January 2016, Vol. 4 No. 1

Journal of Clinical Dentistry

Official Publication of Academic Forum, Govt. Dental College, Kottayam

Editorial Office
Dept. of Periodontics
Govt. Dental College
Gandhinagar, Kottayam-686 008
Cell: 9447279605
e-mail: jcdkdc@gmail.com

Production: Suman Graphics, TVM

For Private circulation only

Contents

Editorial

Greetings from Kottayam. This institution has shown remarkable progress in terms of infrastructure and state of art facility for the teaching and learning of Dental Sciences. Also in the forefront of dental health delivery and public health activities in the region. All clinical departments now offer post graduate courses and every year workshops and seminars are being conducted for continuing dental education. The students are well placed in the university results and alumni of this college are spread out across the globe and are enjoying good professional repute.

Is some thing lacking?

Research and development is the way forward for any discipline and it is the right time for our institution to move in that direction. There are limited resources for biomedical research in this region compared to the capital city. But that cannot be an excuse. Now we have a good strength of post graduate students and senior residents who do some research activity mandatorily as part of their training programme. Health sciences research outcomes should ideally be put to some use by the society in future. The scientific community world over has recognized this angle of research with a human touch. The first step towards making the research outcomes beneficial to the society is to publish the work. Peer reviewed journals are a good vehicle for this. The habit of scientific writing and publishing has to be nurtured among young professionals. The publications like Journal of Clinical Dentistry is a small step towards this.

Baiju R.M.
Editor, JCD

'Preterm birth and its implication on delayed tooth eruption'

Deepak D. Kammath [a], Digesh Balachandran [b], Rahul K. [c]

Abstract

Premature child birth is the most frequent cause of low birth weight. Every year, an estimated 15 million babies are born preterm, and this number is rising. Preterm birth complications are the leading cause of death among children under 5 years of age, responsible for nearly 1 million deaths every year. The time of the eruption of the teeth is a valuable clinical maturation index in children. The appearance of an infant's first tooth is regarded as one of the significant landmarks during a child's development; hence most parents are more anxious about the timing of eruption of first primary tooth.

Address for correspondence:

[a] Senior Lecturer, Department of Pedodontics & Preventive Dentistry, Government Dental College, Kottayam.
[b] Assistant Professor, [c] Senior Resident, Department of Pedodontics & Preventive Dentistry, Government Dental College,Trivandrum.

Premature child birth is the most frequent cause of low birth weight. Every year, an estimated 15 million babies are born preterm, and this number is rising. Preterm birth complications are the leading cause of death among children under 5 years of age, responsible for nearly 1 million deaths every year. The time of the eruption of the teeth is a valuable clinical maturation index in children. The appearance of an infant's first tooth is regarded as one of the significant landmarks during a child's development; hence most parents are more anxious about the timing of eruption of first primary tooth.

Preterm birth is defined as babies born alive, before 37 weeks of pregnancy are completed. Every year, an estimated 15 million babies are born preterm, and this number is rising. Preterm birth complications are the leading cause of death among children under 5 years of age, responsible for nearly 1 million deaths every year. Three-quarters of them could be saved with current, cost-effective interventions. Across 184 countries, the rate of preterm birth ranges from 5% to 18% of babies born.

Many survivors face a lifetime of disability, including learning disabilities and visual and hearing problems. Globally, prematurity is the leading cause of death in children under the age of 5. And in almost all countries with reliable data, preterm birth rates are increasing.

Preterm birth can be defined by the estimated gestational age as a proxy of maturity. Three subgroups are distinguished by the World Health Organisation (WHO): preterm (< 37 weeks gestation), very preterm (< 32 weeks), and extremely preterm (< 28 weeks). In the United States of America and several other countries a classification according to birth weight is generally used. Low birth weight infants are defined as those with a birth weight of 2,500 g or less, which may be due to prematurity, being born small for gestational age (SGA), or both. Similarly, lower cut-off limits for weight have been used to describe more severe cases, i.e. very low birth weight (VLBW < 1,500 g) and extremely low birth weight (ELWB < 1,000 g). In very preterm and/or VLBW infants, gestational age is a better predictor of short-term survival than birth weight.

According to WHO criteria, newborn babies weighing less than 2500 g are classified as Low birth weight (LBW), irrespective of the period of gestation and any Neonate born before 37 weeks (less than 259 days) of pregnancy irrespective of the birth weight are classified as Preterm Baby[1].

Why does preterm birth happen?

Preterm birth occurs for a variety of reasons. Most preterm births happen spontaneously, but some are due to early induction of labour or caesarean birth, whether for medical or non-medical reasons. Common causes

of preterm birth include multiple pregnancies, infections and chronic conditions such as diabetes and high blood pressure; however, often no cause is identified. There could also be a genetic influence. Better understanding of the causes and mechanisms will advance the development of solutions to prevent preterm birth.

Premature child birth is the most frequent cause of low birth weight. Preterm infants are at disadvantage because their internal organs are immature, with a higher risk to develop respiratory illness, hyaline membrane diseases, hyperbilirubinemia, hypocalcemia, anemia and other alterations that affect health and growth. There are several factors that cause premature childbirth: too young mothers, low socioeconomic level, intrauterine malnutrition, cardiomyopathy, incompatibility of the Rh factor, rubella, diabetes, multiple childbirth, abuse of smoking, among others.

Where and when does preterm birth happen?

More than 60% of preterm births occur in Africa and South Asia, but preterm birth is truly a global problem. In the lower-income countries, on average, 12% of babies are born too early compared with 9% in higher-income countries. Within countries, poorer families are at higher risk.

Of 65 countries with reliable trend data, all but 3 show an increase in preterm birth rates over the past 20 years. Possible reasons for this include better measurement, increases in maternal age and underlying maternal health problems such as diabetes and high blood pressure, greater use of infertility treatments leading to increased rates of multiple pregnancies, and changes in obstetric practices such as more caesarean births before term. There is a dramatic difference in survival of premature babies depending on where they are born. For example, more than 90% of extremely preterm babies (<28 weeks) born in low-income countries die within the first few days of life; yet less than 10% of babies of this gestation die in high-income settings.

Preterm Birth and Delayed Tooth Eruption

The time of the eruption of the teeth is a valuable clinical maturation index in children. The appearance of an infant's first tooth is regarded as one of the significant landmarks during a child's development; hence most parents are more anxious about the timing of eruption of first primary tooth. Primary dentition stage starts on the arrival of the central incisors, typically from around 6 ± 2 months, and lasts until the first permanent molars appear in the mouth, usually at 6 years.

Birth weight is the single most important marker of adverse perinatal and neonatal outcome. Globally, between 40% and 80% of infant deaths occur among Low Birth Weight babies. Low birth weight (LBW) is indisputably a very important indirect cause of death in neonates the world over.

Influence of preterm birth on teeth development and eruption has been investigated. Most of the studies reported significant oral findings during primary dentition stage. Many studies reported that preterm children have delayed primary and permanent teeth eruption, if emergence time was compared to chronological age. However some researches showed that if eruption time was related to corrected age (i.e. chronological age in weeks minus gestational age 40 weeks), no difference has been found between dental maturation and eruption times of preterm and term children.

Magnusson [3] reported that the timing of deciduous teeth eruption in preterm children was different in comparison with children born after full term gestation and depended on the baby's maturity by birth. *Seow et al*[4] suggested that birth is the stimulus for tooth eruption and that eruption time is the same in preterm infants as for full-term infants. *Ramos et al*[5] reported that, considering the chronological age, preterm infants show a delay in the time of eruption of the first deciduous tooth when compared to full-term infants, with statistically significant difference (p=0.004) however, when the corrected age is considered there was no statistically significant difference (p=0.997). *Viscardi et al* [6] noted that 40% of prematurely born infants had their teeth erupted on time, whereas the remaining 60% had their teeth erupted later, even taking into account the corrected age and they suggested that the nutritional factors and other complications related to preterm birth such as infants who need sustained mechanic ventilation contribute to delay the eruption of the first teeth.

Literature review

Preterm births occur prior to the 37th weeks of gestational age. Low birth weight is when neonate is weighing less than 2500 gram, very low birth weight is less than 1500 gram at birth, and normal birth weight is more than 2500 gram. Advances in the perinatal medicine have increased the survival rate of preterm and low birth weight infants. The incidence of the various major medical complications associated with prematurity and low birth weight has remained relatively stable. Very low birth weight children (VLBW)(< 1.5 kg) especially extremely very low birth weight (EVLBW) (< 1.0 kg), is highly associated with respiratory distress, cardiac disease,

sepsis, necrotizing enterocolitis; metabolic disorders such as hyperbiluribinemia, hypoglycemia, hypocalcaemia, metabolic bone disease, nutritional and neurological disorders [4].

Ramos et al[5] compared the beginning of eruption of the first deciduous tooth in preterm infants (<38 weeks) with full-term infants (38 and 42 weeks) of normal birth weight (2.500g), low birth weight (< 2.500g) and very low birth weight (<1.500g), to evaluate if premature birth and low birth weight affects tooth eruption. Their results showed that when chronological age is considered, tooth eruption in preterm and very low birth weight infants is importantly delayed. However, when corrected age is considered, no statistically significant differences were found among groups. They concluded that the delayed eruption may be related to the premature birth and not to a delay in dental development.

Seow et al[8] compared the dental eruption status of a group of prematurely born, very low birth-weight (VLBW, < 1500 g) children with a group of low birth-weight (LBW, 1500- 2500 g) as well as a group of normal birth-weight (NBW, 2500 g) children in order to determine if dental eruption is affected by low birth weight and prematurity of birth. Data were analyzed using chronological and corrected (true biological) ages of the prematurely born group. Their results showed that when chronological ages of the children were used, VLBW children have significant retardation of dental eruption compared with LBW and NBW children, particularly before 24 months of age ($P < 0.01$) and when corrected ages of the VLB W children were used, there was no significant difference detected. They concluded that the "delay" in dental eruption may be simply due to their early birth.

Lawoyin et al[9] conducted an epidemiological study of factors related to deciduous tooth eruption. This community based study evaluated the eruption pattern of the Saudi children (0-24 months), living in the North West region of the country. No child under the age of 6 months in this study had any teeth. They observed that age and weight had independent effects on the timing of eruption of deciduous teeth. They also concluded that age was highly correlated with the number of teeth erupted, the wide range of normal prevents delay in teething from being a useful index of inadequate growth in this community.

Al-Sayagh et al [10] conducted a study to determine if primary teeth eruption and the presence of enamel defect were affected by low birth weight and prematurely of birth. They found that there was no significant difference between different genders numbers among in each age group for the preterm and control children. The results showed significant delayed eruption of the primary teeth in the prematurely born children They concluded that the eruption of deciduous teeth was delayed and the percentage of enamel defect was significantly increased in prematurely born children.

Ghassan et al[11] investigated the effect of total parenteral nutrition on deciduous teeth eruption of very low birth weight premature infants. It was a prospective study of 85 healthy preterm infants who were born with birth weight of 800- 1500 grams, and at 27-32 weeks gestational age. The study concluded that the introduction of total parenteral nutrition (TPN) to very low birth weight premature infants resulted with early first tooth eruption. It is concluded that intralipids component of TPN is responsible for this observation, but further studies to evaluate the role of other TPN components are mandatory.

How Preterm birth rate can be reduced?

More than three-quarters of premature babies can be saved with feasible, cost-effective care, e.g. essential care during child birth and in the postnatal period for every mother and baby, antenatal steroid injections (given to pregnant women at risk of preterm labour and under set criteria to strengthen the babies' lungs), kangaroo mother care (the baby is carried by the mother with skin-to-skin contact and frequent breastfeeding) and antibiotics to treat newborn infections.

To help reduce preterm birth rates, women need improved care before, between and during pregnancies. Better access to contraceptives and increased empowerment could also help reduce preterm births.

