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Department of Preventive and Community Dentistry, College of Dental Sciences Davangere, Karnataka

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MOLECULAR BASIS OF CELL CYCLE REGULATION BY CYCLIN : A SHORT REVIEW

Authors:

Soma Susan Varghese[1]
Jithin Jose[2]
Philips Mathew[3]

[1]Reader,
Department of Oral and Maxillofacial Pathology,
Mar Baselious Dental College and Hospital, Kothamangalam, Kerala.

[2]Reader, Department of Oral and Maxillofacial Pathology,
Indira Gandhi Dental College,
Kothamangalam, Kerala.

[3]Assistant Professor,
Department of Oral Medicine and Radiology,
Government Dental College,
Kottayam, Kerala.

Address for correspondence:

Dr. Soma Susan Varghese MDS,
Reader,
Department of Oral and Maxillofacial Pathology,
Mar Baselios Dental College and Hospital,
Thankalam, Kothamangalam,
Kerala, India.
E mail- drsomasusan@yahoo.in
Phone +91 9943066231
+919995566970

ABSTRACT

Eukaryotic cell cycle is under the control of Cyclin Dependend Kinase enzymes which is regulated positively by Cyclins and negati - vely by inhibitors of Cyclin Dependent Kinase (CDK). pRB, the fundamental component of cell cycle restriction point is phosphorylated and dephosphoryated by Cyclin Dependent Kinase enzyme. CDKI (p21) is transcriptionaly regulated by p53, which focus on the major role of Cyclin on cell cycle check point cascade. Dysregulation of normal cell cycle pathway can result in carcinogenesis.

Key words : Cyclin, dependend, kinase, cyclin, Phostho relation.

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Introduction:

The progression of eukaryotic cell cycle is governed by a family of Cyclin-dependent kinases (CDKs), whose activity is positively regulated by sequential formation and degradation of a group of proteins called Cyclin[1]. Activation of specific Cyclin-CDK complexes results in a cascade of protein phosphorylation that is required for the passage through the key checkpoints in cell cycle. Therefore, dysregulation of Cyclin expression can result in the loss of control over cell cycle and ultimately result in tumour development. Cyclins are functionally divided into two groups. The G1 Cyclins (C, D1-D3, E), regulating the passage of cells through the G1 phase and their entry into the S phase, and the mitotic Cyclins (A, B) facilitating the cell through the mitotic phase. Through cell cycle progressions, Cyclin D/CDK4 and Cyclin D/CDK6 are the first complexes to become active, appearing during mid-to-late G1 phase. They are followed by Cyclin E/CDK2 complex in late G1 phase. Cyclin E/CDK2 complex is present in the cell cycle progressions until the cell completes the G1/S transition. Active CyclinA/CDK2 complexes drive S phase progression, and after the cell has entered G2 phase, Cyclin A trades its partner, CDK2, for CDK1. The G2/M phase transition heralds the appearance of Cyclin B-CDK1 [2,3].

II. Cell cycle regulation by Cyclin

Cyclin D is degraded through ubiquitin-proteosome pathway. During G1 phase of the cell cycle; Cyclin D binds to and activates CDK4, forming Cyclin

D-CDK4 complex. This complex has a critical role in the cell cycle by phosphorylation of the retinoblastoma susceptible protein (RB). The phosphorylation of RB protein is the on-off switch of the cell cycle. In the hypophosphorylated state, RB prevents cells from replicating by forming a tight inactive complex with the transcription factor E2F[4].

Phosphorylation of RB dissociates the complex and releases the inhibition on E2F transcriptional activity. Thus RB phosphorylation eliminates the main barrier of cell cycle progression and promotes cell replication. Hypophosphorylated RB present in or early G I phase binds to a protein complex that contains E2F and a subunit called DPI. E2F/DP1/RB complex binds to the promoters of E2F responsive genes. Bound to E2F/DP1/RB complex, such genes are silent because RB recruits Histone deacetylase, an enzyme that causes the compaction of chromatin and inhibition of transcription. When quiescent cells are stimulated by growth factors, the concentration of Cyclin D and Cyclin E will be elevated, resulting in the formation and activation of Cyclin D - CDK4 and Cyclin E-CDK2 at G1/S restriction point and causes phosphorylation of RB (Fig-1).

Hyperphosphorylated RB dissociates from the complex, activating the transcription of E2F target genes that are essential for the progression through S phase. These include cyclin E, DNA polymerase, thymidine kinase and dyhydrofolate reductase[4,5].

Cyclin A expresses from late G1 phase, reaches a maximum during S phase, and is degraded during mitosis just before the metaphase. Cyclin A-CDK2 complex regulates events in mitotic prophase. It is thought to be required, in association with CDK2 and CDC2 (cell-cycle division2), for DNA synthesis during the S phase and progression through the G2/M transition, respectively. Cyclin A has been implicated in cellular transformation by forming complexes with adenovirus E1A protein, transcription factors DP-1 and E2F, and the retinoblastoma protein. Ectopic expression of Cyclin A can lead to adhesion-independent cell proliferation and advance cell entry into S phase [6,7].

Cyclin A over expression is found in a wide variety of human tumors, like esophageal, non small cell lung cancer, hepatocellular, renal, breast, and prostate carcinomas as well as soft tissue sarcomas. Over expression of Cyclin A protein in oral Squamous Cell Carcinomas (oral SCC) could result from gene amplification, mRNA over expression and impairment of proteolytic degradation by a variety of molecular biological mechanism (e.g. oncogene activation, inactivation of tumor suppressor genes or growth factor stimulation) have been postulated as playing a role in oncogenesis of oral SCCs and other tumors[8].

