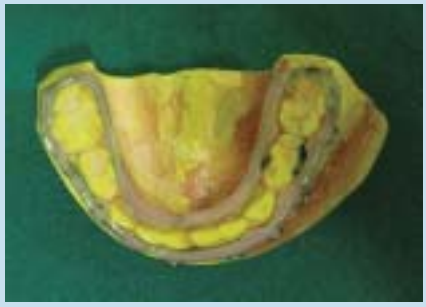

Fig 3. Pre fabricated acrylic splint

and ecchymosis of the face, specially the auricular area, may be a clue to underlying fractures. A chin laceration in a young child usually indicates a superiorly directed force which often results in condylar damage. Examination of the jaw during mouth opening or closing, may reveal deviations or limited mobility. Trismus may also be evident and may be due to muscle spasm and pain. Intraoral examinations may reveal lacerations or a hematoma. This is very helpful in examining fractures of the dentoalveolar complex. Examination of the ear may be rewarding in diagnosing condylar fractures. Gentle palpation must be applied all over the bony surfaces of the mandible. Step offs and instability may be apparent. A fracture of the body of the mandible may affect the inferior alveolar nerve causing numbness of the teeth and chin. Displaced fractures may also affect the lingual and the long buccal nerve causing numbness over the area supplied by them. Lastly assessment of occlusion must be done.

Radiographs play a very important role in the diagnosis of mandibular fractures. Plain radiographs are less helpful in children than in adults due to unerupted tooth buds obscuring fracture lines and an underdeveloped cortex which leads to difficulty in visualising fractures. An Ortho pantomograph is the study of choice but other views including lateral oblique, Postero Anterior view, Townes view, mandibular occlusal view and an Intra Oral Periapical view can also be used. CT scan however is the best diagnostic tool and with the help of 3D CT scan diagnosis is very accurate thus helping the clinician to provide better treatment to children.

Management

The general principles of management of pediatric mandibular fractures are similar to those in adults namely, fixation of the fragments in their correct alignment, with the teeth in occlusion until union has taken place. However the special features discussed previously indicate some modifications in the management of pediatric mandibular fractures.

Emergency management

The provision of an adequate airway and arrest of haemorrhage are the emergency measures that apply to patients with facial injuries, whatever be their age. However major anatomical and physiological changes continue to occur as the child grows older. Thus a three year old cannot be treated in the same way as a ten year old. Due to the small size of the airway in a child, laryngeal edema or retropositioning of the tongue may produce sudden obstruction. In most instances, the presence of any significant upper airway obstruction, is an indication for early intubation, although the insertion of a tongue suture permits forward traction and immediate restoration of the airway. Because of the child's small blood volume hypovolemia may result even with the loss of apparently insignificant amounts of blood. When hypovolemia is present it is important to restore blood volume before operating.

Once the airway breathing and circulation are established, the management of mandibular fractures is done. The specific treatment of mandibular fractures depends on the location of fracture lines, degree of bony displacement, occlusion status and dentition status of the child. Rowe in 1969 divided children into four groups based on the status of their dentition at the time of trauma.(Table I)[3]

Infancy to 2 years old children

Very little anchorage if any can be obtained from the teeth because they are unerupted or incompletely formed in most instances. There are two categories of injuries to consider from the point of view of immobilization. First of all when the fracture line is in the tooth bearing part of the mandible, usually in the symphysis region. The fracture should be treated essentially as an edentulous problem and the technique described by Mac Lennan (1956) using prefabricated gunning type splints lined with softened gutta percha can be used for immobilization, which is done after manual reduction. The splint is retained by two circumferential wires on either side of the fracture line.[4] When the fracture involves the angle of the mandible further immobilization is achieved using the above described splint which is fixed to both occlusal surfaces using circummandibular wires and wires through the piriform fossa. In cases of condylar fractures, the highly vascular and highly osteogenic condylar environment

Fig 4. Mandibular Cap Splint

Fig 5. Orthodontic components for stabilization

necessitates the development of active jaw function which is helped by analgesics as a means of combating joint ankylosis. [5]

2-4 years old

At this age provided there are sound primary teeth present, interdental eyelet wiring can be used. If there are gaps in the dentition an arch bar may be needed. Mini arch bars attached with resin may also be used to treat nondisplaced fractures in this age group, thus avoiding the need for immobilization of the mandible. Several studies have recommended the use of prefabricated acrylic splints (Fig 3) as a treatment for pediatric mandibular fractures. These splints are more reliable than open reduction or IMF techniques with regard to cost effectiveness, ease of application and removal, reduced operation time, maximum stability during healing period, minimal trauma to adjacent anatomic structures and comfort for the young patients.

If the fracture is within the tooth bearing area of the mandible, a single one piece lower cap splint (Fig 4), is a good method of fracture reduction, since immobilization of the lower jaw is avoided. If there is any doubt about the security of the splint then it can be reinforced by means of two circumferential wires.

5-8 years old children

It is between these ages that greatest problems arise with regards to fixation of mandibular fractures. The anterior teeth are of little or no use as the roots are either resorbed as in the case of primary teeth or incompletely formed as in the case of permanent teeth. Reliance must therefore be place primarily on the primary molars, although these may be carious or be extracted prematurely. These difficulties can generally be overcome by constructing partial maxillary and mandibular Gunning type splints with occlusal blocks, the exact construction being dependent on the exact position of the tooth loss.

An alternative method for closed reduction is the one recommended by Laster et al. They described a new treatment modality based on nickel titanium (NiTi) staples which can be inserted in a relatively non invasive manner and their eventual removal if needed is as quick as their insertion due to the fact that they are not osseointegrated.[6]

Other studies recommend the use of orthodontic components (Fig 5) like orthodontic brackets, orthodontic resin ,orthodontic rubber elastics and modified orthodontic splint appliance where bands are applied on to the primary second molars with round stainless steel wire soldered to them on the right and left sides.[7]

9-11 years old

In patients in this age group the development of the permanent incisors and 1st molars has proceeded to a point where they can safely be applied for fixation which can be done by means of cap splints and arch bars. In cases of severely comminuted fractures, open reduction can be done using mini plates and screws, the use of which is considered safe only in the symphyseal and parasymphyseal region. Eppley in 2005 recommended the use of resorbable osteosynthesis plates and screws for children. The avoidance of secondary implant removal operations reduces not only the cost but also the physical and psychological trauma. This method does carry the risk of injuring permanent tooth buds, however it is minimal as the drill hole and tapping of the screw penetrate only the outer cortex and even if a screw tip encroaches on a tooth bud, its tip is blunt and non penetrating.[8]

Condylar fractures

A special mention of condylar fractures is needed as the condyles are very important for the proper growth of the mandible and the face. Condylar fractures can be classified into 3 types

1. Intracapsular fractures involving the articular disc.
2. High condylar fractures above the sigmoid notch which tend to be medially displaced.
3. Low condylar or subcondylar fractures which tend to be greenstick injuries with minimal displacement and are the most common type of pediatric mandibular fractures.

When it comes to management of condylar fractures conservative management is the mainstay of treatment. Jaw function should be encouraged to promote growth by virtue of high regenerative and remodelling potential of condylar cartilage in young children. In extreme cases a brief period of intermaxillary fixation must be considered. In cases where there is significant fracture

Table I

Age in Years	Dental Development
0-2	Eruption of primary teeth incomplete
2-4	Before the roots of primary incisors show marked resorption, although many of the permanent teeth are partly formed
5-8	Before resorption of primary molars is advanced or the roots of the permanent incisors are adequately developed
9-11	After adequate formation of roots of permanent incisors and molar teeth, but before eruption of premolars.

displacement of the condylar process with extensive tissue injury, a myofunctional appliance can be used to help offset subsequent scarring. Surgery has a very limited role in the management of condylar injuries. Certain situations where it may be considered are given in table II.[5]

Complications

Rowe in 1969 reported that injuries to children younger than 3 years produce severe deformities , injuries to children aged 3-6 years result in moderate deformities and injuries to teenagers heal in a similar fashion to adults. [3]

Post operative infection, malunion and non union are rare in children due to the greater osteogenic potential faster healing rates and less frequent requirement for open reduction. Late complications include damage to developing permanent teeth, TMJ dysfunction (recurrent subluxation, noise and pain, limited condylar translation, deviation on opening, ankylosis and growth disturbances (e.g. Secondary mid face deformity, mandibular hypoplasia or asymmetry). These are however seen only in severely comminuted fractures. Mac Guirts in 1987 in a follow up study of patients after childhood mandibular fractures revealed abnormalities of occlusion and dentition in 35% of the patients including avulsed teeth, non-vital pulp, and hypoplastic teeth.

The occurrence of complications also depends on the method of reduction used. In a recent study be Lampier et al in 2003, a lower complication rate was noted in cases treated by closed reduction than in those treated by open reduction. Rates of infection and wound dehiscence was also low in cases treated by closed reduction.[9]

Another parameter to be taken into consideration is the level of root development at the time of trauma. Ranta and Ylipaavaliemi in 1973 pointed out that teeth in which root development has already begun at the time of fracture appear to erupt normally however marked deformation of the crown and root occur in teeth located on the fracture line when calcification is still in progress at the time of fracture.[10]

Table II

Sl. No.	Considerations
1	Failed conservative therapy
2	Gross obstruction to mandibular movement caused by displaced condylar process
3	Pan facial trauma where the mandible is used to stabilize and help to guide the repositioning of the mid face.

Conclusion

The management of condylar fractures in children should be age dependent. Disruption of the periosteal envelope of the mandibular body may have unpredictable effects on growth. Thus closed reduction should be favoured as opposed to open reduction. The use of arch bars, eyelets, prefabricated splints, orthodontic components aid in this conservative management. Along with good treatment it is very important to follow up all cases of mandibular fractures. This will go a long way in preventing untoward sequale associated with improper healing. Growth in particular must be monitored as this bone houses important growth centres.

Thus age defined treatment along with long term follow up as the child grows is the key to reducing both the psychological and physical trauma associated with mandibular fractures. One must always remember that children are not just tiny adults!!!

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Anemia - a risk factor for periodontitis

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Abstract

Anemia is a condition where the erythrocyte count is less than that in normal persons, or Hb % is low, or both.

Anemia of chronic disease (ACD) has been described as one of the most common forms of anemia observed in clinical medicine. This occurs in chronic infections , inflammatory conditions or neoplastic and is not due to marrow deficiencies or inadequate iron stores and vitamins. Periodontitis is one of the most common chronic diseases of humans resulting in the destruction of the supporting tissues of the teeth. In the past decades, it has been associated with various chronic diseases like diabetes, coronary heart diseases.

The following study was performed to investigate whether periodontitis patients showed signs of anemia as noted in other chronic diseases.