WHO (World Health Organization) response

In 2012, WHO and partners published a report "Born too soon: the global action report on preterm birth" that included the first-ever estimates of preterm birth by country. WHO is committed to reducing the health problems and lives lost as a result of preterm birth with the following specific actions: working with Member States and partners to implement "Every Newborn: An Action Plan to End Preventable Deaths" adopted in May 2014 in the framework of the UN Secretary-General's "Global Strategy for Women's and Children's Health"; working with Member States to strengthen the availability and quality of data on preterm births; providing updated analyses of global preterm birth levels and trends every 3 to 5 years; working with partners around the world to conduct research into the causes of preterm birth, and test effectiveness and delivery approaches for interventions to prevent preterm birth and treat babies that are born preterm; regularly

updating clinical guidelines for the management of pregnancy and mothers with preterm labour or at risk of preterm birth, and those on the care of preterm babies, including kangaroo mother care, feeding babies with low birth weight, treating infections and respiratory problems, and home-based follow-up care; and developing tools to improve health workers' skills and assess the quality of care provided to preterm babies.

Guidelines to improve preterm birth outcomes

WHO has developed new guidelines with recommendations for improving outcomes of preterm births. This set of key interventions can improve the chances of survival and health outcomes for preterm infants. The guidelines include interventions provided to the mother – for example steroid injections before birth, antibiotics when her water breaks before the onset of labour, and magnesium sulfate to prevent future neurological impairment of the child. As well as interventions for the newborn baby – for example thermal care (e.g. kangaroo mother care when babies are stable) , safe oxygen use, and other treatments to help babies breathe more easily.

Conclusion

Advances in the perinatal medicine have increased the survival rate of preterm and low birth weight infants[1]. The incidence of the various major medical complications associated with prematurity and low birth weight has remained relatively stable. However these children are susceptible to many serious illnesses which may cause developmental defects of enamel, delayed tooth eruption etc.

Quality Antenatal maternal care strategies have to be implemented to reduce the incidence of LBW babies and survival of newborns. In order to improve the prenatal, neonatal and postnatal care and to propose more efficient conducts; aiming to avoid the dental and systemic changes of children can be accomplished by various health care programs to mothers.

References

1. Norman T. Nelson's Textbook of Paediatrics.19th ed. New Delhi, India: Saunders Elsevier Publishers 2010; Vol.2 Part XVII-XXXIII: 1529.
2. Meherban Singh, Care of the Newborn, Sagar Publications, New Delhi, India, 2011.
3. Magnusson TE. Emergence of primary teeth and onset of dental stages in Icelandic children. Community Dent Oral Epidemiol. 1982; 10: 91-7.
4. Seow WK. Effects of preterm birth on oral growth and development. Aust Dent J. 1999
5. Ramos SRP, Gugisch RC, Fraiz FC. The influence of gestational age and birth weight of the newborn on tooth eruption. J. Appl. Oral Sci. [Internet]. 2006 Aug;14(4):228–327;42:85–91.
6. Viscardi RM, Romberg E, Abrams RG. Delayed primary tooth eruption in premature infants/ : relationship to neonatal factors. 1994;16(1):23–8.
7. Stark A.R ,Eichenwald E.C - Management and outcomes of very low birth weight. N Engl J Med. 2008; 358: 1700-11.
8. Seow WK, Carolyn F, I DD. Dental eruption in low birth-weight turely born children/ : a controlled study. 1988;10(1):39–42.
9. Lawoyin TO, Lawoyin DO, Lawoyin JO. Epidemiological study of some factors related to deciduous tooth eruption. Afr Dent J 1996; 10: 19-23.
10. Al – Sayagh G Dh, Qasim AA, Al – Rawi BA. The effect of premature birth on the primary dentition Al–Rafidain Dent J. 2008; 8(1):18 –22.
11. Ghassan Saleh Ahmed,Yahya Mohammad and Fadi Farhan Ayyash. Impact of total parenteral nutrition on deciduous tooth eruption of very low birthweight premature infants.Pakistan Oral & Dental Journal Vol 32, No. 1 (April 2012).
12. WHO 2015 recommendations on interventions to improve preterm outcomes-www.whoorganization.org.

‘White-eyed’ blowout fracture: Case series

Shiney Dominic [a], N Jayakumar [b], P G Antony [c], K E Raneesh [d]

Abstract

‘White-eyed’ blowout fracture is a clinical presentation of orbital floor fracture in pediatric patients. The purpose of our study is to define the unique aspects of ‘white-eyed’ blowout fractures in children with regard to injury pattern and its clinical outcome. Methods: Review of two cases with ‘white-eyed’ blowout fractures with inferior rectus muscle entrapment, who underwent surgical repair by a single surgeon. Results: ‘White-eyed’ blowout fractures present uniquely with severely restricted extraocular motility and diplopia, nausea and vomiting, and minimal signs of external trauma. One case was operated within 48 hours and showed complete resolution of diplopia and recovery of normal ocular motility. Second case was operated after one month due to delay in diagnosis which resulted in persistence of restriction in ocular motility. Conclusions: Pediatric orbital floor fracture, often of the trapdoor type, requires early surgical intervention. Our experience signifies early surgical intervention within 48 hours results in better postoperative outcomes.

Keywords: White-eyed, pediatric, blowout, fracture

Address for correspondence:

[a] Associate Professor, [b] Associate Professor, [c] Associate Professor, [d] Junior Resident, Department of Oral & Maxillofacial Surgery, Government Dental College, Kottayam

‘White-eyed’ blow out fracture is the term coined by Jordan and co-workers[1] to describe orbital floor fractures with inferior rectus muscle entrapment in pediatric patients. Blowout orbital floor fractures have been well described and studied in adults[2], however, they have not been as well studied in children. Clinical presentations of pediatric orbital floor fractures are different compared to similar fractures in adult patients which are attributable to variations in the facial anatomy of children versus adults. Trap door pediatric orbital floor fractures comprise 27.8% to 93% of cases[3, 4]. The orbital bones in the pediatric patient are thought to be more flexible, which results in the bones snapping back to cause a “trapdoor” fracture in which entrapment of orbital soft tissue contents occurs.[5] Orbital floor fractures in children are mostly caused by low velocity high force[1]. Review of literature suggests that extraocular motility recovery is quicker and more complete if entrapped muscle is released in the first few days after injury. Conversely, adults are more often found to have comminuted, or “open-door,” fractures, without tissue entrapment, that can be observed for 2 weeks prior to repair[6].

Koltai and colleagues reported that orbital roof fractures are the most common pediatric orbital fracture in very young children.[7, 8]. They reported that after the age of 7 years the probability of lower orbital fractures surpasses the probability of roof fractures. Thus as the face and sinuses develop, orbital floor fractures become increasingly common as a result of blunt facial trauma[4].

The purpose of this article is to evaluate the unique aspects of orbital floor fractures in children with regard to clinical presentation, management, and outcomes.

Case Series

Here we present a series of 2 cases reported to our department with white eyed blowout fracture.

Case 1:

A 14 year old female patient reported with chief compliant of visual disturbance and vomiting. She had a history of trauma to right eye while playing. Clinical examination revealed that she had a right eye superior gaze restriction and diplopia (Fig 1). Patient had no periorbital signs of trauma such as periorbital edema and ecchymosis, subconjunctival hemmorhage.

Hess charting confirmed that patient had diplopia in superior gaze. Computed tomography of orbit revealed

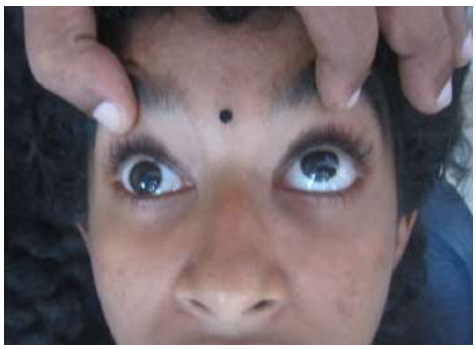


Fig 1: Clinical examination showing restriction of right eye in superior gaze

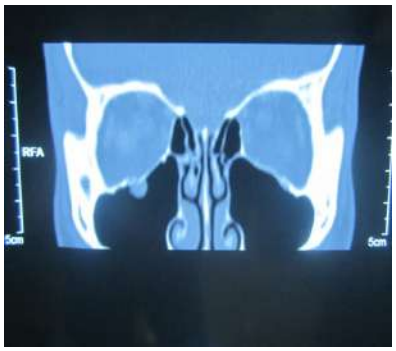


Fig 2: Coronal CT section showing trapdoor fracture of right orbital floor with inferior rectus muscle entrapment

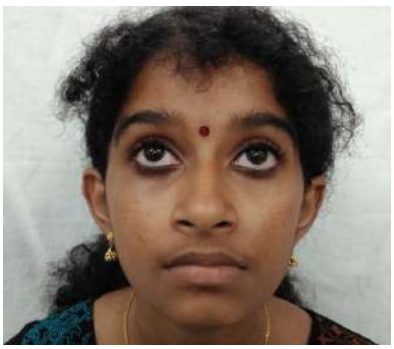


Fig 3: Postoperative photograph showing complete resolution of ocular motility

Fig 4: Clinical examination showing restriction of left eye in superior gaze

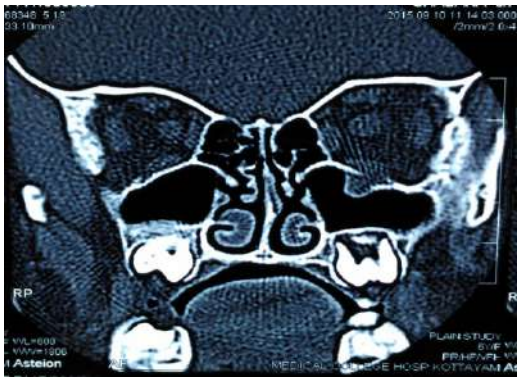


Fig 5: Coronal CT section showing trapdoor fracture of left orbital floor with inferior rectus muscle entrapment

Fig 6: Postoperative photograph showing persistence of restriction in ocular motility

a linear trapdoor fracture of right orbital floor with inferior rectus muscle entrapment (Fig 2).

A diagnosis of 'White-eyed' blowout fracture was made based on clinical and diagnostic investigations.

Surgical procedure was carried out within 48 hours. A standard subciliary incision was made and orbital floor explored, the entrapped inferior rectus muscle was released. Reconstruction of orbital floor was not required as a stable manual reduction of the trapdoor fracture was attained.

Postoperatively, a complete resolution of ocular motility and vision was attained (Fig 3). Patient was followed up for a period of 6 months.

Case 2:

A 6 year old female patient reported with chief compliant of visual disturbance and vomiting. She had history of trauma to left eye while playing one month back. Initially patient consulted local hospital and was admitted to rule out a neurologic cause for vomiting and the orbital floor fracture was undiagnosed.

Clinical examination revealed that she had a left eye superior gaze restriction and diplopia (Fig 4). Patient had no periorbital signs of trauma such as periorbital edema and ecchymosis, subconjunctival hemmorhage.

Hess charting confirmed that patient had diplopia in superior gaze. Computed tomography of orbit revealed a linear trapdoor fracture of left orbital floor with inferior rectus muscle entrapment (Fig 5).

A diagnosis of 'White-eyed' blowout fracture was made based on clinical and diagnostic investigations.

Surgical procedure was carried out. A standard subciliary incision was made and orbital floor explored, the entrapped inferior rectus muscle was released. Reconstruction of orbital floor was not required as a stable manual reduction of the trapdoor fracture was attained.

Postoperatively, a complete resolution of diplopia was obtained but the restriction in ocular motility persisted (Fig 6). On a period of 6 months follow up; superior gaze restriction is still persisting.