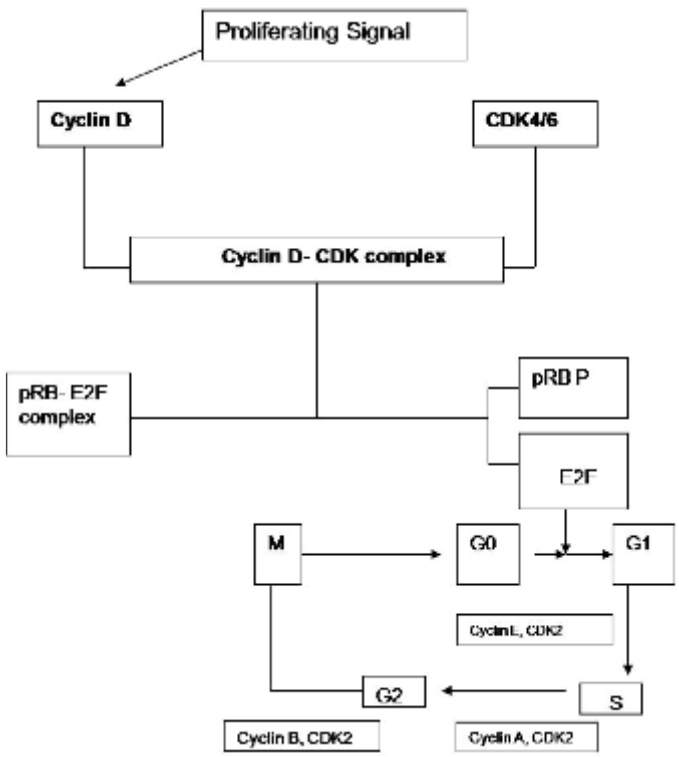
Cyclin dependent kinase inhibitors (CDKI). CDKI's in mammals fall in to two general families; the p21 family (p21Cip1/WAF1, p27Kip, and p57Kip2), and the INK4 family (p15INK4b, p16INK4a, p18INK4c, and p19INK4d). The CDK inhibitor p16 binds to CDK4 and CDK6 and inhibits phosphorylation of Rb, leading to G1 arrest [9]. Loss of p16 function has been demonstrated in a wide vari-

ety of human tumors, including oral and esophageal cancer. P21 acts on multiple Cyclin/CDK complexes to arrest the G1/S-phase of the cell cycle. This protein is frequently expressed in epithelial cells and may play a role in maintaining cells in a non-mitotic condition. p21 has been shown to be transcriptionally regulated by the tumor suppressor protein p53, providing an important possible explanation for P53 mediated cell cycle G1 arrest. p21 induction can also be accomplished by a p53 independent pathway. Over-expression of Rb protein has been shown to block p53-mediated apoptosis, and loss of Rb function has been shown to result in an induction of apoptosis in response.

III. Cyclin as a prognostic marker

Increased expression of cyclin is found in Oral squamous cell carcinoma and epithelial dysplasia. This is suggestive of the key role of mutated Cyclin in tumour progression[10]. Elevated expression of Cyclins, CDK2 and loss of p12DOC-1, p16INK4A and p27KIP1 may contribute to the multistep nature of oral carcino genesis. The mechanisms underlying Cyclin D1 over expression in cancer can be due to gene amplification, chromosomal translocation, and mitogenic stimulation of gene transcription. Cyclin D1 over expression has also been linked to increased risk of occult metastases and poor prognosis in oral cancer patient. Cyclins, the key regulatory proteins of cell cycle check point if mutated can lead to uncontrolled cell proliferation resulting in tumour formation as well as tumour growth. The expression of this molecule can be correlated with the prognosis of the tumour.

Fig.1: Flowchart depicting Cyclin in Cell cycle regulation.



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OBSTRUCTIVE SLEEP APNOEA AND MAXILLOFACIAL SURGERY

ABSTRACT

Obstructive sleep apnoea (OSA) is a disease of the upper airways that leads to a cessation of or a significant decrease in breathing during sleep leading to increased fatigue, difficulty in concentrating and in some cases leading to cardiovascular diseases and even stroke. It affects millions of people around the world. This condition in many of the individuals remain undiagnosed while those who are diagnosed often exhibit poor compliance with the use of Continuous Positive Air Pressure (CPAP), a very effective non-invasive modality. Maxillo-mandibular advancement (MMA) surgery is the most successful treatment for the severe form of this disease. This article reviews various Oral and Maxillofacial management options for the treatment of OSA.

Keywords: Obstructive Sleep Apnea, Maxillo-Mandibular Advancement, Continuous positive airway pressure.

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Authors:
Fiaz Shemshudeen[1]
Joju George[2]

[1]Reader
Department of Oral and Maxillofacial Surgery,
Indira Gandhi Institute of Dental Science,
Kothamangalam, Ernakulam Dt., Kerala

[2,]Sr. Lecturer
Department of Oral and Maxillofacial Surgery,
Indira Gandhi Institute of Dental Science,
Kothamangalam, Ernakulam Dt., Kerala

Address for correspondence

Dr. Fiaz Shemshudeen
Reader
Department of Oral and Maxillofacial Surgery,
Indira Gandhi Institute of Dental Science,
Kothamangalam, Ernakulam Dt., Kerala,
Email- fiazshems@gmail.com
Phone- 9562562566

Obstructive sleep apnoea (OSA) is a disease of the upper airway that leads to a cessation of or a significant decrease in breathing during sleep leading to increased fatigue, difficulty in concentrating and in some cases leading to cardiovascular diseases and even stroke. The term orthognathic is derived from the Greek word "ortho"- straight and "gnathos"- jaw. It includes a wide range of maxillary and mandibular osteotomies done for the correction of deformities of the facial skeleton to achieve proper form, function, and esthetics. Maxillo-mandibular advancement surgery (upper and lower surgical jaw repositioning) is the most successful treatment of the severe form of the disease.