Introduction

Periodontitis is one of the most common diseases of humans. It is a chronic infectious condition of the supporting tissues of the teeth and is caused by the subgingival colonization of specific pathogens to susceptible subjects. The influence of host factors on the pathogenesis and progression of periodontal diseases is widely recognized. Offenbacher has reviewed the mechanisms by which physical, environmental and social host stresses affect and modify disease expression. Models of pathogenesis have been presented in which systemic disorders affecting neutrophil, monocyte and /or lymphocyte function result in altered production or activity of cytokines and inflammatory mediators. These changes in the host response to bacterial challenge may manifest clinically as localized or generalized increase in bone and attachment loss. Perhaps the most vivid examples of such host modification of periodontal disease expression are seen in individuals with genetic neutrophil defects.[1]

Periodontal destruction probably results from the action of various toxic products released from specific pathogenic subgingival plaque bacteria , as well as from the host responses elicited against plaque bacteria and their products. The inflammatory response may result in gingival ulceration around the tooth which can allow intact bacterial cells or their products including lipopolysaccharides, peptidoglycan fragments, and hydrolytic enzymes into the systemic circulation. It is also known that the host response to periodontal infections results in the local production of cytokines and biological mediators including interleukins and prostaglandins, as well as systemic responses such as induction of serum antibodies.[2]

In the past decades, periodontitis has been associated with various chronic diseases like diabetes, coronary heart diseases. Anemia is a condition where the erythrocyte count is less than that in normal persons, or Hb % is low, or both.

Anemia of chronic disease (ACD) has been described as one of the most common forms of anemia observed in clinical medicine. ACD is defined as anemia occurring in chronic infections, inflammatory conditions or neoplastic disorders that is not due to marrow deficiencies or other diseases and occurring despite the presence of adequate iron stores and vitamins.[3]

Aim

The purpose of this study was to investigate whether periodontitis patients showed signs of anemia.

Criteria for patient selection

Study population

A total of 40 subjects were included for the study.

Group I included 20 patients, 10 males and 10 females with clinically periodontium, no history of any systemic diseases, no recent history of any trauma or tooth extractions. and nonsmokers. They had a CPI score <2.

Group II included 20 patients, 10 males and 10 females, with periodontitis and CPI score > 2. All the other criteria remained the same as in group I.

Methodology

Informed consent was obtained from all the patients. 2.5 ml of venous blood was obtained by venipucture in the anticubital fossa without excessive venous stasis. In standardized and automated procedures, number of erythrocytes, mean corpuscular volume of erythrocytes (MCV), hemoglobin concentrations, Erythrocyte sedimentation rates were measured.

The hematocrit, as an overall measure of red blood cell volume in whole blood was calculated as the number of erythrocyes times the MCV. Further, the mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration was calculated.

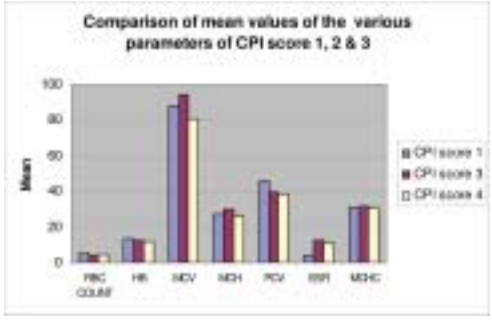
Statistical Analysis

Comparison of CPI values and the variables were done using ANOVA.

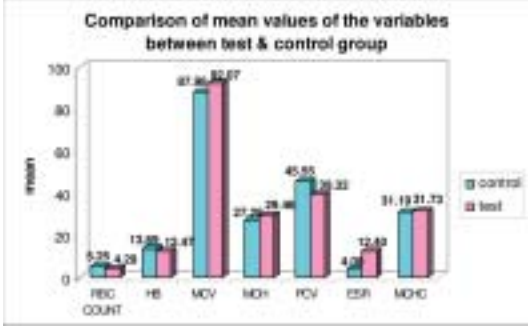
Variables	*F*	*P value*
RBC count	31.633	.000
Hb	2.616	.087
MCV	4.100	.025
MCH		.040
PCV	18.269	.000
ESR	20.958	.000
MCHC	0.177	.838

Results

There was significant difference in the mean values of CPI scores for RBC, MCV, MCH,PCV & ESR

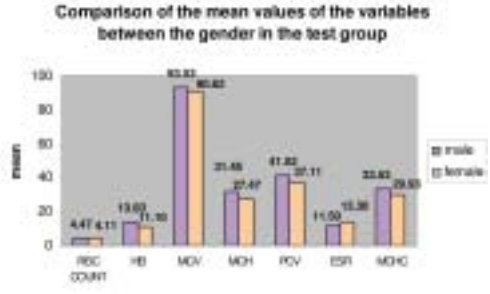


A significant difference for RBC, Hb,PCV & ESR between test & control group.



Males showed higher RBCcount, PCV, Hb, MCV, MCH and MCHC compared to females.

Females showed higher ESR compared to males



Discussion

Epidemeological studies suggest that periodontitis is associated with an increased risk for systemic diseases like cardiovascular diseases, cerebrovascular ischemia and atherosclerosis. Preterm low birth weight infants has also been associated with destructive periodontal diseases.[4]

These associations indicate that periodontitis has systemic effects and that most likely (subtle) signs of s ystemic inflammation must be present.

The present study provides evidence that periodontitis has measurable systemic consequences. Mild to moderate anemia has been reported as frequently associated with many chronic diseases. Anemia has not been identified in the recent literature as a systemic consequence of periodontitis. Also Wakai etal (1999) have not observed a significant correlation between CPITN scores and hemoglobin levels.[5] A few early reports in the literature (Goldstein 1937, Siegel 1945, Lainson etal 1968, Chawla 1971[6]) have observed anemia in periodontitis but except for the report by Seigel (1945), the authors have been led to believe that perhaps anemia was one of the causes of destructive periodontitis rather than to regard this phenomenon as a consequence. Mild to moderate anemia has been reported as frequent manifestation of rheumatoid arthritis. In fact several other chronic diseases such as parasitic diseases, bacterial and fungal infections and neoplastic illnesses, anemia has been noted and hence it has been given the term anemia of chronic disease. The cause for ACD is most likely multi –factorial, however it is currently thought that proinflammatory cytokines from a given chronic disease process such as rheumatoid arthritis, may down regulate the eryhthropoiesis in the bone marrow. In particular, interleukin-1 (IL-1), IL-6 and tumour necrosis factor – alpha have been implicated as cytokines responsible for suppressing eryhthropoiesis. The current study indicates that periodontitis also needs to be considered as a chronic disease which may cause lower numbers of erythrocytes and consequently lower hemoglobin levels in a substantial number of patients. The pathogenesis of the current findings is most likely similar as reported for rheumatoid arthritis, i.e. depression erythropoiesis by systemically circulating proinflammatory cytokines resulting from a local chronic inflammatory process. In support of this concept are observed systemic levels of IL-6 in about 1/3 of patients with localized periodontitis and in about half of the patients with generalized periodontitis.[7]

Smoking is an important factor identified in the literature to affect erythrocytes and related parameters. At the same time, smoking is also a cofactor in the etiology of periodontitis. Hence it was deemed necessary to exclude smoker in the study group.

The ESR is a traditional parameter in medicine of any given inflammatory process. It provides a multifactorial measure of the systemic response to an infectious or inflammatory disease. A change in the plasma concentration of several inflammation related proteins may affect the rouleaux formation by erythrocytes, and subsequent sedimentation. However this parameter of inflammation seems to be of limited

Unpaired T test was used to compare the mean values of different variables between the test and the control groups.

Variables	*Group*	*N*	*Mean*	*Normal values*	*Pvalues*
RBC count	Test Control	20 20	5.249 4.288	4.5±7 4.2±7	.025
Hb	Test Control	20 20	13.690 12.465	14-18 12-16	.000
MCV	Test Control	20 20	87.979 92.072	82-92	.538
MCH	Test Control	20 20	27.281 29.461	27-32	.037
PCV	Test Control	20 20	45.550 39.320	45±7 42±5	.007
ESR	Test Control	20 20	4.000 12.400	1-3 4-7	.476
MCHC	Test Control	20 20	31. 185 31.732	33.3	.034
CPI Scores	Test Control	20 20	1 3.15		

Mean values of the variables between the two gender in the test group were analysed using unpaired T-test.

use as diagnostic tool in periodontitis to measure the systemic involvement. The hematocrit is one of the major factors influencing the ESR. Therefore one must realize that a possible elevation of the ESR due to the inflammatory process, may be marked through a depressed hematocrit, also as the result of the same inflammatory process.

Conclusion

PCV, number of erythrocytes and hemoglobin were significantly lower in periodontitis patients compared to controls.

There was a trend for higher ESR values in periodontitis patients compared to controls.

The MCV, MCH, MCHC were not different among the two groups.

Higher CPI scores were associated with lower RBC count, PCV, MCV, MCH and higher ESR.

In the test group , male patients showed higher RBC count, PCV, MCV, MCH and lower ESR compared to females.

The present study provides evidence that periodontitis, like other chronic conditions, may tend towards anemia, as the number of erythrocytes and levels of hemoglobin are lower in affected patients. These findings may be related to elevated levels of proinflammatory cytokines in the plasma of periodontitis patients.supressing erythropoiesis.

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Photodynamic therapy in the management of periodontal diseases: A review

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Abstract

Periodontitis is a biofilm of mixed aetiology. Periodontitis is conventionally treated with mechanical debridement procedures and adjunctive antimicrobial therapy. Photodynamic therapy (PDT) or photochemotherapy is a novel non-invasive therapeutic approach with increased pathogen and site specificity. PDT involves the use of a photosensitiser that is activated by exposure to light of specific wavelength, to form toxic oxygen species, thereby causing localised photodamage and cell death. Applications of PDT in Periodontics continues to evolve into a mature clinical treatment modality for treatment of periodontal diseases. PDT has been considered as a promising novel therapeutic approach for eradicating pathogenic bacteria in periodontal and peri-implant diseases.

Introduction

Periodontitis is one of the most prevalent disease in the oral cavity caused by periodontopathic bacteria. The main objective of periodontal therapy is to eliminate deposits of bacteria and bacterial niches by removing supragingival and subgingival biofilm. The current treatment modality involves conventional mechanical therapy or the non – surgical phase which involves scaling and root planning, removal of local irritation factors such as decay, overhangs, ill – contoured crowns, mal – aligned teeth and adjunctive microbial regimen. If the initial therapy does not significantly improve the periodontal condition, periodontal surgery is considered in order to help resolve the disease process. However it has been demonstrated that conventional mechanical debridement procedures cannot completely eliminate the entire pathogenic flora due to anatomical complexity of roots, which may contain furcation areas and concavities, especially in deep periodontal pockets and bacteria involving surrounding soft tissues[1,2]. Potential periodontal pathogens such as Aggregatibacter actinomycetumcomitans and Porphyromonas gingivalis are capable of disrupting host epithelial cells and invading into deeper periodontal tissues[3]. Thus elimination of these bacteria from advanced diseased sites by mechanical debridement procedures alone have proven to be less efficacious.