Discussion

Most studies have found that early surgical intervention for children with entrapment results in better outcomes than later intervention.[3] When comparing children to adults, some studies suggest that the overall long-term outcomes of children after surgical orbital floor fracture repair (regardless of timing) are worse than those of adults.[9, 10] Cope and colleagues[10] studied 45 children with orbital floor blowout fractures and found that the youngest age group (< 9 years) had the highest incidence of persistent diplopia (over 50%) and that the diplopia took twice as long to resolve compared to older children. Resolution of diplopia in children > 9 years took between 10 and 18 months, and they were more likely to have small, linear trapdoor defects of the anterior orbital floor with resultant entrapment.[10] The poor outcome in children may be attributed to the reportedly high rate of misdiagnosis and unrecognized orbital floor fractures with entrapment. Review of the literature concerning initial evaluation and management of pediatric orbital floor fractures in the emergency department has shown that white-eyed blowout fractures and presenting with oculocardiac reflex symptoms go unrecognized and uninvestigated in as many as one third of pediatric facial trauma patients because the symptoms are attributed to concussion.[11,12]

Consequently, ophthalmologic evaluation and orbital CT are delayed while the patients undergo observation for neurologic symptoms. This period of observation in children with entrapped orbital floor fractures increases the risk of muscle ischemia, permanent motility restriction, and diplopia. Conversely, some authors[13-15] has found no difference in final outcome between children and adults. Leitch and colleagues[13] found that pediatric patients who underwent blowout fracture repair did not have symptomatic postoperative diplopia compared to adults. Others have found that while children often have a longer period of recovery than adults, they have no difference in complications or long-term outcome.[14, 15] In fact, Criden and Ellis[16] found the recovery of extraocular motility to be prolonged in children despite an early surgical intervention.

Conclusions

Maxillofacial surgeon should always suspect the possibility of a white eyed blow out fracture in pediatric patients presenting with a significant ocular motility limitation and associated oculocardiac reflex symptoms. Our experience signifies that a proper diagnosis and earlier surgical intervention within 48 hours results in better postoperative outcomes. Further studies on a long term basis are required to evaluate the prognosis of ocular motility in patients underwent a delayed surgical intervention due to misdiagnosis.

References

1. Jordan DR, Allen LH, White J, Harvey J, Pashby R, Esmaeli B. Intervention within days for some orbital floor fractures: the white-eyed blowout. Ophthalmic Plast Reconstr Surg 1998; 14:379-90.
2. Tong L, Bauer RJ, Buchman SR. A current 10-year retrospective survey of 199 surgically treated orbital floor fractures in a nonurban tertiary care center. Plast Reconstr Surg 2001; 108:612-21.
3. Egbert JE, May K, Kersten RC, Kulwin DR. Pediatric orbital floor fractures: Direct extraocular muscle involvement. Ophthalmology 2000; 107:1875-9.
4. Baek SH, Lee EY. Clinical analysis of internal orbital fracture in children. Korean J Ophthalmol 2003; 17:44-9.
5. Keshini C. Parbhu, KoriAnne E. Galler, Chun Li, Louise A. Mawn. Underestimation of Soft Tissue Entrapment by Computed Tomography in Orbital Floor Fractures in the Pediatric Population Ophthalmology 2008; 115:1620–1625.
6. De Man K, Wijngaarde R, Hes J, de Jong PT. Influence of age on the management of blow-out fractures of the orbital floor. Int J Oral Maxillofac Surg 1991; 20:330-36.
7. Koltai PJ, Amjad I, Meyer D, Feustel PJ. Orbital fractures in children. Arch Otolaryngol Head Neck Surg 1995; 121:1375-9.
8. Chapman VM, Fenton LZ, Gao D, Strain JD. Facial fractures in children: Unique patterns of injury observed by computed tomography. J Comput Assist Tomogr 2009; 33:70-72.
9. De Man K, Wijngaarde R, Hes J, de Jong PT. Influence of age on the management of blow-out fractures of the orbital floor. Int J Oral Maxillofac Surg 1991; 20:330-36.
10. Cope MR, Moos KF, Speculand B. Does diplopia persist after blowout fractures of the orbital floor in children? Br J Oral Maxillofac Surg 1999; 37:46-51.
11. Lane K, Penne RB, Bilyk JR. Evaluation and management of pediatric orbital fractures in a primary care setting. Orbit 2007; 26:183-91.
12. Cobb A, Murthy R, Manisali M, et al. Oculovagal reflex in paediatric orbital floor fractures mimicking head injury. Emerg Med J 2009; 26:351-3.
13. Leitch RJ, Burke JP, Strachan IM. Orbital blowout fractures - the influence of age on surgical outcome. Acta Ophthalmol (Copenh) 1990; 68:118-24.
14. Kwon JH, Moon JH, Kwon MS, et al. The differences of blowout fracture of the inferior orbital wall between children and adults. Arch Otolaryngol Head Neck Surg 2005; 131:723-7.
15. Carroll SC, Ng SGJ. Outcomes of orbital blowout fracture surgery in children and adolescents. Br J Ophthalmol 2010; 94:736-9.
16. Criden MR, Ellis FJ. Linear nondisplaced orbital fractures with muscle entrapment. J AAPOS 2007; 11:142-7.

Internal derangements of the temporomandibular joint (TMJ) - a review and update

Twinkle S Prasad [a] , Philips Mathew [b]

Abstract

Internal derangements of the temporomandibular joint (TMJ) are described as disturbances in the normal anatomical relationship between the disc and the condyle. Because the temporomandibular joint shows large adaptive and compensatory mechanisms over abnormal disc motion, these disorders may be asymptomatic or minimally evident for a long time. In addition, the clinical signs and symptoms vary from patient to patient. A careful clinical evaluation, supplemented by imaging findings, should help differentiate asymptomatic derangements from painful conditions that may require treatment. When signs and symptoms are correctly interpreted, the application of appropriate therapeutic guidelines can lead to treatment success.

Address for correspondence:

[a] Associate professor,
[b] Assistant Professor
Dept of Oral Medicie and Radiology
Government Dental College
Kottayam

Introduction

The internal derangements are the most common intracapsular abnormalities of the TMJ, being observed even in asymptomatic subjects.[1] The term derangement refers to the abnormal positional and functional relationship between the articular disk and the mandibular condyle and articular surfaces of the temporal bone. Therefore, these alterations have been also referred to as disc derangements. Such derangements change the normal function of the TMJ and may be associated with joint sounds, pain, or limitation or deviation of the range of mandibular motion. Disk displacement of the temporomandibular joint (TMJ) is considered an important cause of facial and TMJ pain, clicking, crepitus, and dysfunction. They differ from degeneration because the quality and structure of the TMJ tissues are not necessarily altered.[2]

Aetiopathogenesis

Many aetiologic factors have been proposed to explain disc derangements. Traumatic events are the most common among them. Trauma may occur as single, sudden blow to the jaw (macrotrauma) or mild, frequent forces over a long period (microtrauma). Macrotrauma may cause stretching, tearing, or rupture of the disc, lateral ligament, or capsule. When bleeding occurs, fibrotic or hyperplastic intra-articular reactions may lead to restricted mobility and pain. Microtrauma is usually caused by malocclusion, altered occlusion (after orthodontic treatment), teeth loss, unilateral chewing habits and bruxism. This compressive overloading may cause soft-tissue responses and lead to permanent intra-articular changes, with long-term effects on the disc function. [3]

Changes in the composition of the synovial fluid may increase the intra-articular friction, leading to deranged disc motion. These biochemical changes may also affect the joint lubrication and nutritional requirements of the articular surfaces which will further aggravate the condition.

An improper activity of the lateral pterygoid muscle during TMJ motion has been also related to disc derangements. A higher propensity toward anterior disc displacement has also been found in subjects in whom the lateral pterygoid muscle activity is abnormal.[4]

Irrespective of the etiologic factors and concurrent predisposing factors, an initial adaptive response, triggered by overloading, induces structural changes in the TMJ. This is a slow but continuous process of modelling that involves all the components of the TMJ. Even though physiological changes occur in the disc, its

Stage	Clinical Findings	Radiologic Findings	Surgical Findings
I	Painless clicking No locking No restricted motion	Slight anterior disk displacement that reduces on opening Normal osseous contours	Normal disk form Slight anterior disk displacement
II	Occasional painful clicking Intermittent locking Headaches	Slight anterior disk displacement that reduces on opening Early disk deformity Normal osseous contours	Thickened disk Anterior disk displacement
III	Frequent pain Joint tenderness Headaches Locking Restricted motion	Anterior disk displacement that does not reduce on opening Moderate disk deformity Normal osseous contours	Disk deformed and displaced Variable adhesions No bone changes
IV	Chronic pain Headaches Restricted motion with crepitus	Anterior disk displacement that does not recapture on opening Marked disk deformity Degenerative osseous changes	Disk perforation, displacement, and adhesions Degenerative changes in condyle and/or fossa
V	Variable pain Joint crepitus	Anterior disk displacement that does not recapture on opening Marked disk deformity Degenerative osseous changes	Disk perforation, displacement, and adhesions Degenerative changes in condyle and/or fossa

Table -1 *Wilkes classification of internal derangement of TMJ*[11, 12]

ability to remodel is lower than that of other soft tissues of the TMJ, such as the capsule, capsular ligaments, and retrodiscal tissues. These adaptative changes can change the biomechanical behaviour of the articular disc. It will lead to loss of coordination of the disc-condyle complex; but the patient is usually asymptomatic in this stage. This is because; the altered mechanical loading is compensated by the structural modelling of the TMJ.[5]

When the adaptative and compensatory responses are exhausted, the changes taking place in the TMJ are known as regressive modelling (i.e., maladaptation). This stage is characterized by pain and other clinically evident signs and symptoms. With time, the clinical manifestations may become less obvious as the disorder enters a chronic phase.[4, 6]

Clinical presentation

Different criteria have been used to classify disc derangements. Normally, in closed mouth, position of disk in temporomandibular joint is in such a way that the posterior band of the disk will be directly superior to condyle. Displacement of the disc may be anterior, medial, lateral, or even in posterior direction. Multidirectional displacements are considered more frequently than unidirectional ones. Posterior derangements are rare. The oblique orientation of the lateral pterygoid muscle and the angulation of the condyle direct most meniscal displacements in an anterior-medial path. [7,8]

Clinically disc displacement can be classified into two groups' disc displacement with reduction and disc displacement without reduction.

In disk displacement with reduction, disk is displaced in closed mouth position but returns to normal on opening. This will lead to joint sounds during mouth opening and closure. In rest position and in centric occlusion, the posterior band of the disc is located behind the apex of the condylar head. Mouth opening occurs with a clicking or popping sound, because the posterior band of the disc slips back over the condylar head. As a result, in the open-mouth position the intermediate zone of the disc will be placed correctly between the condylar head and the eminence of the temporal bone. Because the opening movement relocates the disc in the joint, this stage is referred to as disc displacement with reduction. Occasionally, a second clicking sound is heard during mouth closure ("reciprocal click"), because the posterior band of the disc slips forward off the condyle. Usually normal range of jaw movements is present. But at times pain may limit the range of movements and it is not due to any structural dysfunction. Unilateral cases of anterior disk displacement with reduction will show deviation of mandible to affected side on early opening, then returning to the normal path and completes the opening movement. This deviation of mandible is due to due to temporary arrest in translation caused by displaced disk[8,9].

In disk displacement without reduction disk is

displaced in closing followed by failure to return to normal position on opening. The disc acts as an obstacle, preventing the condyle to overcome the posterior band when mouth opening is attempted. In this condition the joint appears as "locked." Clicking sounds are not heard. Mouth cannot be opened completely due to displaced disk blocking translatory movements. This stage is referred to as closed lock. Eccentric movements are relatively normal to the ipsilateral side but restricted to the contralateral side. Unilateral cases of disk displacement with out reduction will show deviation of mandible to affected side during mouth opening.

Molinari .F et al.[10] grouped anterior disc derangements into four categories based on the degree of dislocation, reversibility during the opening-closing movement, and changes in disc shape. They have also evaluated the MRI finding corresponding to each category. Stage 1 or the early stage in which no joint noise or dysfunction is evident. However, at mouth opening, the patients may feel a slight catching sensation. This may be the earliest sign that a change in the frictional properties of the joint has occurred. Therefore, this stage of derangement has been referred to as TMJ *disc incoordination*. The second stage corresponds to disc displacement with reduction whereas the third stage corresponds to disc displacement without reduction or closed lock. The fourth category is also characterized by a limitation of mouth opening. However this limitation may not be caused by disc displacement. The disc may be in a normal position but advanced degenerative changes have occurred. Adherences are usually found with the disc and the articular eminence, so that only condylar rotation is allowed. On the other hand, adherences also limit the mobility of the disc (i.e., "stuck," "fixed," or "frozen disc").