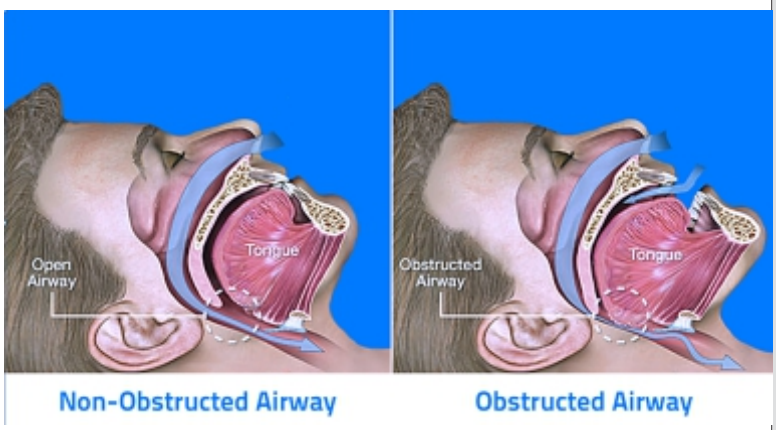
Obstructive sleep apnoea is a chronic disruptive condition in which the person experiences many pauses and episodes of shallow breath while asleep. Pauses in breathing can last a few seconds to a few minutes and typically followed by a chocking sound. The disruption in normal breathing results in poor quality sleep which may lead to disastrous results. This potentially life threatening disease affects 12% of the adult population. Adults are not the only ones affected with estimates putting the children affected by this condition at around 3%. Nearly 80% of all moderate to severe sleep apnoea cases are undiagnosed according to the American sleep apnoea Association.

Diagnosis:

Most of those affected by this condition is made aware by family members. To correctly diagnose OSA the person should see a doctor or a specialist who will make a diagnosis based on the existing signs and symptoms, family history, further examination, or get additional analysis from a sleep specialist. At a sleep center, monitored sleep studies can be administered to a patient to diagnose sleep apnoea and other sleep disorders. The test for diagnosing sleep apnoea is polysomnogram (PSG). It records various relevant biometrics, eye movements, heart rate, blood pressure etc. The results are reviewed by a sleep specialist who will make a diagnosis and help patients with appropriate treatment plan.

Signs of sleep apnoea

1. Daytime fatigue and sleepiness
2. Difficulty in concentration during daytime
3. Disruptive snoring
4. Pauses in breath while sleeping
5. Waking up to a dry mouth or sore throat
6. Waking up with a headache
7. Mood swings
8. Irritability
9. High blood pressure
10. Night time sweating



What happens?

Pharynx is the most common site of obstruction. This may be due to large tongue size, small airway or abnormal anatomy.[1] People with OSA have disrupted sleep and low blood oxygen levels. When OSA occurs, the tongue is sucked against the back of the throat. This blocks the upper airway and airflow stops. When the oxygen level in the brain becomes too low the carotid sinus is stimulated, and the sleeper partially awakens. The obstruction in the throat then clears and the flow of air starts again usually accompanied by a loud gasp. This apneic episode can go on from 5 - 100 times a night or even more. The average number of episodes per hour of sleep is called Apnea-Hypopnea Index (AHI) and is classified into mild (5-15), moderate (15-30) and severe (>30).[2] These episodes are frequently associated with snoring, but it is not considered to be a diagnostic factor.

Repeated cycles of decreased oxygenation may lead to very serious cardiovascular problems. They may

also suffer from excessive daytime sleepiness, depression and loss of concentration. Some have a less severe obstruction called upper airway resistance syndrome (UARS). In either case the individual suffering may have the same symptoms.

The first step in treatment is to recognize the symptom and seek consultation. Oral and maxillofacial surgeons can offer consultation and treatment options. In addition to a detailed history, the doctor will assess the anatomic relationship in the maxillofacial region. With cephalometric analysis the doctors can assess the level of obstruction. Sometimes a nasopharyngeal examination is done with a flexible fibro-optic camera. To confirm the amount of cardiovascular compromise and in case of decreased oxygen levels, a sleep study may be done to monitor the individual overnight.

Examination should include assessment of systolic and diastolic blood pressure, neck girth, evaluation of upper airway to assess the status of uvula, soft palate, tonsils and tongue size, low level of hyoid bone or maxillo-mandibular deficiency.[3]

Mallampati Score (Grade 1- 4) evaluates the size of the tongue in relation to the oral cavity. An increased score suggests that tongue could be the cause of obstruction.[4] Epworth sleepiness scale is a questionnaire used as a subjective measure of a patient's daytime sleepiness. There are many other grading systems which may be used to assess sleep and apnoea.

Lateral cephalometric radiographs are used to assess the size of the posterior airway space, the length of the soft palate and the distance from the mandible to the hyoid bone which are beneficial for decisions concerning surgical management. Cephalometric analysis is very important in OSA patients in diagnosis and treatment planning.[5] Cone-beam computed tomography (CBCT) scans can be used to assess anatomic deformities.[6] Polysomnography is the gold standard test. It is a detailed overnight sleep study which records many functions like brain activity, oxygen saturation, heart rhythm, breathing rate, muscle activity and eye movements. Respiratory disturbance index (RDI), AHI and oxygen desaturation index (ODI) are also used as a screening and diagnostic test.[7]

Treatment

There are several treatment options available ranging from noninvasive to invasive methods. The initial treatment may consist of using nasal continuous positive airway pressure (CPAP) machine that delivers pressurized oxygen through a nasal mask to limit obstruction at night. The non-invasive treatment options include life style modification, postural training and oral appliance therapy.