Conventional mechanical procedures augmented with local/systemic antimicrobial agents (mechano-chemotherapy) further suppress the pathogens and increase the benefits of the former. However as these agents are difficult to maintain at therapeutic concentration in the oral cavity, and can be rendered ineffective due to emergence of resistant micro-organisms and shift of microflora due to their extended and indiscriminate usage, and development of hypersensitivity reactions and side – effects, their use is limited[4].

Phototherapy has been considered as novel treatment approach in Periodontics[5]. Dental lasers have been used as an effective means of decontamination of periodontal pockets due to their high bactericidal properties and have demonstrated effective killing of oral pathogenic bacteria associated with peri–implantitis and periodontitis. High level lasers possess two major drawbacks. (1) irreversible thermal damage to the surrounding periodontal tissues (2) unexpected side – effects aka excessive ablation/thermal coagulation, carbonization/necrosis of root, gingival connective tissues, bone and pulp tissues, depending on the type of laser employed[6].

More recently, an innovative non – invasive treatment modality called photodynamic therapy (PDT), has been employed to treat periodontal diseases, which uses low level laser light. Unlike the high level lasers, PDT is pathogen and site specific. Photodynamic therapy can be defined as eradication of target cells by reactive oxygen species. Photodynamic action describes a process in which light, after being absorbed by dyes, sensitizes organisms for visible light induced cell damage called photolethal sensitisation[7].

Evolution And Medical Application

In 1904, Jodlbaner and Von Tappeneir coined the term photodynamic to describe oxygen dependant chemical reactions induced by photosensitisation, which could inactivate bacteria. In 1978, Daugherty et al, successfully applied this novel technique for treatment of different cancers. In 1999, US Food and Drug Association approved PDT as a treatment modality for treatment of precancerous skin lesions of face/scalp.

While PDT is relatively new to dentistry, the same has been used in various medical applications for over a decade. PDT is currently successfully being used for treatment of some form of cancers, macular degeneration, various dermatological applications and

Table I: *Commonly used photosensitisers in PDT*

Phenothiazine dyes	Methylene blue Toluidine blue O Acridine orange
Phthalocyanine dyes	Aluminium disulphonated phthalocyanine Cationic Zinc (II) phthalocyanine
Chlorines	Chlorine e6 Stannous (IV) chlorine e6 Chlorine e6-2.5 N-methyl-d-glucamine Polylysine and polyethylamine conjugates of chlorine e6
Porphyrines	Hematoporphyrine HCl Photofrin5- aminolevulinic acid (ALA) Benzoporphyrin derivative (BPD)
Xanthenes	Erythrosine
Monoterpenes	Azulene

plasma pooling disinfections.

Mechanism of Action

PDT basically involves 3 non – toxic ingredients[5,7] (1) Visible harmless light (2) photosensitiser (3) oxygen. When a photosensitiser is irradiated with light of specific wavelength it undergoes transition from a low – energy ground state(S_0) to an excited singlet state(S_1). Subsequently the photosensitiser may decay back to its ground state, with emission of florescence or may undergo transition to a higher energy triplet state(T_3). The longer lifetime of triplet state enables the interaction of excited photosensitiser with the surrounding molecules. The triplet state photosensitiser follows two different pathways to react with biomolecules.

Type I reactions involves hydrogen atom abstraction or electron transfer reactions between the excited state of the photosensitiser and an organic substrate molecule of the cells, which produces free radicals and radical ions. These highly reactive free radical species react with endogenous molecular oxygen to produce highly reactive oxygen species such as superoxides, hydroxyl radicals and hydrogen peroxide which are harmful to the cell membrane integrity.

In the type II reaction, the triplet state photosensitiser reacts with oxygen to produce an electronically excited and highly reactive state of oxygen known as singlet oxygen ($^1O_2$) which can react with large number of biological substrates as a result of its high chemical reactivity, inducing oxidative damage and ultimately lethal effects upon the bacterial cell by damaging the cell membrane and cell wall[8]. Singlet oxygen has short lifetime in biological systems(<0.04 μs) and a very short radius of action (0.02 μm)[9]. Thus due to limited migration of singlet oxygen from its site of formation, the reaction takes place within a limited space, leading to a localised response and making it ideal for application to local sites without affecting distant molecules, cells and organs.

The bactericidal effect can be attributed to (1) DNA damage (2) Damage caused to cytoplasmic membrane of bacteria by cytotoxic oxygen species such as inactivation of membrane transport systems, and enzymes, alteration of cytoplasmic membrane proteins, disturbance of cell wall synthesis and loss of potassium ions from bacterial cells.

Table II: *Applications of PDT in Periodontics*

1. With scaling and root planing, when purulence and generalised bleeding on probing present.
2. Management of aggressive periodontitis
3. Refractory and recurrent case of periodontitis
4. Disinfection of class II and III furcation involvement and deep vertical defects during flap surgery
5. Disinfection of roots and furcation areas during regenerative surgery
6. Peri – implantitis treatment
7. Maintenance phase

Table III: *Advantages of PDT in periodontal therapy*

1. Development of resistance to PDT is less.
2. PDT is non invasive, local therapy, hence increased patient acceptance.
3. PDT offers thorough elimination and eradication of pathogens in inaccessible areas of periodontal pockets.
4. The risk of bacteremia after periodontal debridement can be minimised.
5. No need to prescribe antibiotics, hence no possibility of side effects.
6. No need to anesthetise the area.
7. Rapid elimination of periodontopathic bacteria in less than 60 seconds (7).

Photosensitisers

Various chemicals have been used as photosensitizing agents in PDT (Table I) [10]. For antimicrobial PDT, methylene blue and Toluidine blue O are very effective for the inactivation of both gram positive and gram negative periodontopathogenic bacteria.

Light Sources

A laser or visible light source is used to activate the photosensitisers. Early laser systems were complex and expensive. Subsequently diode laser systems that were easy to handle, portable and cost effective were developed.

Non – laser light sources such as light emitting diodes which are cheaper, smaller and of lighter weight were used. More recently visible light sources began to be employed[7]. Human tissues transmit red light efficiently at wavelength of 630nm and 700nm corresponding to penetration depths from 5mm to 15mm respectively. Advantages accrued are as follows: (1) useful in visualising the target area (2) localising the lethal photosensitisation without damaging the adjacent host tissues and the operator.

Currently however the light sources employed are of specific wavelengths: He – Ne lasers(630nm) transmitting red light, gallium aluminium arsenide diode

Fig 1 Periowave™ treatment kit.

Fig 2 Periodontal pocket filled with adequate photosensitizing solution.

Fig 3 Irradiation with the nonthermal diode laser

(Photos courtesy: Dr Veronique Benhamou, McGill University Faculty of Dentistry)

lasers(630 – 690nm) transmitting infra red light, and argon lasers(488 – 514nm) transmitting blue light and non laser light sources such as LED's[5].

PDT and Periodontitis

Polysaccharides present in extracellular matrix of oral biofilm are highly sensitive to singlet oxygen and susceptible to photodamage. Gram positive bacteria are more susceptible to photoinactivation as they have a relatively porous cytoplasmic membrane, peptidoglycans and lipoteichoic acids outside the cytoplasmic layer allow the neutral/anionic photosensitiser to bind efficiently and diffuse into sensitive sites. In gram negative bacteria , the structure of the cytoplasmic membrane is more complex. They form a structural and functional barrier between the cell and its environment, thus making it difficult for the dye to gain access[11]. The diffusion can be enhanced by (1) linking the sensitiser to a polycationic molecule (poly L- lysine chlorine, polymyxin B – nonapeptide). These weaken the intermolecular interactions of the lipopolysaccharide constituents and render it permeable to drugs; (2) use of membrane active agents (tris EDTA), which release LPS or the induction of competence with a sensitised pathogen; and (3) conjugating the sensitiser to monoclonal antibodies that binding to cell surface specific antigens.

The role of virulence factors in the pathogenesis of periodontal diseases are well documented. PDT inactivates the bacterial virulence factors. Following exposure of P Gingivalis to low energy He – Ne laser(632nm) and TBO(25um/ml), the activity of IL–1 and LPS secretion from human peripheral mononuclear cells were significantly reduced. Furthermore there was substantial light dose dependant decrease in proteolytic activity of P gingivalis[12].

Clinical studies exploring the effect of photodynamic therapy in the treatment of periodontal diseases have yielded conflicting results. Braun et al[13] investigated the use of PDT as an adjunct to non-surgical therapy in the treatment of chronic periodontitis. After 3 months the sites treated with PDT showed significant improvement in terms of gingival bleeding, probing depth, gingival recession, attachment level and gingival crevicular fluid flow rates. However, in another randomized controlled clinical trial where PDT was used in periodontal maintenance patients, only bleeding on probing was significantly reduced compared to other parameters at six months interval[14]. The use of PDT in aggressive periodontitis patients was investigated by de Oliveira et al, who found similar clinical outcomes at 3 months evaluation for PDT and non-surgical treatment[15]. A recent study evaluated the short-term effects of photodynamic therapy on periodontal status and glycemic control of patients with diabetes[16]. Forty-five patients with type 2 diabetes and moderate to severe chronic periodontitis were randomly assigned to one of the following three treatment modalities (15 subjects each): scaling and root planing (SRP) only, SRP plus systemic doxycycline, and SRP plus PDT. The plaque and bleeding scores, probing depth, clinical attachment level, and glycosylated haemoglobin (HbA1c) level were recorded at baseline and 3 months after periodontal treatment. No significant differences in periodontal parameters and glucose levels were detected among the three groups. Finally, a recent systematic review and meta-analysis of the effect of photodynamic therapy for periodontitis concluded that PDT as an independent treatment or as an adjunct to scaling and root planing was not superior to scaling and root planing alone[17]. Currently, clinical photodynamic therapy kits that include methylene blue are commercially available. (1) PERIOWAVE™ (Ondine Biopharma Corporation, Vancouver, Canada. This system uses a non-thermal diode laser with a uniform light diffusing tip having a wavelength of 670nm and a maximum power of 150mV along with methylene blue as the photosensitiser (2) HELBO® Photodynamic Systems (GmbH & Co. KG, Grieskirchen, Austria), which uses phenothiazine chloride as the photosensitiser along with. Light: HELBO® Theralite laser – diode laser of 670nm wavelength at 75mW[5].

Treatment Process

In the first step, the photosensitizing solution is directly administered into the periodontal pockets and around the gingival margin of the affected teeth. Each periodontal pocket must be treated individually. It is essential to flood the periodontal pocket to be treated with adequate photosensitizing solution. Too little

solution may negatively affect the results. Next, a low – intensity laser light is placed at the site and the area is illuminated for 60 seconds. Little or no patient discomfort has been reported[18].