Similarly Wilkes has classified internal derangement into five stages based on the clinical severity and radiological findings. (Table-1)

Differential diagnosis

Disc derangements may have clinical manifestations similar to those of other disorders of the facial region. Pain and jaw dysfunction can be related to toothache, pericoronitis, maxillary sinusitis, earache, salivary gland pathosis, temporal arteritis, neuralgias, and tension-type headache. All these conditions should be excluded when assessing patients with clinical suspicion of disc derangements. In addition, two other temporomandibular disorders (TMDs) must be considered in the differential diagnosis of symptomatic disc derangements: myofascial pain and dysfunction (MPD), and painful inflammatory or degenerative conditions of the TMJ. MPD differs from primary TMJ disorders, because in the former pain originates from the masticatory muscles. Myofascial pain is the most common TMD. It is characterized by a dull ache in the TMJ region that increases during function, with other possible ancillary findings (ie, tension-type headache, earache, or toothache; a sensation of muscle stiffness; reduced motion of the mandible). In the same group (MPD) are included regional problems of the muscles, such as myositis, myospasm, local myalgia, myofibrotic contracture, as well as the centrally mediated chronic muscle pain. Systemic disorders, such as fibromyalgia, may also have considerable overlap in clinical features with MPD.

The diagnosis of painful conditions of the temporomandibular region requires a careful evaluation of the history of the patient (dental, medical, and psycho-social data) and a detailed examination of signs and symptoms. Clinical assessment should be always performed before imaging (Fig. 9A to F). If the most important symptom reported by the patient is pain, its characteristics should be assessed as part of a routine diagnostic protocol. When the location, intensity, quality, duration, modifiers, chronicity of pain, and associated symptoms suggest a potential masticatory muscle disorder, a panoramic radiograph should be first obtained to exclude possible dental, periodontal, or other problems of the oral region. If the patient's history and clinical findings suggest an intracapsular joint problem, the assessment of the TMJ should be performed using MRI. Such an imaging tool can be used concurrently to exclude some causes of muscular problems (ie, focal myositis, abscess, muscle atrophy, etc.) and local diseases of the oral region, providing helpful information in the differential diagnosis of TMDs.

Imaging findings

With the development of newer modalities such as arthrography, computed tomography, magnetic resonance imaging (MRI), and, most recently, ultrasonography (US), the understanding of the anatomy and the diagnosis of internal derangements of the TMJ has been improved.

Magnetic resonance imaging (MRI) is highly accurate in diagnosing displacement of the articular disk.1 X-ray computed tomography (CT) can also depict anterior displacement of the articular disk. On axial CT, anterior disk displacement was defined as the presence of an area of soft tissue density that was semilunar in shape, 0.5 cm or more in anteroposterior diameter, located in front of the mandibular condyle, and seen contiguously

on more than 2 images. The density of the soft tissue was isodense to or slightly more hyperdense than adjacent muscles. If the soft tissue density was not seen anterior to the mandibular condyle, the disk was presumed to be in a normal superior position.

For the diagnosis of DD, magnetic resonance imaging (MRI) is often considered the 'gold standard'. The use of the so called '12 o'clock' criterion (a disc position in which its posterior band lies superior to the condyle) for normal disc position may be a reason for this overdiagnosis (15). Alternatively, opto-electronic movement recordings have gained more attention lately. This technique may be superior to MRI because it yields dynamic information on the joint. The use of such recordings for the assessment of IDs is currently being tested by our group. However, in daily practice, the use of MRI and that of opto-electronic movement recordings is limited.

Therapeutic Outlines

Although genetic, biochemical, and histological aspects of the TMDs have been studied, the etiology of these disorders remains largely unknown. Therefore, therapy is largely dependent on the initial clinical assessment of the patient. When signs and symptoms are correctly interpreted, the application of research-based therapeutic guidelines can lead to treatment success. According to literature, a positive out come can be achieved by therapy in 75 to 90% of patients. A wide consensus has been reached through the years on considering conservative and reversible approaches as first-line therapy of symptomatic disc derangement. Included in this group are various medications, such as nonsteroidal anti-inflammatory drugs and muscle relaxant, oral appliances, home care procedures, and cognitive-behavioral information program.

Temporomandibular joint (TMJ) disc displacement without reduction (DDw/oR) is commonly managed by nonsurgical approaches such as physical medicine (eg, physical therapy, home exercises, intraoral appliances), behavioral medicine (eg, counseling, biofeedback, stress management, and relaxation training), and pharmacologic therapy (eg, nonsteroidal anti-inflammatory drugs [NSAIDs], steroidal and antispasmodic medications). Many studies report that the success rate of nonsurgical treatment is approximately 60%, whereas other studies have reported approximately 40% to 70% self-improvement without any treatments. In the past, joints that did not respond to nonsurgical treatment were generally treated with surgical intervention to either change the morphology or the position of the disc or to remove the disc in total. However, these procedures were associated with surgical risks and long-term sequelae. Currently, minimally invasive treatments such as arthrocentesis as well as arthroscopic lysis and lavage are often used as a first-line surgical treatment or in conjunction with nonsurgical modalities, as they have shown to be reversible procedures with low morbidity and high efficacy.

When these approaches fail to produce clinical improvements on painful dysfunctional conditions of the TMJ, surgical procedures (ie, arthrocentesis, arthroscopic surgery, discoplasty, and discectomy) may be indicated. Several case-based studies have shown that surgical management may be effective. However, to increase the chances of treatment success in patients not responsive to conservative therapy, the least invasive procedure that can be effective should be first proposed. Oral appliances (Fig. 9G and H) are used in patients with symptomatic disc derangement for stabilizing or repositioning the occlusal maxillomandibular relationship. The stabilizing approach is reversible. It produces changes in the occlusal behavior of the patient, by inducing consciousness of any oral parafunction. Therefore, it reduces the chance of further wear, chipping, or cracking of the teeth. In unstable occlusion caused by absence of multiple posterior contacts bilaterally, stabilization restores the automatism of normal occlusion. It is also indicated in patients suffering from bruxism and for managing symptoms associated with TMJ disc derangement. The mandibular repositioning appliance has deeper effects on the position of maximum intercuspation and may cause a permanent occlusal change. The theory behind the use of this appliance is to shift the mandibular condyles in a more forward position and, therefore, to allow for recapturing of a displaced disc. This appliance is often used in patients with painful clicking or intermittent locking. However, the results of this strategy may not be as good as expected. Long-term case report studies have suggested that TMJ clicking may recur over time and, even without audible joint sounds, disc position can remain altered.

A number of noninvasive and reversible therapies are widely used and appear to help many patients. Optimally, these therapies should have low morbidity and minimal alteration of underlying anatomic structures. These therapies include:

• **Supportive patient education.** Initial attention should be given to the issue of patient education on what is known about TMD and the fact that most of these problems follow a benign course. Many experts

recommend that patients undergo education directed at eliminating certain behaviors perceived to be harmful, such as clenching and grinding. Some experts recommend exercise and stress management. Rest and dietary modifications may help some patients.

• **Pharmacologic pain control.** Medication may be useful for initial symptom management. The medications useful for TMD are similar to those useful for other painful musculoskeletal conditions. Nonsteroidal anti-inflammatory drugs (NSAIDs) and opiates are the mainstay of pharmacological pain treatment. Some clinicians also have found muscle relaxant medications and low-dose antidepressants of a sedating type to be useful in initial management of TMD. Other medications also have been used for specific indications. In all cases, the clinician must weigh the risk of side effects against potential benefits, along with his or her own professional competence in the administration and management of such medications.

• **Physical therapy.** Physical therapy applications to TMD include a wide variety of evaluative techniques and treatment modalities that have been commonly used in other neurological and musculoskeletal disorders. These therapies generally are conservative and noninvasive. Benefits to TMD patients have been described, although few data are available to document these results.

• **Intraoral appliances.** Stabilization splints are considered noninvasive and reversible and are recommended by many experts for early treatment of these patients. It is important that these appliances are of a type that does not lead to major alteration of the patient's occlusion. Repositioning appliances may appear to be noninvasive but have potential for creating such irreversible changes in occlusion and, consequently, the possibility of precipitating other problems.

• **Occlusal therapy.** Much controversy surrounds the use of occlusal therapy. The advocates argue that occlusal abnormalities and/or joint manipulation precipitate the development of TMD. Occlusal therapies are aimed at modification of the occlusion itself through alteration of the tooth structure or jaw position. Given that this latter therapy is irreversible, and given that the superiority of this treatment over reversible therapies has not been demonstrated in randomized controlled prospective trials, this form of occlusal adjustment probably will not represent best practice for initial management of TMD. However, assessment of occlusion is necessary as part of the initial oral examination to identify and eliminate gross occlusal discrepancies such as those that may inadvertently occur as a result of restorative procedures. After these initial therapeutic interventions, a small number of patients may continue to exhibit symptoms associated with the TMD constellation of conditions. These patients will require consideration for longer term and/or more invasive therapies.

The following surgical procedures are accepted and effective methods for treatment of joints with ID:

1. Arthrocentesis
2. Arthroscopy
3. Condylotomy (indirect arthroplasty)
4. Arthrotomy

Alloplastic implants are not generally indicated for initial surgical treatment of joints with ID/OA. Prosthetic joint replacement may be indicated in selected patients with severe joint degeneration, destruction, or ankylosis. These devices should be considered for use only when their safety and efficacy has been recognized by the FDA.

5. Other Procedures

a. Coronoidotomy/coronoidectomy

b. Styloidectomy (Eagle's Syndrome)

c. Procedures for Recurrent Dislocation

References

1. Hall HD: Intra-articular disc displacement Part II: Its significant rol in temporomandibular joint pathology. J Oral Maxillofac Surg 53:1073, 1995
2. de Bont LGM, Stegenga B: Pathology of temporomandibular joint internal derangement and osteoarthrosis. Int J Oral Maxillofac Surg 22: 71, 1993
3. Paesani D, Salas E, Martinez A, Isberg A: Prevalence of temporomandibular joint disk displacement in infants and young children. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 87:15,1999
4. Kircos LT, Ortendahl DA, Mark AS, Arakawa M: Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofac Surg 45:852, 1987
5. Paesani D, Westesson P-L, Hatala M, Tallents RH, Kurita K: Prevalence of temporomandibular joint internal derangement in patients with craniomandibular disorders. Am J Orthod Dentobac Orthop 101:4, 1992
6. Katzberg RW, Westesson P-L, Tallents RH, Drake CM: Orthodontics and temporomandibular joint internal derangement. Am J Orthod Dentofacial Orthop 109:515, 1996
7. Tasaki MM, Westesson P-L, Isberg,AM, Yan-Fang R, Tallents RH: Classification and prevalence of temporomandibular joint disk displacement in patients and symptom-free volunteers. Am J Orthod Dentofac Orthop 109:249, 1996
8. Larheim TA, Westesson P-L,Sano T: Temporomandibular Joint Disk Displacement: Comparison in Asymptomatic Volunteers and Patients. Radiology 218:428, 2001
9. Ribeiro RF, Tallents RH, Katzberg RW, Murphy WC, Moss ME, Magalhaes AC et al: The prevalence of disc displacement in

symptomatic and asymptomatic volunteers aged 6 to 25 years. J Orofacial Pain: 11:37, 1997