Oral Appliance (OA) Therapy for lower jaw advancement

Different types of dental splints are available to treat mild to moderate obstructive sleep apnoea (OSA). It looks similar to a sports mouth guard and is worn over the upper and lower teeth during sleep. It is suitable for patients with mild to moderate OSA, mainly with tongue level obstruction. The patient's gums and teeth must be healthy and stable in order for the dental splint to anchor well and push the lower jaw forward.

OA positions the lower jaw forward to open up the airway at the back of the tongue. Adjustments may be necessary in the first few months and the OA may need to be replaced after an average of 5 years.

An adult fitted with an adjustable OA with connectors at the side.

A Tap-T oral appliance with a connector in front.

Surgical Options

Surgery is sometimes recommended for severe cases of obstructive apnoea. It is the next option for patients with moderate to severe OSA who are non-compliant to CPAP therapy. Patients must be medically fit to undergo surgery.

All cases for surgery are examined thoroughly in order to customize the treatment plan. Soft and hard tissue surgery are planned to cater to the needs of each patient. In many instances, a multidisciplinary

approach is taken and a team of surgeons is brought in for combined surgery. Sometimes, it may be necessary to stage surgical treatment into separate surgeries done at different times. Oral and maxillofacial surgeons carry out jaw surgery. The otolaryngology (ear, nose and throat) surgeons carry out surgeries to the nose, palate and throat.

Invasive therapy includes surgeries such as uvulo-palato-pharyngo-plasty (UPPP), laser assisted uvulopalatoplasty (LAUP), hyoid suspension and tongue base reduction. Common oral and maxillofacial approaches include genioglossus advancement (GGA), advancement genioplasty, maxillomandibular advancement (MMA) and distraction osteogenesis (DO).[8] The definitive and accepted treatment for OSA is Continuous positive airway pressure (CPAP) but this have a high non-compliance rate.[9] The side effects include the stuffy dry nose, nasal irritation, claustrophobia, noise from the machine, disturbed sleep, unintentional removal of the apparatus during sleep, difficulty in initiating sleep and gastric disorders. Studies comparing oral appliances (OA) and CPAP have shown that the former is less effective (15-55% success), but owing to the side effects of CPAP, patients prefer oral appliances.[10]

The surgical procedures can be divided into 2 stages.

Stage I includes UPPP and GGA, with or without hyoid myotomy.

Stage II includes MMA and is carried out where stage I surgeries may not change the status of the patient. The main aim of the surgical therapy is to cure the disease but it is accompanied by complications such as the post-operative pain, discomfort, edema, risk of surgery and the uncertainty of the success of the procedure.

1. Genioglossus Advancement with/without Hyoid Myotomy - The surgery focuses on either the reduction of tongue mass or the advancement of tongue attachments. The genioglossus and the digastrics muscles are repositioned anteriorly which results in a more anterior position of tongue and also a change in position of the geniohyoid muscle which pulls the hyoid bone superiorly.[11] This is done in patients with a Respiratory Distress Index (RDI) of above 15 per hour of sleep and patients who are reluctant to the use of CPAP. This procedure may also be done in combination with UPPP or MMA in patients with multiple obstructions.
2. Distraction Osteogenisis (DO)- is a technique developed by Illizarov for the correction of deformities of limbs and was later adapted to maxillofacial surgery for the correction of facial deformities using advancement procedures. The technique has five phases: surgery, latency, consolidation, and removal of appliance. Lu et al. described DO as a reliable surgical technique to improve narrow upper airway in young patients. Advancement upto 25 mm has been reported and relapse is comparatively less compared to conventional osteotomies.[12] It is better to advance the mandible using DO because the procedure can be stopped once the desired advancement is achieved and the gradual incremental movements also provides accommodation for the soft tissues, thereby increasing stability, preventing nerve damage and also protecting the tempero-mandibular joint.
3. Maxillo-mandibular advancement (MMA) - considered as a phase II therapy because of its aggressive nature, is considered the most predictable surgical treatment. Patients with severe Maxillofacial skeletal deformity are the best candidates. It causes an expansion in the skeletal framework including the nasal, pharyngeal and hypopharyngeal airways leading to airway expansion and reduces lateral pharyngeal wall collapse. Patients with severe maxillo-mandibular deficiency, young patients with OSA who needs a permanent correction, patients who prefer a single stage surgery are thought to be ideal candidates for MMA.[13] Surgeries include: Maxillary advancement with Le Fort I osteotomy which pulls forward the velum and valopharyngeal muscles along with the maxilla.

   Mandibular advancement with bilateral sagittal split osteotomy (BSSO) along with genioplasty advances the tongue and the suprahyoid muscles along with the mandible. Maxilla is advanced first followed by the mandible which is moved into occlusion. There is a 70-100% success rate with 90% improvement in the quality of life in those who had MMA.[14] The limitation is that

advancement of more than 10-12 mm is not possible due to soft tissue limitations and the tendency to relapse with larger advancements. It is an invasive and complex surgery with potential complications such as profuse bleeding, infection, paresthesia, occlusal disharmony and esthetic changes.

Conclusion

OSA is a common disorder which can lead to serious complications, but not diagnosed routinely. It requires a multidisciplinary approach for the proper diagnosis and management. There are different modalities for the treatment ranging from behavioral management to invasive surgical procedures. Oral appliance therapy is less effective compared to CPAP therapy. Sleep apnoea surgery has advanced with new surgical techniques and instruments. Dentists must be able to recognize the disease by early evaluation and propose a treatment plan or refer to a specialist for further definitive treatment.

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OBLIQUE FRACTURE OF PARASYMPHYSIS

CASE REPORT AND MANAGEMENT

Authors:
Anoop Kurian Mathew[1]
Anis Ahmed[2]

[1,2]Reader,
Department of Oral Medicine and Radiology,
Indira Gandhi Institute of Dental Sciences,
Kothamangalam, Ernakulam,
Kerala, India.