Adverse Effects

Some adverse effects have been reported depending upon the type of photosensitiser used. (1) gingival ulceration, muscle necrosis, necrotising sialometaplasia of salivary glands were observed in rabbits following administration of disulphonated phthalocyanines(5mg/kg and 20J at 675nm)[19]. (2) Vesicle formation on tongue with oedema, cellular infiltration and reduction in number of vessels but muscle fibres remained intact with hematoporphyrin derivative[20].

Conclusion

The new strategy of using PDT is less traumatic and quicker in the treatment of periodontal diseases but it is still in the experimental stage of development and testing. Development of new photosensitisers, more efficient light delivery systems and further clinical studies are required to establish the optimum treatment parameters for PDT.

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Congratulations

President Elect of IDA Kerala State Dr.Santhosh Sreedhar is adjudged as the Best President in Rotary International District 3202 for the year 2009-10. He received the Award from District Governor Rtn. Dr. E. K. Sagadhevan during the Award Ceremony held on 27th June 2010.

Case report

Immediate removable partial denture using patient's own tooth

*Veena Raj V., ** Anandkumar R., *** K. Nandakumar

Abstract

Extrusion of a tooth means change in the normal position of the tooth in the occlusal or incisal direction within the same arch. It can be physiological or pathological.Pathologic migration refers to tooth displacement that results when the balance among the factors that maintains the physiological tooth position is disturbed by periodontal disease. In a periodontally compromised tooth, it can be treated by replacing with RPD, FPD or Implant. Fabrication of RPD with the same extracted tooth is one of the method can be adopted. This is a convenient technique to incorporate the patient's own tooth for the fabrication of a removable partial denture to overcome the "artificial look" of immediate denture. This is a time saving, esthetic and cost effective method for the teeth replacement following relectance to accept the conventional removable partial denture with the artificial teeth.

Introduction

Replacement of a natural tooth is essential to maintain the normal function, esthetics and to prevent migration of adjacent tooth. A tooth can be replaced by using a removable partial denture, fixed partial denture or implant supported prosthesis. But in compromised cases, the decision to replace tooth with the extracted tooth in the same position in RPD is a good option and can be implemented effectively.

Pathophysiology of Migration

Two major factors play a role in maintaining the normal position of teeth.

1. The health and normal height of periodontium.
2. The forces exerted on the teeth

1. The health and normal height of periodontium

A tooth with weakened periodontal support is unable to withstand the forces and moves away from the opposing forces. It is important to understand that the abnormality in pathological migration rests with the weakened periodontium. The force itself need not be abnormal. Forces that are acceptable to an intact periodontium become injurious when periodontal support is reduced. Pathologic migration may continue even after a tooth no longer contacts its antagonist.

2. Changes in the forces exerted on the teeth

Changes in the forces may occur as a result of

a) Unreplaced missing teeth
b) Failure to replace first molar.
c) Pressure from tongue
d) Pressure from granulation tissue of periodontal pocket
e) The food bolus during mastication.

Pathologic migration is also an early sign of localized aggressive periodontitis. Weakened by loss of periodontal support, the maxillary and mandibular anterior incisor drift labially and extrude creating diastemma between the teeth.

Extrusion

Extrusion of a tooth means change in the normal position of the tooth in the occlusal or incisal direction within the the same arch. It can be physiological or pathological. Pathologic migration refers to tooth displacement that results when the balance among the factors that maintain the physiologic tooth position is disturbed by periodontal disease.

Case report CASE 1

A female patient aged 32, reported to the Department of Periodontics of Azeezia College of Dental Sciences and Research. She had a complaint of pain and mobility of maxillary right central tooth for the last 6 months. The tooth has been splinted 2 years back due to mobility (Fig.1). On clinical examination, tooth 11 was pathologically migrated with Grade III mobility with gingival recession. On periodontal probing, the tooth showed all around a pocket depth more than 8 mm (Fig.2). IOPA reveals complete absence of lamina dura, widening of periodontal ligament space and considerable amount of bone loss (Fig.3). Diagnosis was chronic periodontitis with extrusion of tooth 11. While considering all treatment options, since the prognosis of tooth was poor, extraction was suggested. While fabrication of RPD the same extracted tooth was used instead of artificial tooth in the denture. It gives more natural appearance and self confidence to the patient.

Treatment plan is carried out in two phases

1. Modification of Extracted tooth.

2. Fabrication of Immediate removable Partial Denture.

Modification of extracted tooth

The tooth was extracted under L.A and socket was filled with alloplastic bone graft to prevent the collapse of the ridge. The sutures were placed to get a primary




Case 1 *Fig 1* *Fig 2* *Fig 3 IOPA* *Fig 4,5,6* Modification of Extracted tooth






Fig 7, 8, 9 RPD with extracted tooth **Case 2** Preoperative view Extracted site







Extracted teeth Prepared teeth RPD with prepared teeth Post operative view

closure of the extracted socket. This procedure also helps in future implant placement in the defective bone site. The extracted tooth was preserved in normal saline. (Fig.4) An alginate impression was taken to get the exact duplication of the site. The measurement was made in the cast to assess the exact length of the pontic and then assess the root portion to be resected to give the same exposure of the root as seen in the preextracted site. (Fig.5) The residual pulp tissue was removed using files and broaches; complete irrigation was done. The apical opening of the crown was sealed using composite resin.(Fig.6) Then retention grooves were prepared on the cervicolingual margin of the pontic for the mechanical retention.

Fabrication of immediate RPD

The modified extracted tooth was incorporated during the fabrication of the partial denture. For having a natural appearance, the same alignment and characterisation was given. Finishing and polishing was done.(Fig.7) Immediate removable partial denture was delivered to the patient.(Fig.8) On review after one week ,there was no colour change in the prosthesis and the healing was proper (Fig 9).

Discussion

This technique requires maximum of 3 hours of chairside time. This technique is accepted if the patient wants to go out of the clinic without any change in her dentition and esthetics.It is to be considered more of a long term replacement for those patients who, because of the periodontal prognosis of abutment teeth or financial or medical reasons, cannot have a more traditional fixed bridge or implant prosthesis. Weakened by loss of periodontal support, if the maxillary anterior incisors drift labially and extrude, creating diastema between the teeth, replacement with same natural tooth on RPD can correct the diastema with regaining an efficient esthetic appearance.

CASE 2

The same treatment procedure was performed to replace Periodontally compromised teeth (21 and 22) with RPD.

These case reports illustrates a technique where there is an immediate replacement of the missing tooth using RPD with patient's own tooth, so that natural esthetic appearance can be reproduced. This method can overcome the "artificial look" of normal removable partial denture.

***Final year BDS student, ** Reader, Dept. of Prosthodontics, *** Principal and Head of the Dept. of Periodontics, Azeezia College of Dental Sciences and Research, Kollam**

Dental practice management - 10 Tips on reaching your practice potential

Introduction

Every practice has the potential to reach the next level. Many dentists are doing fairly well, but could take their practices to the next level by focusing on a few key factors. With the demands of patient care, it can be difficult to find the time to manage the practice, lead the team, and make necessary changes. The following tips can help you release your practice potential:

1. Upgrade Your Scheduling System

Practices should adopt an accelerated scheduling model to improve productivity and staff efficiency. Switching from 15-minute to 10-minute units can create additional time in the schedule, allowing the practice to treat patients more efficiently and provide better customer service. Remember that your scheduling system controls both production and stress. A more efficient schedule creates better patient flow and reduces practice stress.

2. Enhance Case Presentation Skills

Scripting is a critical communications tool that can enhance the verbal skills of the entire team. During morning and monthly meetings, team members can review and rehearse case presentation scripts that focus on patient benefits. The goal is not to memorize scripts word-for-word, but to provide patients the right information in the team member's own words. Scripting should also emphasize comprehensive treatment—an approach that gives patients more options to achieve optimal oral health.

3. Maximize the Hygiene Department

The hygienist spends more time with patients than any other team member, including the dentist. A well-trained hygienist can educate patients about a wide variety of practice services, provide in-depth information about cosmetic services, and motivate patients to consider more comprehensive treatment.

4. Provide Flexible Financial Options

Practices can dramatically increase case acceptance by ensuring that a wide scope of financial options are available to patients. Make paying for cosmetic and need-based dentistry easy by offering these options: * 5% discount for full payment * Half up front and half before treatment is completed * Credit card payments * Third-party financing

The more financial options you give your patients, the greater the opportunity to increase case acceptance, especially for implants and cosmetic dentistry. The right financial options, combined with other case presentation strategies, can make an attractive smile and an enhanced appearance a reality for more patients.

5. Update Your Budget

Every practice needs to track revenues, expense patterns, and resulting profitability. A budgeting process allows the dentist to determine financial goals and then develop systems that support those goals. Be aware that budget control includes not only cost-control measures, but also specific production and collection goals. Be consistent when monitoring practice performance.

6. Expand Services

Adding new services can help practices achieve growth now and in the future. The key is to select services that are profitable, manageable, easy to implement, and, most importantly, desirable to your existing patient base. Expanding the service mix provides the practice a greater opportunity to present comprehensive dentistry, increase production and boost profitability. Practices should offer a variety of need-based and elective services, including:

* Whitening * Veneers * Crowns and Bridges
* Implants * Periodontal Maintenance * Preventive Services

The right service mix is key to providing appropriate choices for your patients as well as helping the practice to succeed. Promoting comprehensive dentistry can lead to increased production, less stress, and greater profitability.

7. Implement Documented Systems

Total Quality Management demands the dental practice evaluate all practice operations and determine the best way to perform each function. Systems are critical in a dental practice because practices rely on limited resources. Maximum profitability can only be achieved when these systems are consistently effective and efficient.

8. Hold Regular Morning Meetings

Organized morning staff meetings are critical for a productive office. This brief meeting is designed to prepare the staff for the day and ensure the best quality care for patients. Productivity is achieved in three ways. First, performance is benchmarked against goals for all key categories (production, collections, case presentation, accounts receivable and accounts payable). Second, the schedule is reviewed to determine if any issues exist, including emergencies. Third, opportunities are identified for case presentation.

9. Provide Written Job Descriptions

Team members must have clearly defined job descriptions outlining specific responsibilities. It is important to incorporate accountability into the systems. Taking into account how hectic a dental practice schedule may be, the goal is to ensure that team members: know their responsibilities, are well trained to perform their duties efficiently, and gain professional satisfaction from their work.

10. Improve Customer Service

Patients must feel special from the moment they walk into the office, whether it is the first time or the fiftieth. Exceeding patient expectations requires what I call "WOW" customer service. This occurs when every interaction has been exceptional, causing patients to be amazed at the level of customer service, making them say "WOW" as they leave the office.

Enthusiasm is a critical component of excellent customer service. It is not enough for staff members to thank patients for selecting the practice. The dental team must do it the right way—sincerely and enthusiastically. Simply going through the motions reflects poorly on the practice. After all, patients are the reason the practice exists.