10. F. Molinari, P.F. Manicone, L. Raffaelli, R. Raffaelli, T. Pirronti, L. BonomoTemporomandibular joint soft-tissue pathology, I: disc abnormalities Semin Ultrasound CT MRI, 28 (2007), pp. 192–204
11. Wilkes CH: Internal derangements of the temporomandibular joint: Pathological variation. Arch Otolaryngol Head Neck Surg 115:469, 1989
12. Wilkes CH: Surgical treatment of internal derangements of temporomandibular joint. Arch Otolarngol Head Neck Surg 117:64, 1991
13. Hall HD, Navarro EZ, Gibbs SJ: One-and three-year prospective outcome study of modified condylotomy for treatment of reducing disk displacement. J Oral Maxillo Surg. 58:7, 2000
14. Hall HD, Navarro EZ, Gibbs SJ: Prospective study of modified condylotomy treatment of nonreducing disk displacement. Oral Surg Oral Med Oral Oral Radiol Endod 89:147, 2000
15. Nickerson JW, Boering G: Natural course of osteoarthrosis as it relates to internal derangement of the temporomandibular joint. Oral Maxillofac Surg Clin North Am 1:27, 1989
16. Werther JR, Hall HD, Gibbs SJ: Type of disc displacement and change in disc position after modified condylotomy in 80 symptomatic T-M joints. Oral Surg Oral Med Oral Pathol 79:668, 1995
17. Weinberg S, LaPoint H: Cervical extension-flexion injury (whiplash) and internal derangement of the temporomandibular joint. J Oral Maxillofac Surg 45:653, 1987
18. Kai S, Kai H, Tabata O, et al: Long-term outcomes of nonsurgical treatment in nonreducing anteriorly displaced disk of the temporomandibular joint. Oral Surg Oral Med Oral Pathol 85:258, 1998
19. Boering G: Temporomandibular joint osteoarthrosis (1966 Doctoral thesis, University of Gronigan). Gronigen, the Netherlands, Drukkerij, van Denderen BV, 1994 (English-language version)
20. Link JJ, Nickerson JW Jr: TMJ internal derangements in an orthognathic surgery population. Int J Adult Orthodon Orthognath Surg 7:161, 1992
21. Schellhas KP, Pollei SR, Wilkes CH: Pediatric internal derangements of the temporomandibular joint: Effect on facial development. Am J Orthod Dentofac Orthop 104:51, 1993
22. Rasmussen OC: Temporomandibular arthropathy: Clinical, radiologic, and therapeutic aspects, with emphasis on diagnosis. Int J Oral Surg 12:365,
23. Lundh H, Westesson P-L, Eriksson L, Brooks SL: Temporomandibular joint disc displacement without reduction. Treatment with flat occlusal splint versus no treatment. Oral Surg Oral Med Oral Pathol 73:655, 1992
24. Sato S, Kawamura H, Nagasaka H, Motegi K: The natural course of anterior disc displacement without reduction in the temporomandibular joint: Followup at 6, 12, and 18 months. J Oral Maxillofac Surg 55:234, 1997
25. Kurita K, Westesson P-L, Yuasa H, et al: Natural course of untreated symptomatic temporomandibular joint disc displacement without reduction. J Dent Res 77:361, 1998
26. Sato S, Takahashi K, Kawamura H, et al: The natural course of nonreducing disk displacement of the temporomandibular joint: Changes in condylar mobility and radiographic alterations at one-year follow-up. Int J Oral Maxillofac Surg 27:173, 1998
27. Sato S, Sakamoto M, Kawamura H, Motegi K: Long-term changes in clinical signs and symptoms and disc position and morphology in patients with nonreducing disc displacement in the temporomandibular joint. J Oral Maxillofac Surg 57:23, 1999
28. Brand J, et al: The effects of temporomandibular joint internal derangement and degenerative joint disease on tomographic and arthrotomographic images. Oral Surg Oral Med Oral Path 67: 220, 1989
29. Harms SE, Wilk RM, Wolford LM, et al: The temporomandibular Joint: magnetic resonance imaging using surface coils. Radiology 157: 133, 1985
30. Katzberg RW, Bessette RW, Tallents RH et al: Normal and abnormal temporomandibular joint: MR imaging with surface coil. Radiology 158: 183, 1986
31. Wilkes CH: Arthrography of the temporomandibular joint in patients with the TMJ pain-dysfunction syndrome. Minn Med 61:645, 1978
32. Katzberg RW, Dolwick MF, Helms CA et al: Arthrotomography of the temporomandibular joint. AJR 134: 995, 1980
32. Katzberg RW: Temporomandibular joint imaging. Radiology 170: 297. 1989

Adieu to Cavities - Recent Concepts in Caries Management

Rita Zarina A [a], Ektah Khosla [b], Padmakumari B [c]

Abstract

Dental caries is the most prevalent chronic disease affecting human race. In many ways it can be considered a disease of modern times as the occurrence of caries seems to be much higher in the last few generations. Huge amounts of money and time is spent in treating caries. Hence the prevention and control of dental caries is the main aim of public health work, eventually the ultimate objective is to eliminate the disease itself.

Address for correspondence:

[a] Professor, Dept of Pedodontics, Govt Dental College, Kottayam; [b] Professor, Mar Baselios Dental College, Kothamangalam; [c] Asso. Professor, Govt Dental College, Trivandrum

Introduction

Dental caries has a huge prevalence in India and affects appropriately 50 to 60 per cent of population. It is a multi-factorial disease requiring the presence of a susceptible host, cariogenic microflora and a diet conducive to enamel demineralisation. (Fitzgerald and Keyes model) These three factors together with time factor are responsible for disease initiation and progression.[1] Removal of any one element ostensibly leads to interception of the disease process. Dental caries is a site specific disease that undergoes many cycles of demineralization and remineralization during the lesion development. Because of its development characteristic dynamics, the caries can be arrested or repaired at its early stages without operative intervention.

Traditional thinking about caries management was not diagnosis but about restoring caries. Current management of dental caries emphasise on prevention and treat it as a dynamic and reversible process.

Approaches to Caries Managements

Oral health care devices - They play a critical role in mechanical removal of plaque and thereby help in achieving optimum oral health. Role of tooth brushes, interdental devices and dental floss are considered as gold standards of oral health care. Recommendation of proper night brushing is considered to be most important for reduction of dental caries. Studies have demonstrated that the efficacy of tooth brush is reduced with usage over a period of time. Thus, tooth brush should be changed at an interval of three months or earlier if needed as per ADA guide lines.[2]

Fluoride programme – Fluorides play a vital role in prevention of dental caries. Both systemic and topical fluorides showed to have better anticaries properties. Water fluoridation, milk and salt fluoridation, fluoride incorporated in tooth paste, mouth washes, and dental materials are common delivery methods. Slow release fluoride devices like co-polymer membrane and slowly dissolving fluoride glass beads are also effective tool against caries.[3] The glass beads are attached to the buccal aspect of molar teeth using acid etch composites.

Probiotics - They are live microorganisms which when administered in adequate amounts confer a health benefit on the host. They selectively remove only the pathogen while leaving the reminder of the oral echo system intact. To establish a cariostatic effect probiotic should adhere to dental tissues and this should be part of the biofilm. For this installation of probiotic in the oral environment seems to be important. Ideal vehicles for probiotic installation are yogurt, milk and cheese.[4]

Sugar substitutes - The use of sugar substitutes like xylitol, manitol and sucralase are advocated for caries preventive programmes. Studies showed that xylitol gum chewing and fluoride usage resulted in a significantly lower incidence of caries than fluoride alone.[5] Xylitol is

not fermented by cariogenic plaque bacteria and thus, does not lower plaque pH. As the plaque pH does not decrease, enamel demineralization is prevented and plaque bacterium does not proliferate. Xylitol reduces the accumulation of plaque and it has specific inhibiting effect on the growth of S. Mutans in the mouth.

Action of Ozone – Progression of caries lesion occurs when conditions are suitable for acidogenic bacteria to release acids as a by-product. This acid produced may lead to breakdown of mineralized tooth structure. Ozone has severely disrupting effect on the bacterial population, as it has an oxidative effect on acid pyruvate, one of the strongest naturally occurring acids manufactured by bacteria. It acts as a bactericide by disrupting the integrity of the bacterial cell wall. Studies proved that ozone treatment for ten seconds significantly remineralized lesions and could be used as an alternative option for pit and fissure lesions.[6]

Caries vaccine - The concept of vaccine can be visualized primarily with the recognition of S.mutans as a key microorganism in the development of caries and the development of a method of immunization specifically targeted at neutralizing S.mutans that are generated. It may be related with IgG and systemic vaccination using a cell wall constituent of S.mutans or with the oral route of vaccination and stimulation of IgA. Active immunization, passive immunization and transgenic plants are developed for vaccine administration.[7]

Lasers - Lasers can be used to alter the tooth surface of enamel and make it less prone to caries. Nd:YAG lasers can be used for inhibiting incipient early caries lesions.[8] Lasers can eliminate the fear factor associated with the child patients as they can be used for caries prevention, pain less caries removal and even cavity preparation.

Altering surface morphology/Increasing tooth resistance - Surface active polymeric agents for surface adhesive binding has been developed comprising of application in two stages for increasing tooth resistance. It involves initial application of an acidified calcium phosphate solution followed by suitable fluoride solution. eg-: chewing gum of ACP followed by use of fluoride tooth paste.[9]

Chemotherapeutic plaque control agents - The role of plaque in caries formation is well documented. Many chemical agents are formulated for controlling plaque formation. Most commonly used chlorhexidine has got antibacterial effects that include binding well to bacterial cell membrane, increasing their permeability, initiating its leakage and precipitating intracellular components. Plaque modifying agents like urea peroxidase and enzyme systems intended to alter plaque architecture and adherence are also developed. Delmopinol which affect by binding to salivary proteins and altering the cohesiveness and adhesiveness properties of the film have also been formulated. [10]

New approaches

Studies found that new methods of genetic profiling may distinguish bacterial populations that cause severe dental decay in children and be used as a basis for intervention and prevention. It is demonstrated that PCR based 16srRNA gene DGGE (denaturing gradient gel electrophoresis) is a valuable tool for differentiating the microbial composition of oral plaque in children with caries from caries free child.[11]

A newly developed peptide, embedded in a soft gel or a thin, flexible film (MSH OR melanocyte stimulating hormone) when placed next to a cavity, encourage cells inside the teeth to regenerate.[12] A potent purified fibrinolytic enzyme- producing bacterium was isolated from the traditional Korean condiment Chungkookjang and identified as Bacillius vallismortis Ace02, has been used for caries prevention.[13] Replacement therapy - S.mutan strain BCS3-LI is a genetically modified effector strain designed for use in replacement therapy to prevent dental caries.[14] The gum of Cratoxylum formosum, commonly known as mempat, is extensively used for caries prevention. This has got antimicrobial activity against different strains of S.mutans.[15]

Conclusion

There are practically no geographic areas in the world whose inhabitants do not exhibit some evidence of dental caries. Though it is not life threatening, the sequelae associated with it are far reaching. Therefore it is mandatory to understand the magnitude of this problem and risk factors in the community, to plan suitable preventive measures and management strategies in the early stages, and implement them timely to ensure that a better health care service is provided.