Corresponding author:

Anoop Kurian Mathew
Reader,
Department of Oral Medicine and Radiology,
Indira Gandhi Institute of Dental Sciences,
Kothamangalam, Ernakulam,
Kerala, India.

ABSTRACT

Mandibular fractures are more common among maxillofacial trauma. Fractures of the parasymphyseal region of mandible occur in 15% of all cases of mandibular fracture. The diagnosis of mandibular fractures must begin with a careful history and clinical examination. Mandible is one of the strongest bone of facial skeleton, but it is frequently fractured due to its prominent position, mobility, anatomic configuration as well as less bone support. This paper presents a case report with a facial trauma affecting the mandibular parasymphysis with an emphasis on its management.

Keywords : Fracture, Mandibular parasymphysis, ecchymosis

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Introduction

Mandible is strong bone with a prominent position in the facial skeleton and has a specific anatomic configuration. Fracture of mandible accounts for 36% to 59% of maxillofacial trauma[1]. The most typical causes for fractures of mandible were fall (64%), traffic accidents (22%) and sports related accidents(9%)[2]. Following any form of orofacial trauma, patient can be affected psychologically, which can usually affect aesthetic as well as functional structures[3].

Majority of fractures of mandible are undisplaced because of elasticity of mandible and embedded tooth buds that holds the fragments together "like glue"[4]. If any of the fractures are displaced, then closed reduction and immobilization are performed. This is followed by intermaxillary fixation and surgical plating with screwing to be done with regular follow up[5].

This paper will review the triage, evaluation and management of an oblique parasymphyseal fracture of mandible in a middle aged man.

Case Report

A 41year old male patient reported to our department with chief complaint of painful swelling over the right side of the face. In history of present illness, he gives history of swelling present for the past 1 week which is associated with pain following a trauma 1 week back (hit by log). Swelling was of sudden onset following trauma and gradually increased to the present size since last 4 days. Associated pain was throbbing type, increases on touch, relieved upon no medication. Patient also reported with difficulty in wide opening of his mouth, also associated with difficulty in mastication, swallowing as well as phonetics. However, not associated with loss of consciousness or bleeding from nose, ear or any vomiting. Patient also gave history of fever present before 2 weeks. There were no relevant past medical, past dental as well as family history.

On extraoral examination, on inspection, the swelling was diffuse over the right side of the lower third face measuring approximately 5×3 cm in size which is roughly oval in shape. It extends anteriorly 2.0cm from the midline, posteriorly 4cm from the right angle of the mandible. Superiorly it extends 2cm below the imaginary line drawn from the right ala of nose to the tragus of the ear. Inferiorly it extends 2cm below the inferior border of the mandible. Skin over the swelling appeared to be normal overall except for a small area of ecchymosis in the anterior part of swelling. There were no surface ulcerations, bleeding or any bleeding spots present over the skin. On palpation, inspectory findings such as extent, size and shape were confirmed. The swelling was tender, is firm hard in consistency. It was non mobile, non-fluctuant, non-compressible, non-reducible and hard in consistency. There was local rise in temperature.

On local intraoral examination, on inspection, swelling was appreciated on the lingual aspect of 44, 45. The swelling extends from mesial aspect of 44 upto the mesial aspect of 45. It was 1 cm in diameter with normal surrounding mucosa. In addition, there were lacerations present in relation to 44, 45 with profuse and spontaneous bleeding. Healed ulcerations (scars) were present on the right side of upper and lower lip and over the labial mucosa. Obliteration of the right buccal vestibular area was well appreciated. Mouth opening and occlusion was normal with no step deformity. But generalized attrition of teeth was appreciated. On palpation, inspectory findings such as site, extent and shape of the swelling were appreciated. Swelling was tender to palpate, no bleeding or discharge present. Tenderness of right buccal vestibule was present.

Fig 1. Extraoral examination revealed the presence of a diffuse swelling over the right mandible

Fig 2& 3. Intraoral examination revealed lacerations with respect to 44, 45 & healed ulcerations over labial mucosa

Bleeding was appreciated through the lacerated wound in relation to 44, 45. There was grade II mobility in relation to 45.

Based on the history given by the patient and the clinical examination carried out, a provisional diagnosis of mandibular body fracture was given.

Differential diagnosis of Dentoalveolar fracture was considered but here there is mobility of the entire segment in the region of fracture. There may be occlusal disharmony.

Panoramic radiograph was taken as part of investigation which revealed an oblique radiolucent band suggestive of a fracture line starting obliquely from the interdental region in relation to 44 and 45, runs downwards and backwards to involve the inferior border of mandible. Also reveals 45, the tooth in line of fracture. However there is no displacement of the fractured segment pf right mandible.

Considering the history, examination and investigation, final diagnosis of Oblique Fracture of right parasymphysis was given.

Treatment plan made include the following:

Preliminary treatment - Control of bleeding, antibiotic coverage, analgesics

Followed by Surgical management with extraction 45, complete oral prophylaxis with restoration of 14, 15, 18, 38, 47, and 48.

Following medications were prescribed:

Amoxicillin (Cap. MOX) 500mg TID - 5 days.

Metronidazole (Tab.METROGYL) 400mg TID-3days .

Diclofenac sodium (Tab. DICLO) 50mg BD - 5days

Ranitidine (Tab. RANTAC) 150 mg BD - 5 days.

Patient was then admitted. Surgical plating and screwing was carried out after intermaxillary fixation.