Improving customer service requires a systemized approach that is repeated for every patient, every hour, every day. An effective way to ensure quality customer service is to provide patients with a survey form that allows them to rate aspects of your practice. We recommend sending a thank you to all new patients after their first visit to the practice. It also is a good idea to send a survey to capture the patient's experiences.

Summary

You have the power to improve your practice and your life. These 10 tips can help you release your practice potential. Levin Group sees clients make remarkable progress in a year or less. These tips can help you maximize your practice performance.

If you would like a sample copy of our Thank You brochure, please call 888-973-0000 or e-mail customerservice@levingroup with "thank you brochure" in the subject line. Please include your contact information (name, phone, address, and specialty) within the body of the e-mail.

Courtesy: Roger P. Levin, DDS

Research Findings

*** Bindu R. Nayar**

Decay of Baby Teeth May Be Linked to Obesity, Poor Food Choices

ScienceDaily (June 23, 2010) — Preschool children with tooth decay may be more likely to be overweight or obese than the general population and, regardless of weight, are more likely to consume too many calories, a new study indicates.

Poor eating habits may play a role in both tooth decay and obesity in preschoolers"Dental decay is the most common chronic disease of childhood, and obesity in youth is a growing problem. To prevent these problems, the dentist's office may be an important place to educate families about nutrition. Each child's height and weight were measured before the procedure to calculate the body mass index, or BMI. For most people, BMI reliably indicates the amount of body fat.

However, the questionnaire showed that both the normal-weight and overweight children consumed more calories a day than recommended for their age (1,440 and 1,570 calories respectively). Seventy-one percent of children consumed more than 1,200 calories per day although the daily recommended caloric intake ranges from 1,000 to 1,400 calories depending on age and gender of the child.Further analysis is needed to explore whether consumption of juice and sweets accounts for the excessive calorie intake and links high BMI and dental decay

Dental Pulp Cells for Stem Cell Banking

ScienceDaily (June 17, 2010) — Defined sets of factors can reprogram human cells to induced pluripotent stem (iPS) cells. However, many types of human cells are not easily accessible to minimally invasive procedures.K. Takahashi, T. Tanaka and S. Yamanaka, et al evaluate dental pulp cells as an optimal source of iPS cells, since they are easily obtained from extracted teeth and can be expanded under simple culture conditions.

From all six cell lines tested with the conventional three or four reprogramming factors, iPS cells were effectively established from five lines. Furthermore, determination of the HLA types of 107 DPC lines revealed two lines homozygous for all three HLA loci and showed that if an iPS bank is established from these initial pools, the bank will cover approximately 20 percent of the Japanese population with a perfect match.

Analysis of these data demonstrates the promising potential of dental pulp cell collections as a source of cell banks for use in regenerative medicine. Direct reprogramming of patients' somatic cells would allow for cell transplantation therapy free from immune-mediated rejection. An alternative approach is to establish an iPS cell bank consisting of various human leukocyte antigen (HLA) types. Safety issues must be considered as to which types of somatic cells should be used for such iPS cell banks.. "Although at an early stage of development, this innovation offers prospects for cell therapy approaches for the treatment of human disease."

Tray Bleaching May Improve Oral Health of Elderly, Special-Needs Patients

ScienceDaily (June 28, 2010) — A tooth-bleaching agent may improve the oral health of elderly and special-needs patients.Standard oral hygiene, such as brushing and flossing, can be difficult or impossible for patients with mental challenges or impaired manual dexterity. Additionally, when health problems or medications cause xerostomia, or dry mouth, the lack of saliva reduces the mouth's natural protective mechanisms. These problems lead to plaque accumulation, cavities and periodontal disease, and could further impact the patient's health.

By applying the tooth whitener carbamide peroxide through a custom-fit mouth tray might combat those problems.Dentists have used carbamide peroxide, or urea peroxide, for decades to whiten teeth, but its original use was as an oral antiseptic. It removes plaque, kills bacteria and elevates the mouth's pH above the point at which enamel and dentin begin to dissolve, which results in fewer cavities. "All these benefits lead us to believe that tray bleaching can be a very effective supplemental method of oral hygiene for patients facing greater challenges keeping their mouths clean," The trick is in the tray, After a complete dental exam, the dentist can make the custom-fit tray that the patient can wear comfortably at night or for several hours during the day. The carbamide peroxide gel can be prescribed or purchased over-the-counter.

Putting Teeth Into Forensic Science

ScienceDaily (June 16, 2010) — In a large natural disaster, knowing the birth date of an individual can guide forensic investigators to the correct identity among a large number of possible victims. . The radiocarbon analysis showed that dating the teeth with the carbon-14 method would estimate the birth date within one year.Age determination of unknown human bodies is important in the setting of a crime investigation or a mass disaster, because the age at death, birth date, and year of death, as well as gender, can guide investigators to the correct identity among a large number of possible matches.

"Above ground testing of nuclear weapons during the Cold War (1955-1963) caused a surge in global levels of carbon-14 (^{14}C), which has been carefully recorded over time. The radiocarbon technique determines the amount of ^{14}C in tooth enamel. Scientists can relate the extensive atmospheric record for ^{14}C to when the tooth was formed and calculate the age of the tooth and its owner.44 teeth from 41 individuals were analyzed using racemization (a chemical process in which one amino acid is converted to its counterpart) analysis of tooth crown dentin or radiocarbon analysis of enamel, and 10 of these were split and subjected to both radiocarbon and racemization analysis. Combined analysis showed that the two methods correlated well. Carbon-14, or radiocarbon, is naturally produced by cosmic ray interactions with air and is present at low levels in the atmosphere and food. Although nuclear weapons testing was conducted at only a few locations, excess levels of ^{14}C in the atmosphere rapidly dispersed and equalized around the globe.Since 1963, as a result of a worldwide test ban treaty, ^{14}C levels in the atmosphere have been decreasing exponentially with a mean half-life of 16 years. Carbon-14 levels have not decreased because of radioactive decay (^{14}C has a half-life of 5,730 years), but rather ^{14}C has moved out of the atmosphere due to mixing with large marine and terrestrial carbon reservoirs."Because radiocarbon is incorporated into all living things, this bomb curve forms a chronometer of the past 60 years".

***** Professor, Dept. of Periodontics, Govt. Dental College, Thiruvananthapuram**

Diagnose the following case

* Nityasri V., ** Anita Balan

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 1 Intraoral photograph showing buccal aspect of the swelling,

Fig. 2 Mandibular occlusal radiograph showing the expansion of lingual cortical plate,

Fig. 3 Axial CT slice showing the mandible with an expansile lesion on the right side with lytic changes and cortical thinning,

Fig. 4 Panoramic radiograph showing the lesion in relation to 34, 35 and 36.

A 20 year old female patient reported to the OPD, Govt. Dental College Trivandrum with the complaint of a painless swelling in relation to lower left back teeth of 3 months duration. Clinical examination revealed a swelling of size 3X3 cm in relation to 34 - 36, buccally and lingually, which was bony hard on palpation. Mandibular true occlusal radiograph showed an expansion the lingual cortical plate in relation to 35 and 36. Panoramic radiograph revealed a multilocular radiolucency with well defined borders associated with the roots of 34-36 with distal inclination of crown of 35 and mesial inclination of crown of 36. The most probable diagnosis is -

ANSWER:

Ameloblastoma

Ameloblastoma is a benign odontogenic tumour usually located in jaw bone with an increased frequency reported in the mandible. Three clinico-radiologic groups occur: solid or multicystic, unicystic and peripheral. Unicystic ameloblastoma has a large cystic cavity with luminal, intraluminal or mural proliferation of ameloblastic cells and is a less aggressive variant with a low rate of recurrence.

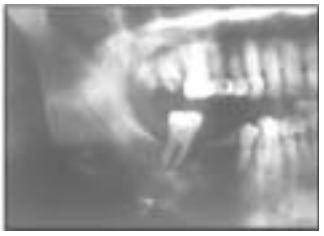
***PG student **Professor and Head,**
Department of Oral Medicine and Radiology,
Govt. Dental College, Trivandrum

Quiz

* Sajna Hemaraj, ** Nileena R. Kumar, ** Sharafuddeen K. P., *** Anita Balan

1. A 63 year old man presented with a complaint of an ulcer on the chin of 2 months duration. On examination, the ulcer had a rolled margin which was hyperpigmented. The floor was covered with slough. Radiographs revealed bony erosion in the area. Identify the lesion

a. Trophic ulcer; b. Basal cell carcinoma
c. Malignant melanoma d. Tuberculous ulcer



2. A 35 year old lady presented with a growth on the lower gingiva of one month duration. She also had paresthesia over the right corner of mouth and right side of chin. She didn`t have any deleterious habits. She gave no history of drug intake. Radiograph revealed an ill defined lytic lesion in the region. Identify the lesion.

a. Pyogenic granuloma b. Non hodgkins lymphoma
c. Fibroma d. Gingival hyperplasia

3. A sixty year old lady presented with a complaint of ulcers in mouth of two years duration. On examination, there were multiple erosive areas along with intact bullae. There was scarring of the mucosa however she didn't have any ocular or genital lesions. The lesion is most likely to be

a) Pemphigus b) Erythema multiformae
c) Cicatricial pemphigoid d) Bullous lichen planus

4. Bone pain, renal calcinosis, giant cell lesions of jaw are seen in

a) Hyper parathyroidism b) Fibrous dysplasia
c) Hyperthyroidism d) Pagets disease

Answers:
1-c; 2-b; 3-c; 4-a; 5-c; 6-c; 7-c; 8-d; 9-b; 10-c

5. A 63 year old female patient presented with a firm swelling on the left preauricular region. Skull radiograph revealed multiple punched out radiolucencies. Identify the disease.

a) Sickle cell anaemia b) Thalassemia
c) Multiple myeloma d) Lymphoma

6. A 65 year old female patient presented with erosions and burning on the tongue of one week duration. There was no history of any systemic illnesses, no vesicle formation, nor history of any medications. She gave a recent history of change of toothpaste. The lesion is

a) Erythema multiformae b) Pemphigus
c) Allergic stomatitis d) Lichen planus

7. Talons cusp is characteristic of which syndrome?

a) Edwards syndrome
b) Klinefelters syndrome
c) Rubinstein Taybi syndrome
d) Downs syndrome

8. Characteristic radiographic appearance of Ewing's sarcoma

a) Ground glass appearance
b) Sun ray appearance
c) Cotton wool appearance
d) Onion skin appearance

9. Identify the condition

a) Amelogenisis imperfecta
b) Dentinogenesis imperfecta
c) Dentinal dysplasia
d) Fluorosis

10. A 60 year old male patient presents with unilateral facial paralysis, crusted lesions in external auditory meatus, hearing defects, and vertigo. Most probable diagnosis is :

a) Bells palsy
b) Melkerson Rosenthal syndrome
c) Ramsay hunt syndrome
d) Moebius syndrome

*** PG Student, ** Asst. Professor,**
***** Professor and Head,**
Govt. Dental College, Kozhikode

Secretary's Report and Association News

Secretary's Message

Dear Member,

The dynamic functioning of any organization hinges a lot on the unity of its members in action. If we stand together, whatever be the threat directed at us, our concerted effort will always have the strength to shatter them all.