References

1. Fitzgerald RJ,Jordan HV,Archard HL:Dental caries in gnotobiotic rats infected with a variety of Lactobacillus acidophilus,Arch Oral Bio11:473-476,1966.
2. Park KK,Martis BA,Christen AG:Choosing an effective tooth brush,Clin Prev Dent7(4):5-10,1985.
3. Bonner BC,Clarkson JE,Dobbyn L,Khanna SS,Slow release fluoride devices for control of dental decay.Cocharne Data base of systematic reviews 2006,Issu4.Art.No:CD005101

4. Anderson MH,Shi W.A Probiotic approach to caries management.Pediatr Dent2006;28:151-153
5. Isokangas Pet al:Xylitol chewing gum in caries prevention:a field study in children,J Am Dent Asso117:315-320,1988
6. Brazzelli M et al:Systamatic review of the effectiveness and cost effectiveness of Heal ozon for the treatment of ocllusal pit and fissure caries and root caries. Health Technol Assess 2006 May;10(16)-80
7. Mattos Graner RO,Smith DJ:The vaccination approach to control infections leading to dental caries.Braz J Oral Sci 2004,608
8. Glenn Van AS ,Dent Clin N America 2004;48:1011-1059
9. Featherstone JDB.Caries prevention and reversal based on the caries balance.Pediatr Dent 2006;28:128-132.
10. Mandel ID:Chemotherapeutic agents for controlling plaque and gingivitis,J Clin Periodontol15:488-498,1988.
11. Y.Li,Y.Ge,D.Saxena,PW Caufield.2006.Genitic profiling of the oral microbiota associated with severe early childhood caries.Journal of Clinical microbiology,45.1:81-87
12. http://news.discovery.com/tech/tooth-regeneration-gel.html
13. Kim JB,Jung WH,Lee YJ,Junk JK,Jang HW,Kim SW.Identification of a fibrinolytic enzyme by Bacillus Vallismortis and its potential as a bacteriolytic enzyme against Streptococcus mutans.Biotechnol Lett.2007Apr;29(4):605-10
14. Anusavice KJ,Present and future approaches for the control of caries.J Dent Educ2005;69(5):538-54
15. Suddhasthira T, Thaweboon S, Dendoung N, Thaweboon B, Dechkunakorns. Antimicrobial Activity of Cratoxylum Formosum on S.Mutans. Southeast Asian J Trop Med Public Health 2006 November; 37 (6): 1156 – 9.

Horizontally impacted bilateral mandibular molars with supernumarary teeth –a rare entity: Case report

Shiney Dominic [a], N Jayakumar [b], George Varghese [c], Muhammad Ali T [d]

Abstract

The impaction of permanent tooth is not uncommon, but bilateral horizontal impaction of permanent tooth along with supernumerary tooth is a rare entity. Here we report a rare case of accidentally discovered bilateral impacted third molar which were surgically removed

Keywords: Impaction, Third molar, Supernumerary, Bilateral

Address for correspondence:

[a] Associate Professor, [b] Associate Professor, [c] Principal & Professor, [d] Junior Resident, Department of Oral & Maxillofacial Surgery, Government Dental College Kottayam

Introduction

Impacted teeth are those which fail to erupt in normal occlusion within chronologic age[1]. Any teeth may get impacted but the most common are mandibular third molar[2]. The causes for impaction can be adjacent tooth, dense overlying bone, soft tissue, and lack of space in the jaw, aberrant path of eruption or abnormal positioning of tooth bud[3,4]. Generally third molars erupt between the age of 17 and 21 years[5]. The average age for eruption of mandibular third molar in male is three to six month ahead of female and most authors claim that the incidence of mandibular third molar impaction is higher in females[6]. Most adults have four wisdom teeth, but it is possible to have fewer or more, in which case the extras are called supernumerary teeth.[7]

Supernumerary teeth are defined as those in excess when compared to the normal series[8]. Reported prevalence is 0.3 to 0.8% in primary dentition and 0.1 to 3.8% in permanent dention[9]. Supernumerary teeth can occur as single, multiple, unilateral, bilateral in maxilla, mandible or both[10]. Both genetic and environmental factors have been considered as aetiology for supernumerary teeth. Several theories like Atavism, Dichotomy theory and Dental lamina hyperactivity theory are proposed as aetiology. Yosef et Al reported that 6.9% of non syndromic multiple supernumerary teeth occur in mandible and 44% in mandibular premolar region[11].

Damage to the inferior alveolar nerve (IAN) during third molar extraction is a major concern for patients and clinicians.[12] The incidence of IAN injury reported in the literature ranges from 1.3% to 5.3%.[13]. The risk of this complication depends mainly on the position of the impacted tooth in relation to the inferior alveolar canal.[14] To reduce this risk, several approaches have been proposed such as orthodontic- assisted extraction of the impacted mandibular third molars.[15,16] or partial odontectomy that is, the surgical removal of the anatomic crown leaving the roots in place.[17,18]. This case report describes surgical extraction of horizontally impacted bilateral third molars with bilateral supernumerary tooth in close contact with the IAN.

Case report

A 23 year old male patient reported with complaints of pain on right side of lower jaw since 3 weeks. Pain was dull and intermittent in nature. There was no relevant medical history associated. Intraoral examination revealed slight swelling in right mandibular 3rd molar region with clinical absence of both lower third molars. A dental panoramic radiograph was taken. Interestingly x- ray revealed the presence of bilateral horizontally impacted mandibular molar with bilateral supernumerary teeth above it (Fig.1). Impacted teeth were in close proximity to IAN.

The patient was informed about the condition and treatment plan was formulated to remove the impacted

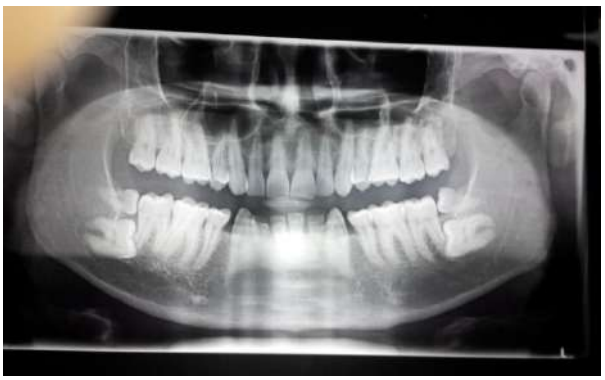


Fig 1: Showing bilateral horizontally impacted mandibular molar with bilateral supernumerary teeth

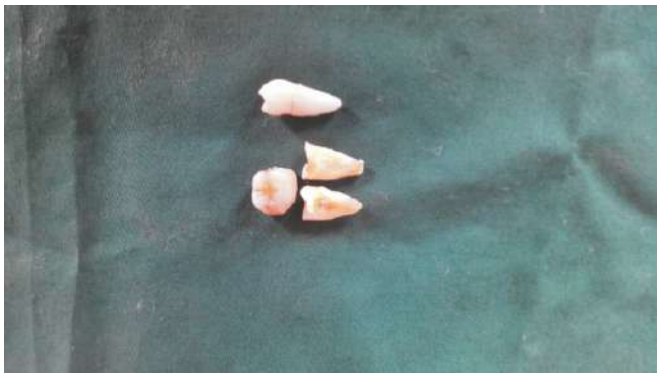


Fig 2: Showing sectioned impacted tooth and the supernumerary tooth in one piece

teeth surgically under local anaesthesia. The risk versus the benefits of surgical removal of deeply seated third molar was explained to the patient. After obtaining a written informed consent, surgical removal of supernumerary and impacted tooth was done. The two impacted molars were removed in two sittings. The first tooth surgically extracted was the right mandibular third molar, with the patient under local anaesthesia (lignocaine with epinephrine, 1:100,000). Full-thickness flap was designed and raised to expose the impacted tooth. Ostectomy was carried out using a fissure bur. The supernumerary tooth was removed in one piece. With regard to third molar; the anatomic crown was partially sectioned and removed. Then root was sectioned and removed separately (Fig.2). Surgical removal of supernumerary and impacted teeth on the left side was carried out after a period of three months. Both the procedures were done by the same surgeon.

Patient was followed up at intervals of 1 week, 1 months, 3 months and 6 months. Minimal discomfort was reported with mild inferior alveolar nerve parasthesia during the initial follow up period. This gradually subsided subsequently.

Discussion

The incidence of third molar impaction is increasing in the present generation due to insufficient space in the upper and lower arch. This results in pain and so need arises for their extraction surgically.

Only a few cases of bilateral impacted third molars have been reported in literature.[19] the present one is a rare case report of bilateral impacted teeth with bilateral supernumerary teeth. The incidence of wisdom tooth removal was estimated to be 4 per 1000 person[20]. Interestingly, the highest proportion of asymptomatic third molars was seen to have a horizontal angulation[21]. If there is an adequate depth and distance from adjacent second molars, these horizontally angulated impacted teeth may remain disease free[21]. Neurologic disturbance of the IAN after surgical removal of an impacted third molar is a serious complication. Although the incidence of such a complication is relatively low, its frequency increases when the roots of the impacted tooth are located near the nerve bundle[22]. The presence of a supernumerary tooth should alert the clinician to the possibility of the development of pathologies like cyst. The presence of a supernumerary tooth is also one of common cause for failure of eruption of teeth. Delayed eruption of associated teeth has been reported to occur in 28"60% of caucasians with supernumerary teeth. Once a clear indication for extraction has been made, the clinician should devise a strategy to reduce the risk of this complication. The frequency and severity of untoward events may be related to the procedure, patient, and/or surgeon. A meticulous planning and a proper surgical technique will reduce the incidence of IAN injury even in deep seated third molar impactions.

Conclusion

Impaction of mandibular third molars was most commonly seen in patients in their third decade of life. The most common pattern of impaction was mesioangular, with position A depth and class II ramus relation. The overall frequency of cysts and tumours associated with impacted mandibular third molars was relatively low. Therefore, patients having third molars with these unfavourable angulations, patterns and positions could be considered the candidates for prophylactic removal of impacted mandibular third molars. Moreover, early diagnosis of associated pathologies and proper management of impacted third molar is necessary to prevent further consequences.

Reference

1. Wisdom Teeth". American Association of Oral and Maxillofacial Surgeons. Retrieved 2010-09-28. "This generally occurs between the ages of 17 and 25"
2. Nasir A et al assessment of different pattern of impacted mandibular third molars and their associated pathologies
3. Ishihara Y, Kamioka H, Yamamoto TT, Yamashiro T. Patient with nonsyndromic bilateral and multiple impacted teeth and dentigerous cysts. Am J Orthod Dentofacial Orthop 2012; 141: 228-41.
4. Sheikh MA, Riaz M, Shafiq S.Incidence of distal caries in mandibular second molars due to impacted third molars – A clinical & radiographic study. Pakistan Oral & Dent J 2012; 32: 364-70.
5. Hupp JR, Ellis III E, Tucker MR. Contemporary Oral and Maxillofacial Surgery Principles of management of impacted teeth. 6th Ed. Philadelphia: Elsevier Publishers; 2014: 143-67.
6. Juodzbalys G, Daugela P. Mandibular third molar impaction: Review of literature and a proposal of a classification. J Oral Maxillofac Res 2013; 4: 1-12.
7. Marciani RD (2012). "Is there pathology associated with asymptomatic third molars (review)". J Oral Maxillofac Surg 70 (Suppl 1): 15–19. doi:10.1016/j.joms.2012.04.025
8. Ashish sha .Diagnosis and Management of Supernumerary Teeth.
9. Stafne, EC. Supernumerary teeth. *Dent Cosmos* 1932; 74: 653"659.
10. Rajab LD, Hamdan MAM. Supernumerary teeth: review of the literature and a survey of 152 cases. *Int J Paediatr Dent* 2002; 12: 244"254.
11. Yusof WZ. Non-syndrome multiple supernumerary teeth: literature review. *J Can Dent Assoc* 1990; 56: 147"149.
12. *Luca Landi, Stefano Piccinelli* , *Alessandro Raia*.Staged Removal of Horizontally Impacted Third Molars to Reduce Risk of Inferior Alveolar Nerve Injury
13. Bataineh AB: Sensory nerve impairment following mandibular third molar surgery. J Oral Maxillofac Surg 59:1012, 2001
14. Bui CH, Seldin EB, Dodson DB: Types, frequencies and risk factors for complications after third molar extraction. J Oral Maxillofac Surg 61:1379, 2003
15. Checchi L, Bonetti GA, Pelliccioni GA: Removing of high-risk impacted mandibular third molar. A surgical orthodontic approach. J Am Dent Assoc 127:1214, 1996
16. Bonetti GA, Bendandi M, Laino L, et al: Orthodontic extraction: Riskless extraction of impacted lower third molars close to the mandibular canal. J Oral Maxillofac Surg 65:2580, 2007
17. Phillips C, Norman J, Jaskolka M, et al: Changes over time in position and periodontal probing status of retained third molars. J Oral Maxillofac Surg 65:2011, 2007
18. O'Riordan BC: Coronectomy (intentional partial odontectomy of lower third molars). Oral Surg Oral Med Oral Pathol Oral Radiol Endod 98:274, 2004
19. Juodzbalys G, Daugela P (Apr-Jun 2013). "Mandibular Third Molar Impaction: Review of Literature and a Proposal of a Classification MaxillofacRes 4 (2): e1. doi:10.5037/jomr.2013.420
20. Juodzbalys G, Daugela P (Apr-Jun 2013). "Mandibular Third Molar Impaction: Review of Literature and a Proposal of a Classification (review)". J Oral Maxillofac Res 4 (2): e1. doi:10.5037/jomr.2013.4201.
21. Krishnan B, El Sheikh MH, El-Gehani R, Orafi H. Indications for removal of impacted mandibular third molars: a single institutional experience in Libya. J Maxillofac Oral Surg 2009; 8: 246-8.
22. Renton T, Hankins M, Sproate C, et al: A randomized controlled clinical trial to compare the incidence of injury to the inferioralveolar nerve as a result of coronectomy and removal of mandibular third molar. Br J Oral Maxillofac Surg 43:7, 2005.