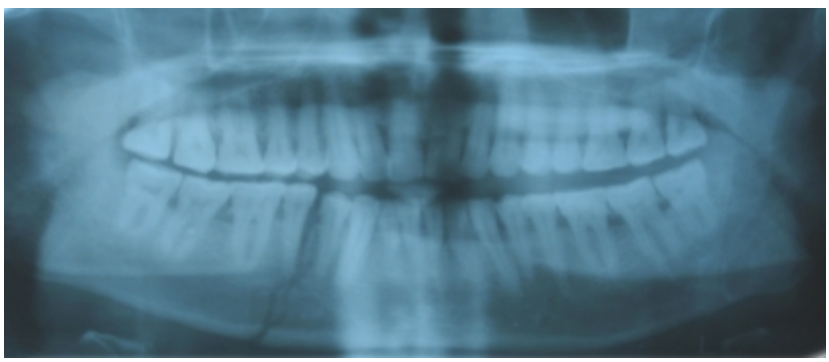

Fig 4: Panoramic radiograph revealed an oblique radiolucent band suggestive of a fracture line on right parasymphysis



Fig 5,6,&7: (A)Intermaxillary fixation. (B) Fracture line detected. (C) Surgical plating & screwing with respect to the fracture site in right body of mandible.

Fig 8: recall after 3 weeks

Discussion:

One-third of lower facial skeleton is composed of mandible which is at a great risk for direct external trauma. Fractures of mandible are usually common in adults as well as in pediatric patients[6]. Mandible fractures are usually seen in the weak portions of the jaw. Angle fracture is most common among all fractures that occur following any form of violence[7]. Vertical fractures are generally seen in following direct trauma and horizontal fractures are usually rare. The clinical signs and symptoms of a fractured mandible usually includes pain, swelling, trismus, derangement of occlusion, sublingual hematoma, step deformity, deviation, loss of sensation due to nerve damage, bleeding, ecchymosis, temporomandibular joint (TMJ) problems, tenderness, movement restriction. However, for the present case, signs and symptoms were pain, swelling, bleeding, ecchymosis and tenderness. In a study done by Posnick and colleagues' , thirty-nine percent of all the fractures were of the mandible[8].

The main goal of management is to restore the underlying bony architecture to its preinjury state with minimal residual esthetic and functional impairment[9]. The management in child differs from that of adult because of concern for possible disruption of growth[10]. In severe fractures, the treatment options can vary from intermaxillary fixation, cap splints to plating with mini plates or resorbable plates. Mandibular parasymphyseal fractures without displacement and malocclusion are managed merely by close observation, a liquid-to-soft diet, avoidance of physical activities such as sports and use of analgesics[11,12]. For minimally displaced fractures, conservative closed reduction is the most frequently recommended treatment. The closed reduction and immobilization approach can be achieved by means of lingual acrylic splints, circumferential wiring, arch bars, or gunning splints[13]. In the present case report, patient was under close observation following reduction and intermaxillary fixation was done for minimally displaced fracture. Surgical plating and screwing was done after the wiring.

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PROSTHETIC MANAGEMENT OF A UNILATERAL CLEFT PALATE PATIENT
A CLINICAL REPORT

Authors:
Mohammed Shahid[1]
Nidhin Alex[2]
Fathima Seethi[3]
Afsa Ahmed[4]

[1]Senior lecturer,
Department of Prosthodontics,
Indira Gandhi Institute of Dental Sciences, Kothamangalam.

[2]Senior Resident,
Department of Prosthodontics,
Govt. Dental College, Calicut.

[3]Senior lecturer,
Department of Prosthodontics,
Indira Gandhi Institute of Dental Sciences, Kothamangalam.

Address for correspondence:

Dr. Mohammed Shahid
Senior lecturer,
Department of Prosthodontics,
Indira Gandhi Institute of Dental Sciences, Kothamangalam.

ABSTRACT

Cleft lip and palate is a congenital deformity that is associated with maxillary discrepancies along with dental anomalies. Patients who have not received treatment early in life are the most challenging to treat prosthetically later in life. These patients can be managed prosthetically by obturator prosthesis. This article reviews the management of cleft palate with a closed hollow bulb obturator prosthesis and the steps involved in its fabrication.

Key words: Unilateral cleft lip and palate, Maxillary hollow bulb obturator , functional and aesthetic corrections.

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Introduction

Cleft lip and palate (CLP) is a congenital deformity that is associated with maxillary sagittal, transversal, and vertical discrepancies along with dental anomalies. It is characterized by features such as deficient midface development resulting in a class III tendency, severe maxillary transverse deficiency, alveolar cleft, hypodontia, hyperdontia, and transpositions[1]. CLP patients might also have decreased facial and dental esthetics, resulting in low self-confidence and difficulties in social interactions leading to psycho-social problems[2,3].

Conventional treatment for cleft palate correction includes an interdisciplinary approach involving surgical, orthodontic, and prosthetic and other specialties to obtain functional and esthetic outcomes. Contemporary treatment concepts lay emphasis on early surgical corrections which can improve speech and hearing functions to a considerable amount[4]. Patients who have not received surgical or orthodontic care early in life are the most challenging patients to manage prosthetically later in life[5].

These patients can be prosthetically managed by obturator prosthesis replacing the missing teeth and compensating the tissue deficiencies that interrupt the continuity of the dental arch[6,7].The obturator prosthesis facilitates speech and deglutition by replacing deficient tissues, reduce nasal regurgitation and hyper nasal speech, and improve articulation, deglutition, and mastication. This article discuss about the management of a unilateral cleft palate patient with a closed hollow bulb obturator prosthesis.

Clinical Report

A 34-year-old lady was referred to the Department of Prosthodontics for rehabilitation of a cleft palate defect. The patient's history indicated a congenital cleft lip and palate defect. Patient gives history of cleft lip surgery in early childhood and had not undergone any treatment for cleft palate management.

On examination a unilateral cleft on left side involving the alveolar arch and the entire hard and soft palate was noticed (Fig 1A, 1B). Only teeth present on the maxillary arch was 11, 13, 23, and 25. The edentulous alveolar arch was high rounded with mild bilateral undercut in the maxillary tuberosity region. Mandibular arch was partially edentulous with missing 36, 37, 46, and 47. She had deep overbite, anterior cross bite relationship and had a mouth opening of 34 mm.