Attacks on hospitals and clinics are on the rise everyday in our state and it is the constitutional right to carry out one's work fearlessly without any restrain that is being violated here.

At a time, when strong legal protection is the need of the hour to shield our profession from these kind of assaults, the lackadaisical attitude of our government to implement the hospital protection bill is indeed deplorable, but what was commendable was the manner in which our members rose in unison to support the hunger strike that was observed to protest this indifferent attitude of the authorities.

In a way, the epic strike has opened new avenues for joining hands with the IMA to face unitedly any threat that may imperil the medical fraternity.

To all IDA members who took part in this protest and all those who single mindedly inspired this noble idea, a BIG BIG thanks from the bottom of my heart.

Yours in IDA,

Dr. Shibu Rajagopal

Kollam
13.07.2010

THRISSUR

CDE Programme: CDE programme was conducted in Hypnosis in Dentistry by Dr. Raghu Nandanan Bsc, MSW, MCH on 29th May 2010 was attended by 56 members.

CDH Activities: A Dental Checkup camp was conducted at St Antony's Church, Arimbur., Fr. John Payyapilly was Chief Guest. 92 patients were screened at the camp and treatment needs assessed to provide free treatment at RDC, Chalissery as a tie-up with IDA. Attended by Dr. Tony Paul, Dr. Joe Francis attended.

IV GB Meeting: IV GB Meeting, Ladies Wing Inauguration, on 29th May at Hotel Trichur Towers. Dr Serena Gilvaz

HOD,Obst&GyneacJM Medical college, TSR inaugurated ladies wing. Dr. Aneena Joseph was elected as Ladies wing president, Dr. Smitha Babu as ladies wing Secretary. General Body was presided by Dr. Alex Mathew. Minutes of previous G B, Exe Committee Passed. Action taken report by Secretary Dr Geo Francis. Meeting adjourned and followed by a Goan Food festival. 56 members attended.

Anti Tobacco Day observed on 31st May at IDA hall. Pledge by Exe Members of TCR branch to quit smoking & be a model for other IDA members of IDA branch.

KOCHI

IDA Kochi had executive committee meetings in April and May at Lotus club and June at IMA house. We had our second and third general body meetings on 22nd April and 27th June in Lotus club and Park Central Hotel respectively.

The last three months saw IDA Kochi conducting three major inter branch CDE programs in addition to the three conducted earlier this year and an intra branch CDE program. All these programs had Kerala State CDE wing and Kerala Dental Council approval and had credit points. Certificate of attendance were given to the participants in all these programs.

Intra branch CDE program was conducted on the 22nd of April at Lotus club with Dr. Eldo Koshy speaking on Conservative Alternatives to conventional crowns. The program was supported by Colgate Palmolive India Pvt. Ltd.

The inter branch CDE Programs were on the 1st of May evening from 7pm to 10pm at Park Central Hotel Kaloor, 23rd May at Kallenchery Retreat, Kumablanghi and 27th June at Park Central Hotel, Kaloor. Dr. Rajeev Verma on Predictable Fixed Prosthodontics, Prof. Dr. Varghese Mani, Dr. Srikanth Mallan, Dr. Prem Nanda and Dr. Prashanth Pillai on the various aspects of Implantology and the current concepts on the single piece and two piece implants, Dr. Arvind Shenoy on Onederful Esthetic Creations - your one solution to every restoration were the speakers and topics for each of these programs. The programs were well attended and well received. Colgate was the sponsor for each of these programs.

The IDA Colgate Academic module program II was conducted at Amrita Dental College on the 3rd of May from 10am to 12.30pm. The medical director of the Amrita Institute was the chief guest and the Dean of the college presided over the meeting. Our members Dr. Siby T Chennankara and Dr. Pramod John were the speakers for the day with Advanced Periodontitis and Dentine Hypersensitivity as the topics. The secretary's role was to brief the students on IDA and to invite them to join the Indian Dental Association. Mr. M.N. Prasad of Colgate made a short presentation on Colgate products and the video of the new product launch was also shown to the students. Dr. Alice, Dr. Balagopal Varma and Dr. Anjana helped in organizing and conducting the program.

IDA Kochi conducted the Students Cricket Tournament on the 16th of May at Mar Baselious Dental College. The event was really successful with four dental colleges participating. There were hard fought matches, eventually the hosts were the winners.

This years' Shuttle and TT tournament was conducted at D.D. Retreat, Thammanam at 3pm on the 20th of June 2010. Dr. Binu Augustine and Dr. Kaushik were the winners and runner up respectively in Table Tennis. In shuttle badminton Dr. Kaushik and Dr. Shyam Sundar were the winners and Dr. Vinod Mathew and Dr. Sanjeev R were the runners up.

IDA Kochi had DJ night and Live Foot Ball show on the 27th of June from 7:30 pm to 10:30 pm at Park Central Hotel, Kaloor. Around 25 families came for the DJ night and it indeed was a memorable night.

President Dr. P.C. Sunil with his team of office bearers during the oath taking during installation ceremony

QUILON

CDH ACTIVITIES : Dental camp : Old age home treatement conducted on 21/4/10 and 18/5/10 . 60 patients were treated. Dr Sunil george. Dr Santhosh kumar,DrSreejesh, Dr Desmond and Dr Biju kumar were attended.

Anti tobacco booklet release

Anti tobacco day banner release

CDE by Dr Alexander

Anti tobacco day: On May 30 anti tobacco day banner of ida Quilon branch was released by state president Dr Samuval K Ninan at hotel central park,Kadappakada, Kollam. Anti tobacco booklet of ida Quilon branch was released by Dr Samuval K Ninan ,presenting the booklet to immediate past state president Dr Prathap Kumar. 25 Banners were put in the prominent places of Quilon town. Anti tobacco day programme was sponcerd by jhonson & jhonson company.

CDE programe: One cde prorame conducted at hotel Dona Castle,Kollam, on 19/6/2010

Topic Fever – Current scenario by Dr Alexander from holy cross hospital kottiyam, 40 Members were attended.

Monthely meetings were conducted on every 3rd saturday at Lions hall, Kollam

TRIVANDRUM

CDE PROGRAM: CDE program of IDA Trivandrum branch was held at the hotel Residensy towers, Trivandrum, on the 20th June 2010. The title of the program was "Clinical Restorative Dentistry: Pediatric to Geriatric: A journey from 04 to 94 years". Faculty was Dr. Rajeev Thaper. 6 credit points was awarded to the participants.

World no tobacco day : 31 may 2010:- IDA Trivandrum branch observed the World no tobacco day by organizing Anti tobacco campaign at the central prison Thiruvananthapuram. The program was inaugurated by Sri Jayan Babu, Worshipful Mayor of Trivandrum Corporation. The inmates of the central prison sang a prayer which marked the opening of the formal meeting. Sri K RadhaKrishnan (Superintendent of central Prison, Trivandrum) welcomed the gathering. Dr Sangeeth K Cherian, President of IDA Trivandrum branch, delivered the presidential address. Sri KA Kumaran, Chief welfare officer, offered felicitations. Sri Johnson J Idayaramula ADIC India, in his felicitation address requested the IDA to take up the matter for organizing a permanent dental unit in the central prison. Dr C Rajeevan (Chief medical officer Central Prison) Sri Baburajan (Jailor, CP, Tvpm) and Chandra Babu (welfare officer) also offered felicitations. Dr Mukesh T Past president IDA Trivandrum, offered vote of thanks

The inaugural program was followed by oral cancer screening of the central prison in mates, by the members of IDA

Trivandrum Branch. The team comprised of Dr Capt Vivek V, Dr Arun R, Dr Prasanth, Dr Suresh Kumar G, Dr CP John, Dr Gins Paul, Dr Sangeeth K Cherian, Dr Joseph Alancheril, Dr Vinoth MP, Dr Sundeep Krishna, Dr Sony Thomas, Dr Achutan Nair, Dr Mukesh, Dr Lin Kovoor. Almost 1600 inmates were given an opportunity to get themselves examined, and seek advice and treatment. 60 odd patients with pre cancerous lesions were identified and counseled. There was a video presentation and poster presentation on the tobacco menace, for the benefit of the inmates. The jail authorities and the administration, appreciated the efforts of IDA Trivandrum branch, as the program was referred to several times, as the first of its kind in the central prison by dentists.

SYMBIOSIS: THE CLINICAL CLUB OF IDA TRIVANDRUM

Three programs were organized by symbiosis.

On the 13 th April DR JOJI THOMAS presented the topic "ASEPSIS IN DENTISTRY".

On the 12 th May DR JOMY VARGHESE presented on "OROFASCIAL INFECTIONS"

On the 8th June DR KRISHNA KUMAR presented DREAM, DARE, SUCEED".

MALANAD

IDA Malanadu Branch Implimented 101 free denture programme by delivering dentures to the deserving patient's in and around Malanadu Branch by the co-operation of Kudumbasree at IMA Hall Thodupuzha on June 5 2010. Chief Guests were Dr. Samuel K Ninan (State President), Dr. Alias Thomas (National Vice President), Mr. Marco Vanninni (Italy), Prof. Jessy K Antony (Municipal Chairperson, Thodupuzha). Dr. Marilyn Alias (President), Dr. Ciju A Paulose (Hon. Secretary). Free Denture project implemented by the whole hearted support of members of Malanadu Branch and Dent Care Dental Lab Pvt Ltd.

IDA State President Dr. Samuel K. Ninan addressing the gathering
Dr. Alias Thomas, Mr. Marco Venninni, Dr. Marilyn Alias, Dr. Ciju A. Paulose

Prof. Jessy Antony, Municipal chairperson giving denture to a deserving patient
Dr. Alias Thomas, Mr. Marco Vanninni, Dr. Marilyn Alias, Dr. Ciju A. Paulose, Dr. Samuel K. Ninan, Dr. A.V. Pius

Dr. Samuel K. Ninan inaugurating free denture programme by giving denture to a deserving patient
Mr. Marco Vanninni, Dr. Marilyn Alias, Dr. Ciju A. Paulose, Dr. Byju Paul Kurian

THIRUVALLA

Installation ceremony: Installation ceremony of IDA Thiruvalla Branch for the year 2009-2010 held on 24 January 2010 at Travancore Club Auditorium Thiruvalla. Dr Samuel K Ninan President IDA Kerala state installed the new President Dr. A Devadathan.DrOommenAju Jacob, Principal, Pushpagiri College Dental Sciences was the chief guest. Dr George Thomas, IDA National President Elect for the year 2011 was the guest of honour. Rev.Fr. Philip Payyampallil .Direcor Pushpagiri Medicity, Presidents and Secretaries of neighbouring IDA Branches felicitated the new team. Dr Binoy Mathews N, Hon. Secre-tary IDA Thiruvalla proposed the vote of thanks & followed by cultural programmes by the student members of IDA Thiruvalla.