Dermatoglyphics - an indicator of oral disease

Rita Zarina [a], Ektah Khosla [b], Digesh B [c], Deepak K [d], Aby Mathew [e]

Abstract

Dermatoglyphic analysis is now beginning to prove itself as an extremely useful tool for preliminary investigations into conditions with a suspected genetic basis. Significant investigations have been carried out into the dermatoglyphic indicators of congenital heart disease, leukemia, cancer, celiac disease, intestinal disorders, rubella, schizophrenia as well as other forms of mental illness. This article throws some light on dermatoglyphics as an indicator of oral disease as the study of dermatoglyphics is considered as a window of congenital abnormalities and is a sensitive indicator of intrauterine anomalies both dental as well as systemic.

Address for correspondence:

[a] Professor, Dept of Pedodontics, Govt Dental College, Kottayam; [b] Professor, Mar Baselios Dental College, Kothamangalam; [c] Asst. Professor, [d & e] Senior Lecturer, Dept of Pedodontics, Govt Dental College, Kottayam;

The term "Dermatoglyphics" originated from two greek words "Dermato" means skin and "Glyphics" meaning carving and refers to the branch of science, which deals with the study of ridge patterns on fingertips, palms, soles and toes This term was coined in 1926 by Dr. Harold Cummins, the father of American fingerprint analysis.

In ancient India, the study of ridge pattern was known as "Samudra Shastra". The epidermal ridge patterns were classified into "Chakra, Shankya and Padma" which are today known as the Whorl, Loop and Arch system of modern classification. It was in 1823, that the skin stripes were recorded and documented for the first time by Czech doctor Pa Jinjie. At that time he had taken note that the surface layer of the palm is covered by wrinkles, which are made up of specific lines. He also noted that the fingerprint of each person is unique and fingerprints on the right hand are not the same as the ones on the left hand. While the fingerprints increase in size from childhood to adulthood, it will not change, as long as one is not seriously injured. Even when there is damage to the dermis, fingerprints will begin to show again along with healing of the wound

Widespread interest in epidermal ridges developed only in the last several decades when it became apparent that many patients with chromosomal aberrations had unusual ridge formations. Significant investigations have been carried out into the dermatoglyphic indicators of congenital heart disease, leukemia, cancer, celiac disease, intestinal disorders, rubella, schizophrenia as well as other forms of mental illness. Dermatoglyphic analysis is now beginning to prove itself as an extremely useful tool for preliminary investigations into conditions with a suspected genetic basis. Thus the study of dermatoglyphics is considered as a window of congenital abnormalities and is a sensitive indicator of intrauterine anomalies both dental as well as systemic.

It is widely known that by the seventh week of intrauterine life, the primary palate and lip develops in human embryo. Likewise the secondary palate development is completed by 12th week of intrauterine life. The development of dermatoglyphic patterns also begins in the 6th week of gestation with the appearance of fetal pads, that reach a maximum size between 12th and 13th weeks and ends with the appearance of finished patterns on the surface of the skin in the 24th week of gestation. This implies that genetic message present in the genetic make up of a person, normal or abnormal, is transmitted during this period and is also reflected by dermatoglyphics. Thus, the resulting ridge configurations are genetically determined. From this stage onwards, they are unaffected by the environment, and this explains their unique role, as an ideal marker for individual identification and the study of populations, as well as detection of defects due to intra-uterine irregularities in the early weeks of pregnancy.

The ectoderm, from which the epidermis is derived from, has a role in the formation of many specialized structures such as the teeth. When an intrauterine dermal damage occurs, naturally a tooth anomaly should be expected.

Methods of recording dermatoglyphics

Ink method

Faurot Inkless method

Transparent adhesive tape method.

Photographic method.

The most commonly used, inexpensive and rapid method is the Ink method. In *Ink method, the* Fingers and palm prints are recorded using the ink method described by Cummins and Midlo. The hands of the children are first washed with soap and water to remove dirt and oil from the ridged skin and blot dried to improve the quality of the prints. The finger prints (right and left) are recorded for the study by using black duplicating ink, which is applied on the fingers with cotton swab. The digits are guided and pressed firmly against the white bond paper clipped on to a board. The palm prints (right and left) are recorded using black duplicating ink, which is smeared on the palms and pressed on a sheet of recording paper which was kept firm. A sponge was placed beneath the paper, to record the hollow of the palms so as to eliminate any incorrect interpretation of the epidermal ridge pattern

Dermatoglyphic landmarks

The three basic dermatoglyphic landmarks found on the fingertip patterns are tri-radii, cores and radiants

i) Tri-radius: It is formed by the confluence of three ridge systems that form angles of approximately 120º with one another.

ii) Core: It is in the approximate centre of the pattern.

iii) Radiant: These emanate from the tri-radius and enclose the pattern area.

DERMATOGLYPHIC ANALYSIS: Dermatoglyphic analysis includes the following: Qualitative analysis that includes fingertip patterns and Quantitative analysis that includes total finger ridge count and finger ridge count

Qualitative Dermatoglyphic Analysis

Fingertip patterns

Galton in 1892 divided fingertip pattern into three: arches, loops, and whorls.

i) Arches: They are the simplest pattern found on fingertips. It is formed by succession of somewhat parallel ridges, which traverse the pattern area and form a curve that is concave proximally (Figure 1). Sometimes, this curve is gentle; at other times it swings more sharply so that it may also be designated as a low or high arch pattern respectively. The arch pattern is further subdivided into two types:

a) Simple or plain arch composed of ridges which cross the fingertip from one side to the other without recurving.

b) Tented arch composed of ridges that meet at a point so that their smooth sweep is interrupted.

ii) Loops: It is the most common pattern on the fingertip. A series of ridges enter the pattern area on one side of the digit, recurve abruptly, and leave the pattern area on the same side (Figure 2). Loops may vary considerably in shape and size. They may be large or small, tall or short, vertically or horizontally oriented, plain loop or double loop. Occasionally, transitional loops can be found which resemble whorls or complex patterns.

The loop pattern is subdivided into two types:

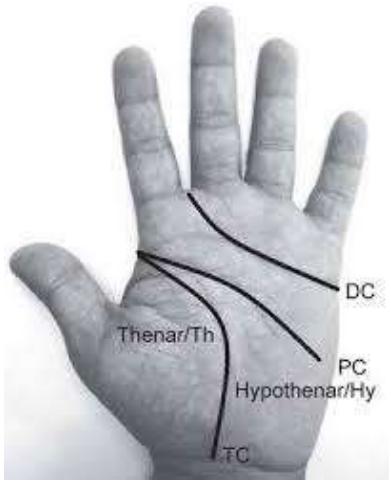
a) Ulnar loop composed of ridges that open on the ulnar side.

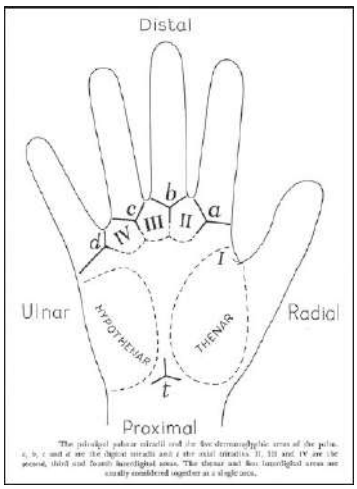
b) Radial loop composed of ridges that open on the radial side.

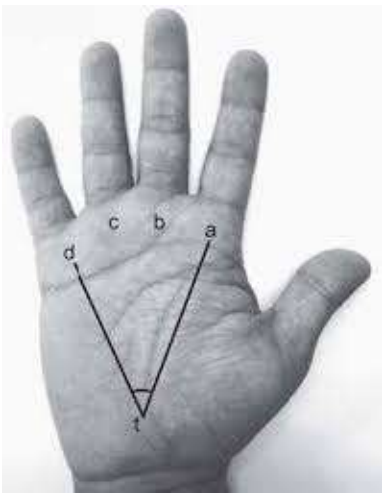
iii) Whorls: It is any ridge configuration with two or more tri-radii. One tri-radius is on radial and the other on the ulnar side of the pattern (Figure 3).

Subtypes of whorl patterns include:

a) Plain/simple/concentric whorl composed of ridges that are commonly arranged as a succession of concentric rings or ellipses.







b) Spiral whorl is a configuration in which ridges spiral around the core in either a clockwise or a counter clockwise direction.

c) Central pocket whorl is a pattern containing a loop within which a smaller whorl is located. Central pockets are classified as ulnar or radial according to the side on which the outer loop opens.

d) Lateral pocket/twinned loop pattern is composed of interlocking loops.

e) Accidental /complex patterns are one in which patterns cannot be classified as one of the above patterns. Some represent a combination of two or more configurations such as a loop and a whorl, triple loops and other unusual formation.

Quantitative Dermatoglyphic Analysis

Finger ridge count

A ridge count is made by drawing a line (blue line) from the triradius (green dot) to the center (red dot) of the pattern (core) and determining the number of intersected ridges between these two points. Arches score zero because they have no triradii and thus there are no ridges to count. A loop has one triradius. In whorls, which have two triradii, counts are made from each triradii and the larger one is used.

Total ridge count (TRC)

A total ridge count is the summation of the ridge count for all 10 fingers.

Palmar pattern

The palm is divided into 5 areas :Thenar area, Hypothenar area, Interdigital area II,III,IV, Palmar creases (DC, TC and PC) and atd angle. The thenar areas do not show any patterns. The 3 major palmar creases are-:

Distal crease (DC): originates from the lateral side of the palm and ends between the index finger and the middle finger.

Proximal crease (PC): starts from the hypothenar area and ends between the thumb and index finger.

Thenar crease (TC): originates from the base of the palm and ends between thumb and index finger finger.

Distal deviation of axial triradius or atd angle**.**

A feature of the palm that captures the relative position of three triradii - a and d, usually located on distal palm just inferior to the 2 nd and 5 th fingers, respectively and t whose location can vary on the proximal palm from just distal to the wrist, up to the center of the palm. The atd angles are measured for each palm print by drawing two straight lines through the "a" and "t" triradii and the "d" and "t" triradii and measuring the resulting angle.

The atd-angle is known for it's significance in Down syndrome (trisomy 21): most people who have Down syndrome have an atd angle > 57^{o} (80%), while in healthy people this characteristic is far less common (7%).

Advantages

The major advantages of the dermatoglyphics are:

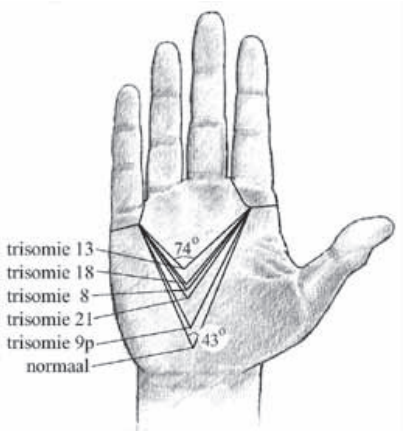
i) They are fully developed at birth and thereafter remain unchanged for life

ii) Scanning or recording of their permanent impressions can be accomplished rapidly, inexpensively, conveniently and without causing any trauma to the patient or hospitalization.