The case was discussed and decided to rehabilitate with acrylic maxillary hollow bulb obturator engaging the remaining natural abutment teeth and mandibular removable partial denture prosthesis due to financial constraints. The impression was made with alginate (Tropicalgin, Zhermack) and impression compound (Rolex Impression Composition; Ashoo Sons) using metallic perforated stock tray (Fig 2). The defect area was initially molded with impression compound on the stock tray in order to slightly engage the undercuts on lateral wall of the defect and was extended to half the depth of the cleft. The roof of the defect was then packed with a gauze piece and an alginate impression was made with the stock tray on which the impression compound was molded. The cast was then poured with type III gypsum(Kalstone; Kalabhai Karson Pvt. Ltd.) (Fig 3).

Temporary denture base was fabricated with shellac base plate (Supernal, S.D. Dental Corporation)extending the entire cleft depth. Overbite correction was done during jaw relation record and 2mm of vertical dimension of occlusion was restored. Teeth arrangement was done maintaining anterior cross bite relationship. Try-in was done which was found acceptable by the patient. Circumferential wrought wire clasp was made with 19 gauge stainless steel orthodontic wire on abutment teeth 13 and 25 and the denture was then waxed up for processing.

The denture was fabricated using light pink heat polymerizing acrylic resin (DPI Heat Cure; Dental Products of India) with compression molding technique involving a long polymerization cycle (74°C for 8 hours). The denture was then retrieved which was of open hollow design. The defect area was then filled with table salt and was then covered with light pink self-polymerizing acrylic resin(DPI Cold Cure; Dental Products of India). Upon completion

of the polymerization a small hole was made on the covered region. Water was then syringed through the hole and the saline was then flushed out. The hole was then sealed with self-polymerizing resin turning the obturator prosthesis to a closed hollow bulb design (Fig 4A, 4B). The prosthesis was then inserted in the patient and the retention, stability, esthetics, and occlusion was verified (Fig 5A - 5D). Periodic recall was made on a weekly basis and the necessary corrections were made. A marked improvement in mastication, deglutition, phonetics, and esthetics was noticed and appreciated by the patient (Fig 6A, 6B).

Discussion

An obturator is a maxillofacial prosthesis that is used to close the cleft defect and make separation between oral and the nasal cavities. The prosthesis option can vary from a simple removable partial denture replacing missing teeth and supporting tissues to tooth or implant supported overdentures[5]. Later offers better treatment outcome with respect to the prostheses function, facial esthetics, and patient acceptability. But the treatment is expensive, time consuming, and requires natural abutment teeth at critical positions and sufficient interarch space. A cast removable partial denture offers better stability, retention, and functional outcome[8]. However the treatment is more laborious and expensive.

The design of an obturator is to engage the remaining natural teeth and tissue-bearing areas to optimize retention and stability[9]. Increasing the lateral wall height, and extending the prosthesis into the anterior nasal aperture can enhance the retention and stability of the prosthesis at the expense of its weight and bulkiness[10]. Hollowing the obturator can significantly reduce the weight of the prosthesis. Both open and closed hollow obturators allow for the fabrication of a lightweight prosthesis that is readily tolerated by the patient while effectively extending in to the defect. The closed design can prevent fluid and food collection, reduce air space, and allow for maximum extension[11].

An acrylic hollow bulb obturator was planned in this patient because of financial constraints. The treatment plan pose a dilemma of reduced prosthesis retention, stability, and functional outcome. But the treatment option is more ecocnomical, and less time consuming.

Summary

This clinical report describes the prosthetic rehabilitation of a patient with a unilateral cleft palate that have not been treated in childhood. The patient has been rehabilitated with an acrylic hollow bulb obturator prosthesis that has improved her mastication, deglutition, phonetics, and esthetics. The treatment offers an economical alternative in the management of cleft palate in adults.

Fig 1A. Intra oral view showing palatal defect

Fig 1B. Intra oral view showing anterior extend of the defect

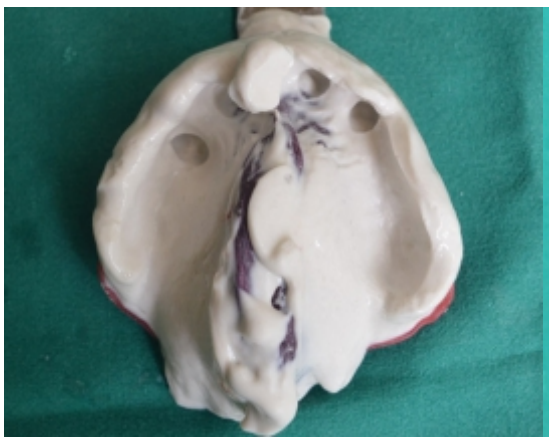

Fig 2. Impression

Fig 3. Master cast

Fig 4A. Hollow bulb obturator: cameo surface

Fig 4B. Hollow bulb obturator: intaglio surface

Fig 5A. Prosthesis insertion: Occlusal view

Fig 5B. Prosthesis insertion: Frontal view

Fig 5D. Prosthesis insertion: Left lateral view

Fig 5C. Prosthesis insertion: Right lateral view

Fig 6A. Pre-operative view

Fig 6B. Post operative view

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APEXIFICATION USING MINERAL TRIOXIDE AGGREGATE

CASE REPORT

Authors:
Varun Mathew Manakunnath[1]
Romel Joseph[2]
Dinesh Kamath[3]
Ajay Joseph[4]

[1,4]Senior Lecturer
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences, Kothamangalam, Ernakulam Dt., Kerala

[2]Prof. & Principal
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences, Kothamangalam, Ernakulam Dt., Kerala

[3]Prof. & HOD
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences, Kothamangalam, Ernakulam Dt., Kerala

Corresponding author:
Dr. Varun Mathew Manakunnath
Senior Lecturer
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences, Kothamangalam, Ernakulam Dt., Kerala
Email: drvarunmathew@gmail.com
Mob. 9446028102

ABSTRACT

It is difficult to treat immature teeth with necrotic pulp and periapical lesion through conventional endodontic treatment.