CDE Programme: Conducted a CDE programme on January 30th 2010. Topic: Medico Legal aspects in dentistry. Faculty: Dr George Paul MDS. Venue: Pushpagiri College of Dental Sciences, Thiruvalla.

Orphanage Visit: Members of IDA Thiruvalla Branch visited the orphanage (Mercy Bhavan) at Kunnarnthanam on 07 February 2010. Sponsored the meal for inmates and donations were given in cash and kind to the orphanage by the members.

CDE Programme: Conducted a CDE programme on February 2010. Topic: Basic Life Support (Lecture and Hands on). Faculty: Team of doctors from Malabar Institute of Medical Sciences, Calicut. Venue: Pushpagiri College of Dental Sciences.

IDAThiruvalla Branch hosted a State CDE Programme which was jointly con-ducted by Kerala Dental Council and IDA Kerala State on 14 March 2010. Topic: Full mouth rehabilitation Speaker: DrAqueel Sajjad Reshamwala Venue: MAC FAST Auditorium Thiruvalla

Dr Mathew Joseph Vayalil, President, Kerala Dental Council was the chief guest. Dr Samuel K. Ninan, President, I DA Kerala State and Dr. Shibu Rajagopal, Hon. Secretary IDA Kerala State were the guest of honour. 547 members registered and 487 delegates attended.

Family get together: Afarnily meetwas conducted onApril 25th at Green valley resortAdoor DrShibu Rajagopal, Honourable Secretary IDA Kerala State was the Chief Guest. Dr Samuel K Ninan, President, IDA Kerala State was the guest of of honour. Various games were organized for kids and ladies.Members were enjoyed the summer splash at swimming pool.The cultural programme started with dance by kids followed by orchestra by Idea star singer fame Mr Jayakrishnan and team. The highlight of meet was the lip smacking dosa varities for the dinner.

Students conference: Conducted a students conference on 23 May 2010. Topic: Oral Cancer (Workshop) Faculty: Prof. DrK. Raj KumarMDS(HOD Dept. of Oral Pathology, SRM Dental College Chennai) & Team. Venue: Pushpagiri Medical College (Senate Hall) 132 student members from various dental colleges attended the conference. Colgate palmolive India Ltd. was the principal sponsor

MALAPPURAM

MAY 2010

The 4th Inter-Branch CDE was held in association with Coltene-Whaledent at Chengara Heritage, Perintalmanna on 2nd May, 2010. Dr. Rajiv Verma spoke on "Predictable Fixed Prosthodontics". A hands-on session was held after the lecture.

The 6th Executive Committee Meeting was held on 6th May, 2010 at Hotel Prashant, Malappuram. It was presided by Dr. Deebu, President of IDA, Malappuram.

The 5th CDH camp was held in association with JCI, Kotakkal at Thehsinul Muslimeen Madrasa, Kooriyad on 16th May, 2010. A total of 262 patients were checked and an awareness class was taken for the public. Oral hygiene kits were distributed.

The No-Tobacco Observation Day was celebrated by IDA, Malappuram on 31st May, 2010 in a very unique way. 10Ft x 10Ft flex banners depicting the ill-effects of tobacco and its products were raised and placed at various spots in ten important towns of the district. It was a runaway hit with the public.

JUNE 2010

The 7th Executive Committee Meeting was held on 1st June, 2010 at Hotel Prashant, Malappuram. It was presided over by Dr. Deebu, President of IDA, Malappuram.

The MIDA X'press tour to Veegaland was conducted on the 6th June, 2010. A total of 54 people were present and all had a jolly good time.

The 5th Inter-Branch CDE was held in association with Ivoclar-Vivadent at Chengara Heritage, Perintalmanna on 12th June, 2010.

Dr. Kshitij Mawlikkar spoke on "Post and Core". A hands-on session followed the lecture.

Prateeksha Bhavan, a home for the differently challenged children, at Tavanur was adopted by the branch. A treatment camp for its 50 inmates was held at 4 clinics in Valanchery that belonged to our fellow members. A sumptuous lunch was also served to the inmates after the treatment was completed. Clothes that were collected from among our members were distributed among the inmates.

IDA, Malappuram Branch with the support of Rotary Club of Tirur observed the World Environment Day on 5th June, 2010 by planting tree saplings at Overbridge Junction, Tirur. The programme was attended by Rtn Kumari Sukumaran, Dr. Rajesh Raveendranathan, Hon. Secretary, IDA, Malappuram and Dr. Abdul Razak, CDH Convenor, IDA, Malappuram Branch.

KASARAGOD

CDE Programme: Conducted a CDE programme on 29-4-2010 at IMA Hall Kasaragod. The topic was "Road safety principles" and the speaker was Mr. Kunhambu Nair, Rtd. Deputy Transport Commissioner. The meeting was followed by dinner.

Executive committee meeting: An executive committee meeting was held on 17-6-2010 at KIMS Hospital, Kasaragod. Discussion about NOHP was done.

Combined meeting of IDA and IMA members of Kasaragod

On 19-6-2010 a combined programme was conducted at IMA hall Kasaragod. A talk about our diet titled "We are what we eat" was presented by Dr. Srinivas Kakkillaya, an eminent Physician from Mangalore. The talk was very informative followed by good discussion with the members. The dinner was sponsored by Intima, a division of IPCA Laboratories.

NEDUMBASSERY

1st to 5th APRIL –FAMILY TRIP TO MALAYSIA: IDA Nedumbassery conducted a family tour to Malaysia from april 1st to 5th. A total of 62 packs consisting of members and their family took part in the trip.

3rd APRIL – GENERAL BODY MEETING AT MALAYSIA: 2nd General body meeting was held at Hotel Kwality, Kuala Lumpur, Malaysia. It was attended by 31 members and their family.

26th APRIL – CLINICAL CLUB INAUGURATION

Venue - Periyar club Aluva Time - 8:00 pm

Clinical club of Nedumbassery branch was inaugurated by Dr. Joy Mathew Dept. of Endodontics, Mar Bascelious dental college, Kothamangalam. It was followed by a discussion on Endodontics. Dr Reji Thomas and Dr Sateesh asok along with Dr Joy guided the programme. 17 members attended the programme

4TH MAY – 2ND CLINICAL CLUB PROGRAME

Topic: minor surgical procedures; Faculty: Dr. Varghese Mani

Venue: Hotel GeeBee palace , Angamaly; Time: 8:00 pm

Attendance - 21 members

14th MAY - 3RD EXECUTIVE MEETING

Venue: Hotel Surya Angamaly Time: 8:30 pm

Attendance – 13 exe. Members.

29TH MAY – 3RD GENERAL BODY MEETING

Venue: Hotel Mahanami, Aluva; Time: 8:00 pm

After the general body meeting in malaysia

Attendance - 42 members

We had a talk on Post Endodontic restorations by Dr Prabhath singh, Associate Prof., Dept.of Endodontics, Amritha school of dentistry

31st MAY - ANTI TOBACCO DAY

Posters were printed and posted at public areas and in clinics

18th JUNE -4TH EXECUTIVE MEETING

Venue: Hotel Surya ,Angamaly; Time: 8:30 pm

Attendance: 12 exe members

Family trip to Malaysia

Banner of IDA displayed on the bus in Malaysia

MALABAR

2nd Executive Meeting: The second executive meeting of the IDA Malabar branch was held on 29-04-2010 at IDA Hall, Ashokapuarm at 7.30 pm. Various important decisions were taken.

World No Tobacco Day Celebrations: World No Tobacco Day Celebrations of the Kerala State Branch was conducted at Calicut by the IDA Malabar Branch with various programmes. A Notice regarding all these programmes were distributed along with the newspapers at Calicut City on Sunday May 30th and details of the programme was inserted in the today's programme column of all the leading newspapers on May 31st.

The public function was conducted at Jubilee Hall, Calicut at 5.00 pm. The IDA Kerala State first Vice President Dr. R.K. Nizarosiyo chaired the programme. State CDH Convener Dr. Joseph. C.C welcomed the gathering. Worshipful Mayor of Calicut City Sri. M. Bhaskaran inaugurated the function. Dr. C. Ravindran, Principal, Medical College, Calicut delivered the No Tobacco Day message. Dr. Binu Purushothaman (President, Calicut JCI), Mr. Ashokan Aalapurath (President, Kerala blood Donors Forum) and Dr. Antony Thomas felicitated on the occasion. IDA Malabar Branch President Dr. Sharafuddeen. K.P proposed the vote of thanks. Dr. Bijoy Oommen (President, IDA Wayanad Branch), Dr. K.S. Ravindran Nair, Dr. Anto Joseph, Dr. Joy Philip, Dr. Jamal. N.P, Dr. Ranjith. R and Dr. Manoj Joseph were also attended the function. After the public function, an anti tobacco tableau was arranged on a vehicle with the documentary show, was flagged off by the Worshipful Mayor of Calicut City Sri. M. Bhaskaran. The documentary was screened at different important spots in the Calicut city repeatedly in the evening.

5th CDE Programme: Our 5th CDE programme was held on 13th of June 2010 at 9.30 am at IDA Hall. It was a one-day comprehensive seminar on "Pain & Palliative Care", which is a prestigious project of IDA Kerala State this year.

6th CDE Programme: Our 6th CDE programme was held on

Worshipful Mayor of Calicut Sri. M. Bhaskaran flaging off the No tobacco documentary vehicle

26th of June 2010 at 6.30 pm at IDA Hall. The topic was "Simple & Easy Aesthetic Dentistry – Now at its best" by Dr. Arunachalam. D, Professor, Dept. of Periodontics, Priyadarshini Dental College, Thiruvallur. 84 members attended the programme.

Branch Shuttle Tournament: Branch shuttle tournament of IDA Malabar branch was held on 27th of June 2010 at Officer's Club, Calicut. Many of our members participated in the events. Dr. Praveen. S was the winner of the Men's Singles title and Dr. Binu Purushothaman was the runner up. The winners of the Men's Doubles event were Dr. Santhosh. V.C and Dr. Roshan Khan beating Dr. Praveen. S and Dr. Premanandan. P.K. The Ladies' Singles title was won by Dr. Nisha Soumithran beating Dr. Rekha Binu.

CENTRAL KERALA KOTTAYAM

APRIL 2010 **CDH ACTIVITES**

ORAL HEALTH AWARENESS CLASS: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College,Chelad and Kottayam Social Service Society conducted oral health awareness class for 116 people at SS Peter and Paul Church parish hall, Cherpunkal on 17th April 2010.