Use of calcium silicate material in dentistry became popularised due to various chemical application and its inherent advantage namely biocompatibility, sealing ability, regenerative capacity and antibacterial characteristics.

Previously calcium hydroxide was used for apexification but due to its drawbacks modified calcium silicate was introduced to overcome these difficulties.

Keywords: Apexification, calcium silicatemineral trioxide aggregate.

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Introduction

One of the reasons for open apex in developing teeth is trauma and in such cases obturation is difficult. For a successful root canal treatment an apical barrier should be created. The material used previously for hard tissue formation was calcium hydroxide, but it used to take around 6-18 months to do so[1]. Other than the delay in hard tissue formation, its long-term use weakens the dentin and lead to tooth fracture.[2]

Inorder to overcome the drawbacks of calcium hydroxide a new material named MTA - Mineral Trioxide Aggregate was invented by Torabinejad in the year 1993.[3] Studies have indicated that the thickness of hard tissue formation for MTA is more than Calcium hydroxide.[4]

This is the reason why we have used MTA for apexification in the following case.

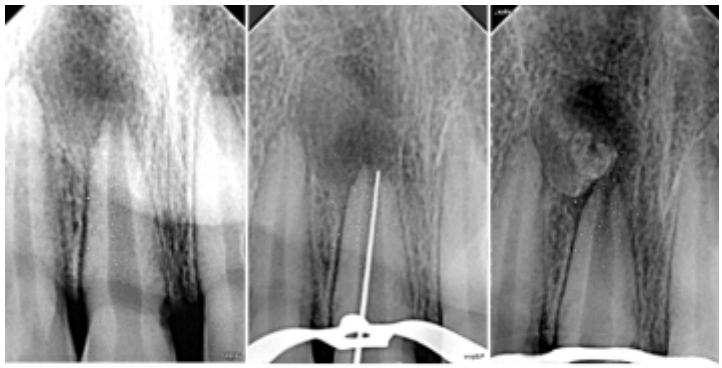
Case Report

A 10 year old boy came to the clinic complaining of pain in the upper front teeth. On taking previous history we understood that the patient met with an accident 2 years back.

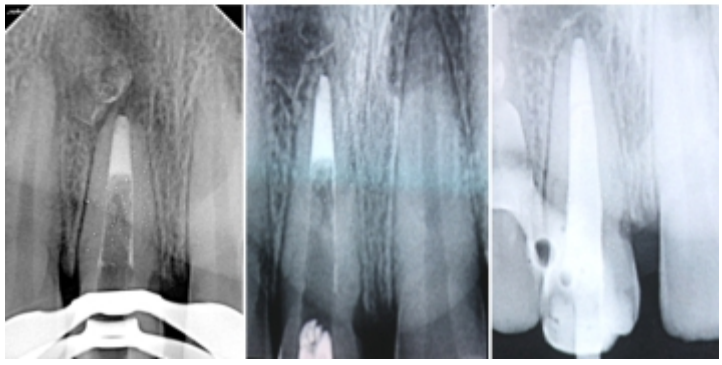
On clinical examination Ellis class III fracture on 11was found. The tooth was tender on percussion. Intraoral radiograph showed incomplete root end formation of 11. On pulp testing there was no response from the patient which indicated that the concerned tooth was non- vital.

The patient was explained the treatment plan in detail and consent was obtained. The treatment was commenced. Access opening was done, working length was determined, cleaning and shaping was done, canal irrigated with 5.25% sodium hypochlorite and normal saline. Calcium hydroxide dressing was given for 2 weeks and temporary restoration was given. The patient was recalled after 2 weeks for review.

After 2 weeks, on examination the tooth was asymptomatic and then the temporary restoration was removed, canal was irrigated and dried with paper point. Suitable plugger was selected and MTA was mixed with distilled water to a required consistency and placed in increments in the apical region of the canal using micro apical placement system and compacted with plugger. IOPA was taken for confirming that there was 5mm of MTA left in the canal. Moist cotton was placed for the material to set and cavity was temporized. The patient was recalled after 24 hours for obturation.



Pre Operative view | W/L Determination | After removal of Ca(OH)2



After MTA placement | 1 week after MTA placement | After Obturation

Discussion

Open apex is one of the many challenges to the endodontist due to the absence of apical barrier. In order to overcome this difficulty the first aim is to induce apical closure. Apexogenesis and apexification are the two treatment options for open apex cases. Apexogenesis is the treatment for main-

taining the vitality in apical part of the root canal for the formation of root apex [5].

Apexogenesis was not possible in our case due to complete necrosis of the pulp. That's the reason we used apexification to obtain apical closure. Calcium hydroxide which was previously used for apexification had pH of 12.5 which was highly alkaline caused changes in the mechanical behaviour of dentin which led to root fracture. [2,6]

Torabinejad introduced MTA as the material of choice for apexification and its constituents are calcium silicate, bismuth oxide, calcium carbonate, calcium aluminate, calcium sulphate. Advantages of MTA are hard tissue formation and bio compatibility. Mechanism of action MTA are it has the ability to produce cementoblast cells which induces hard tissue formation and thereby preventing the entry of microorganisms. [7,8,9,10]

CONCLUSION

The apical barrier formation by using MTA enhances the root strength and decreases the fracture rate of the teeth. In addition to this it reduces the number of visits to the clinician.

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