DENTAL TREATMENT CAMP: IDA Central Kerala Kottayam branch in association with St Gregorious Dental College, Chelad and Kottayam Social Service Society conducted a dental treatment camp for 116 people at SS Peter and Paul church parish hall, Cherpunkal on 17th April 2010.

TEACHER'S TRAINING PROGRAMME: IDA Central Kerala Kottayam branch in association with St Gregorious Dental college, Chelad and Kottayam Social Service Society conducted a oral health awareness training programme for 15 school teachers at Pachira Matha LP School on 17th April 2010.

ASHA KIRAN: IDA Central Kerala Kottayam branch conducted oral hygiene and dental awareness class for the children of HIV affected people on 23rd April 2010 at a camp organized by Atmata Kendram at St Anne's GHS, Changanacherry. Dr Augustine, President IDA Central Kerala Kottayam branch took the oral hygiene awareness class. Oral hygiene kits were distributed to all the participants.

FAMILY MEET: The second family meet of the year was held on 25th April 2010 at Hotel Backwater Resorts, Kumarakom. 62 families attended the programme and enjoyed the boat cruise in the backwaters, swimming pool, gala dinner and entertainment.

MAY 2010 **CDH ACTIVITES**

ORAL HEALTH AWARENESS CLASS AT UZHAVOOR: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College, Chelad and Kottayam Social Service Society conducted oral health awareness class for 147 people at St Stephen's Forane Church parish hall, Uzhavoor on 14th May 2010.

DENTAL TREATMENT CAMP AT UZHAVOOR: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College, Chelad and Kottayam Social Service Society conducted a dental treatment camp for 147 people at St Stephen's Forane Church parish hall, Uzhavoor on 14th May 2010.

ORAL CANCER AWARENESS AND DETECTION CAMP AT THADIYAMPADU: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College, Chelad and Kottayam Social Service Society conducted oral cancer awareness and detection camp for 160 people at Maria Sadan Animation Centre, Thadiyampadu, Idukki on 21st May 2010.

DENTAL EXHIBITION AT THADIYAMPADU: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College, Chelad and Kottayam Social Service Society conducted a dental exhibition for around 500 people at Maria Sadan Animation Centre, Thadiyampadu, Idukki on 21st and 22nd May 2010.

DENTAL TREATMENT CAMP AT THADIYAMPADU: IDA Central Kerala Kottayam branch in association with St. Gregorious Dental College, Chelad and Kottayam Social Service Society conducted a dental treatment camp at Maria Sadan Animation Centre,Thadiyampadu, Idukki on 21st and 22nd May 2010 as a part of the 5th annual SSG fest and highrange karshika mela of KSS. Around 340 people undertook dental treatment at the camp.

JOURNAL RELEASE: The second issue of the branch journal SMILE was released during the state executive at Wayanad by Dr. K. Nandakumar, editor KDJ.

BRANCH BULLETTIN: The 3rd issue of the bimonthly branch bulletin DENTAL BOND was sent to members.

WAYANAD

EXECUTIVE MEETINGS:-Three executives were held during the month of May & June.

The 1st executive was held at Hotel Edakal heritage, Meenagadi on 7-5-10. 12 members attended

The 2nd executive was held at Pulpally on 18-5-10. 10 members attended.

The 3rd executive was held at IMA Hall Mananthavady on 22-6-2010. 10 members attended.

FREE DENTURE PROGRAMME:-The distribution of dentures under the free denture programme of IDA Wayanad branch

was held on 13-6-2010 at hotel Harithagiri Kalpetta.The programme was inaugurated by IDA state vice president Dr Renjith C.K. 30 dentures were distributed to patients falling under BPL category.

IDA KERALA STATE EXECUTIVE MEETING:- The 3rd executive meeting of IDA Kerala was hosted by IDA Wayanad branch on 9-5-2010 at hotel Vythiri village, Vythiri.

ATTINGAL

Inauguration of the Palliative care unit AMMA in association with Poorna charitable trust was done on 9th of May 2010 at MGM school Varkala by MLA Varkala Kahar. Municipal chairman, Adv.Biju was the guest of honour. Dr Vijayan, a senior member of the branch initiated the programme. IMA President Dr Vijayakumar and IDA President Dr Premjith was also present at the function. AMMA Vridhasadanam and AMMA day care was inaugurated at MGM School Varkala on the same day. A three wheeler cycle was given to a handicapped person.165 persons attended the programme.

A public dental camp was conducted at Kadakkavur on 22/05/2010 in association with kadakavoor residence association.150 patients attended thecamp. Dr Arun.s lead the camp.

A cancer detection programme was conducted at Kadakkavur on 30/05/2010. 25 patients were screened on that day.

Branch website inauguration was done on 16/05/2010 at hotel Hindusthan retreat, Varkala by Dr. Vijayan.

Dr. Arun S. screening a patient

Two CDE programmes was conducted. 1.A hands-on CDE programme by Dr Binoy Ambooken on fixed orthodontics was organized at TAJ Varkala on 29/05/2010 and 30/05/2010. 11 young aspirants attended the CDE. 2.A talk by Dr Aqueel Reshamvala on different aspects of Fixed Prosthodontics was organized at Club house, Technopark on 20th June 2010. 6 credit hours were awarded to the delegates who attended the programme. 62 delegates attended this programme.

MAVELIKARA

Summer Splash 2010 – A family get together programme was held on 2nd may 2010 at Green Valley park and swimming pool, Adoor. The program started at 2 with our members enjoying in the pool and later there was a musical delight with an orchestra and dance program by professionals.

CDE on Periodontics—A CDE on periodontics in general practice was held on 30th may 2010 at Hotel Maryland. The faculty for the program was Dr.Nebu George Thomas, snr lecturer, pushpagiri dental college,tiruvalla.

CDE on Laminates—A CDE on LAMINATE VENEERS was held on 27th june 2010 at Hotel Vandanam, Mavelikara.The faculty for the program was Dr.Eldo Koshy.

NORTH MALABAR

EXECUTIVE MEETING : 6-5-2010 : 6th Executive Meeting was held at Hotel Mascot. 18 members attended the meeting.

8-6-2010 : 7th Executive meeting was held at Hotel Mascot.17 members attended the meeting.

CDE ACTIVITIES

CDE CLUB: 20-5-2010 : 3rd CDE Club of IDA North Malabar Branch was held at Hotel Malabar Residency on the topic PULP THERAPY IN CHILDREN by Dr Faizal.

23-6-2010 : 4th CDE Club of IDA North Malabar Branch was held at Hotel Mascot on the topic ORAL MUCOSAL LESIONS by Dr Aswin.

FAMILY TOUR : 14 members with their family participated in a 2day tour programme to Mysore on 15th &16th of may 2010 . The group visited Vrindavan gardens, Karangi lake, Mysore Zoo, Mysore Palace .

KODUNGALLUR

The 4th CDE programme was held on 30.04.2010 at IMA hall Kodungallur. The class was conducted by Dr. Balagopal Varma. The topic was 'Pulp Therapy in DeciduousTeeth'.

Our 5th Dental Health Check-up camp was conducted at Kalariparambu School, Mathilakam on 06.03.2010. 230 patients where examined.

The 5th CDE programme was held on 24.06.2010 at IMA hall Kodungallur. The faculty Dr. Mahesh Narayanan conducted a class on ' Applications of Laser in Dental Clinics'.

Our 5th GBM was conducted at IMA hall, Kodungallur on 30.04.2010.

Our 6th GBM was on 24.06.2010 at IMA hall, Kodungallur.

3rd Executive Meeting conducted at IMA hall, Kodungallur on 03.06.2010 to discuss about our family trip and journal publication.

There was a funfilled family get-together on 23.05.2010 at IMA hall, Kodungallur. It was a very interesting programme with variety entertainment by members and their families.

Homage...

Dr. B. Retnamma

Dr. B. Retnamma was born in an aristocratic family in Aleppey during the year 1937. She took her BDS from the Govt. Dental College, Trivandrum in 1960. Later she took MDS in Conservative in the University of Kerala.

Dr. Retnamma has started her professional career as the dental surgeon in T.D. Medical College, Aleppey. She had prolific private practice. Later she opted medical education service. Dr. Retnamma retired as professor of conservative dentistry from Dental College, Trivandrum. After retirement she has worked as principal of Rajas Dental College, Vadakkankulam, Tamilnadu.

Soft spoken, compassionate and kind hearted, she was loved by all. She was married to Dr. Gopakumaran Nair and has two daughters. All are dental surgeons.

May the almighty give her eternal peace.

Dr. P. Rajasekharan Pillai

Dr. P. Rajasekharan Pillai graduated in dentistry from the Osmania university and later took MDS in Periodontics from Govt. Dental College Trivandrum. Dr. Pillai joined the Health Services as Dental Surgeon and retired as Civil Surgeon. After retirement, he joined Amrita School of Dentistry as Superintendent and worked in the post till his last day. He was instructed in the function and development of the Amrita School of Dentistry. Dr. Pillai was the founder president of the Kerala Govt. Dental Specialists Association. He is survived by wife and one son.

Dr. K.G. Gopakumar

Dr. K.G. Gopakumar was a graduate and Post-graduate of Govt. Denal College, Trivandrum. He joined the Department of Orthodontics as teaching faculty. He retired as professor. Later he joined Pariaram Dental College as principal. He is survived by Wife and two children

Dr. P.I. John

Puthusseril Ittoop John graduated from the prestigious R.A. College of Dentistry, Caulcutta in 1956. He took MDS in Oral and Maxillofacial Surgery from the university of Bombay in 1967. He had training in U.K. under the Common Wealth Scholarship scheme for 1 year.

Dr. John joined the Govt. Service in Kerala as Honorary Dental Surgeon at Palaghat and worked for about 2 years. He was one of the first to join a teaching faculty in the Dental College, Trivandrum in the year 1958. He had a long and meritorious service at the Dental College, Trivandrum. He retired as joint Director of Medical Education in 1984. After retirement he worked mostly in the Dental College at Erode, Tamilnadu.

Gentle, pleasing and kind hearted, Dr. John commanded respect from the staff and students alike. He is one of the founder members of the IDA, Kerala state branch. Dr. John married Smt. Leela and they have four daughters. May his soul rest in peace.

Dr. M.K. Mani

Meppadathu Kurian Mani belongs to Ernakulam. He had graduated from the R.A. College of Dentistry, Calcutta. He took his Masters in Conservative Dentistry from the University of Bombay in 1965. Dr. Mani started his official career as a Tutor in Govt. Dental College, Trivandrum. After his retirement, he serviced as Principal of different Dental Colleges of Goa an Karnataka state. Though very simple and humble in behaviour, Dr. Mani was strong willed person. He has contributed immensely to the development of Trivandrum Dental College during is infancy. Dr. Mani married Dr. Anna who retired as professor of Preventive Medicine. They have a son Rana Kurian. May his soul rest in Peace.