Limitations

1) The study of dermatoglyphic pattern becomes difficult if there is gross malformation of the limbs.

2) Recording of the prints must be done properly. More or less of ink material will lead to dark or light improper print.



3) The size of atd angle is important .it can be affected by the amount of spreading of the fingers during recording. pressure exerted can also affect the angle.

Dermatoglyphics in dentistry- related studies.

Recently, the association of irregular fingerprint in relation to congenital anomaly cleft lip palate, dental caries, periodontitis, malocclusion etc has drawn attention of researchers to the field of dental dermatoglyphics. The work of some researchers in the field of dental dermatoglyphics is summarized below.

Dermatoglyphics and dental caries.

Atasu et al found that there was significant difference in dermatoglyphic patterns in caries free group and extensive caries group children. The caries free students showed more ulnar loops on the fingertips and the students with extensive caries showed more whorls on the finger tips.

Sharma et al. found that the subject group had positive correlation with loops and Streptococcus mutans growth.

Mental Retardation

In a comparative study among 100 children (50-healthy, 50-mentally challenged) an increased frequency of loops and transverse palmar crease line among the mentally challenged children were observed.

Malocclusion

Tikare *et al.* (2010) revealed a statistical association between whorl patterns among Class 1 and 2 malocclusion children.

Bruxism

More of whorls and a less of ulnar loops were noticed in patients with bruxism than the controls. They also demonstrated a lower frequency of angle than controls.

Squamous cell carcinoma, leukoplakia

Venkatesh *et al.* (2008) in their study to determine the dermatoglyphic pattern in subjects with leukoplakia and oral squamous cell carcinoma found that among 30 patients diagnosed with leukoplakia, 30-70% had whorls, 6.30% had loop and arch type of fingerprint respectively and in oral squamous cell carcinoma patients it was found that 60 - 70% had a loop, 30-32 % had whorls, and 7.0% had an arch pattern of fingerprints.

Cleft lip and palate

Balgir (1992) in his study noticed an increased frequency of ulnar and radial loops in patients with cleft lip and palate than arches and whorls in patients without cleft lip and palate.

Scott et al showed increased radial and ulnar loops in CL/P patients.

Mathew et al studied dermatoglyphic patterns of 100 children between age of 5-15 years with no difference between sexes and observed that oral cleft individuals had an increased frequency of ulnar loops as the ridge configuration as compared to control group.

Saxena et al. studied 294 subjects (48 cleft subjects and 50 healthy controls with both their parents) and found increased frequency of loops and arches and low mean total ridge count in cleft subjects. Increased frequency of loops and arches with decreased frequency of whorls, mean total ridge count, and atd angle of right hand was found in parents of cleft group as compared with the parents of the controls.

Down's syndrome

A marked increase of the ulnar loops on the fingertips is virtually a constant feature of the dermatoglyphics in Down's syndrome.

Conclusion

Dermatoglyphics can prove to be an extremely useful, noninvasive and cost-effective tool for preliminary investigations into conditions with a suspected genetic base. Early detection can aid the clinician to anticipate oral health problems in the susceptible children and initiate preventive oral health measures at a very young age. Over the past 150 years, in addition to being the best and most widely used method for personal identification, dermatoglyphics has been a useful tool in understanding basic questions in biology, medicine, genetics and evolution.

However it still remains at infancy in the world of dentistry. Further large scale observations need be undertaken to evaluate the significance of these variations in the dermatoglyphic pattern in oral diseases.

References

1. Abhilash PR, Divyashree R, Patil SG, Gupta M, Chandrasekar T, Karthikeyan R. Dermatoglyphics in Patients with Dental Caries: A Study on 1250 Individuals. J Contemp Dent Pract 2012;13:266-274.
2. Mukherjee DP. How Scientists read palms. Sci Today 1980:15-21.
3. Bazmi BA, Ghosh C, Kar S , Mubtasum H, Sarkar S , Sengupta AB. A cross sectional study of dermatoglyphics and dental caries in Bengalee children. J Indian Soc Pedod Prev Dent 2013;31:245-248.
4. Madan N, Rathnam A, Bajaj N. Palmistry: A tool for dental caries prediction!. Indian J Dent Res 2011;22:213-218.
5. Pratibha Ramani, Abhilash PR, Herald J Sherlin, Anuja N, Priya Premkumar Chandrasekar, G.Sentamilselvi, et al. Conventional Dermatoglyphics–Revived Concept: A Review. International Journal of Pharma and Bio Sciences 2011; 2: 446-458.
6. Roopam K Gupta, Aruna K Gupta. New, easy and effective method to take dermatoglyphic prints. National Journal of Medical Research 2013; 3: 45-47.
7. Tikare S, Rajesh G, Prasad KW, Th ippeswamy V, Javali SB.Dermatoglyphics-a marker for malocclusion? Int Dent J 2010;60:300-4.
8. Balgir RS. Dermatoglyphics in cleft lip and cleft palate anomalies.Indian Pediatr 1993;30:341-6.
9. Preus M, Fraser FC. Dermatoglyphics and syndromes. Am J Dis Child 1972;124:933-43.
10. Kiran K, Rai K, Hegde AM. Dermatoglyphics as a non-invasive diagnostic tool in predicting mental retardation. J Int Oral Health 2010;2:95-100.
11. Atasu M. Dermatoglyphic findings in dental caries: a preliminary report.J Clin Pediatr Dent. 1998;22:147-9.
12. Sharma A, Somani R. Dermatoglyphic interpretation of dental caries and its correlation to salivary bacteria interactions: an in vivo study.J Indian Soc Pedod Prev Dent. 2009 Jan-Mar;27(1):17-21
13. Scott NM, Weinberg SM, Neiswanger K, Brandon CA, Daack-Hirsch S, Murray JC.Dermatoglyphic fingerprint heterogeneity among individuals with nonsyndromic cleft lip with or without cleft palate and their unaffected relatives in China and the Philippines. Hum Biol. 2005 Apr;77(2):257-66.
14. Mathew L, Hegde AM, Rai K. Dermatoglyphic peculiarities in children with oral clefts. J Indian Soc Pedod Prev Dent. 2005 Oct-Dec;23(4):179-82.
15. Saxena RS, David MP, Indira AP. Dermatoglyphic evaluation in subjects and parents of cleft lip with and without cleft palate. Cleft Palate Craniofac Journal .2013 Nov;50(6)

INFORMATION FOR AUTHORS

Journal of Clinical Dentistry

GUIDELINES

Manuscripts: Articles should be type written on one side of A4 size (21x28cm) White paper in double spacing with a sufficient margin. One Original and two high quality photostat copies should be submitted. The authors name is to be written only on the original copy and not on the two photostat copies. **In addition to the printed version, a CD containing the article file also should be submitted compulsorily.** Use a clear and concise reporting style. Journal of Clinical Dentistry reserves the right to edit,manuscript,to accommodate space and style requirements. Authors are advised to retain a copy for the reference.

Title Page: Title page should include the title of the article and the name, degrees, positions, professional affiliations of each author. The corresponding authors, telephone, e-mail address, fax and complete mailing address must be given.

Abstract: An abstract of the article not exceeding 200 words should be included with abbreviated title for the page head use. Abstract should state the purpose of the study, investigations, basic procedures and the main findings.

Tables: Tables should be self explanatory, numbered in roman numbers, according to the order in the text and type on separate sheets of paper like legends for illustrations.

Illustrations: Illustrations should be clearly numbered and legends should be typed on a separate sheet of paper, while each figure should be referred to the text. Good black and white glossy photographs or drawings drawn in black Indian ink on drawing paper should be provided. **Colour photographs will be published as per availability of funds. It will incur printing cost. Otherwise the cost of printing will be at the expense of authors and charges will be advanced by Journal of Clinical Dentistry at the time of receipt of accepted manuscripts.** Photographs of X-rays should be sent and not the original X-rays. Prints should be clear and glossy. On the back of each print in the upper right corner, write lightly the figure number and authors name; indicate top of the photograph with an arrow of word Top Slides and X-ray photographs should be identified similarly.

Photographs:- Authors photograph - Stamp size, Black and White, glossy photographs should be sent along with the article.

Reference: Reference should be selective and keyed in numerical order to the text in Vancouver Style (not alphabetical). Type them double spaced on a separate sheet of paper. Journal references must include authors names, article title, journal name, volume number, page number and year. Book reference must include authors or editors names, chapter title, book title, edition number, publisher, year and page numbers.

Copy right: Submission of manuscripts implied that the work described has and not been published before (except in the form of on abstract or as part of published lectures, review or thesis) and it is not under consideration for publication else where, and if accepted, it will not be published else where in the same form, in either the same or another language without the comment of copyright holders. The copyright covers the exclusive rights of reproduction and distribution, photographic reprints, video cassettes and such other similar things. The views/opinions expressed by the authors are their own. The journal bears no responsibility what so ever.

The editors and publishers can accept no legal responsibility for any errors, omissions or opinions expressed by authors. The publisher makes no warranty, for expression implied with respect to the material contained therein. The journal is edited and published under the directions of the editorial board who reserve the right to reject any material without giving explanations. All communications should be addressed to the Editor. No responsibility will be taken for undelivered issues due to circumstances beyond the control of the publishers.

Books for review: Books and monographs will be reviewed based on their relevance to Journal of Clinical Dentistry readers. Books should be sent to the Editor and will become property of Journal of Clinical Dentistry.

Return of articles: Unaccepted articles will be returned to the authors only if sufficient postage is enclosed with the manuscripts.

Subscription Rates: Free distribution for all the members of the Academic Forum, Govt. Dental College, Kottayam.

Inland Subscription: Rs. 500 per issue, Rs. 1000 for 1 Year, Rs. 2000 for 2 years, Rs. 3000 for 3 years.

All correspondence may please be send to the following address :

Dr. Baiju R.M.
Editor, Journal of Clinical Dentistry, Dept. of Periodontics,
Govt. Dental College, Gandhinagar, Kottayam - 686 008
Phone: 94472 79605 E-mail: jcdkdc@gmail.com

Management of an impacted central incisor - A case report

Rita Zarina [a], Ektah Khosla [b], Digesh B [c],

Abstract

Management of an impacted permanent maxillary central incisor can be often challenging as it plays an important role in facial aesthetics. Early detection of such impacted teeth is important to avoid complications that may arise due to further space loss. Surgical exposure followed by orthodontic extrusion using a ligature wire, hooks and elastics in a removable appliance was the treatment protocol to satisfactorily align this central incisor into place .

Address for correspondence:

[a] Professor, Govt. Dental College, Kottayam
[b] Professor, MBDC, Kothamangalam
[C] Asst Prof, Govt. Dental College, Kottayam

Impaction of permanent tooth is rarely diagnosed during the mixed dentition period, but an impacted central incisor is usually diagnosed accurately when there is delay in the eruption of tooth.

Tooth impaction may result from a number of local causes.

Causes of non-eruption and impactions are:

1. Arch length discrepancy
2. Presence of supernumerary teeth
3. Mucosal or bony barrier
4. Retained deciduous teeth.

Non eruption of maxillary incisors requires monitoring or intervention when-

a) There is eruption of contralateral teeth that has occurred greater than 6 months previously.

b) If the lower incisors have erupted more than 1 year previously.

c) There is deviation from normal sequence of eruption.

Treatment alternative for an impacted central incisor includes.

- Extraction of the impacted central incisor and restoration with a bridge or an implant later when growth had ceased.
- Extraction of the impacted central incisor and closure of the space substituting the lateral incisor for the central incisor with subsequent prosthetic restoration.
- Surgical exposure, orthodontic space opening and traction of the impacted central incisor into proper position.

Careful planning is required when moving an impacted tooth by orthodontic treatment. Impacted teeth can be properly positioned with orthodontic traction.

This article presents a case with horizontally impacted left central incisor. Combined surgical and orthodontic method was employed to bring the impacted teeth into proper position.

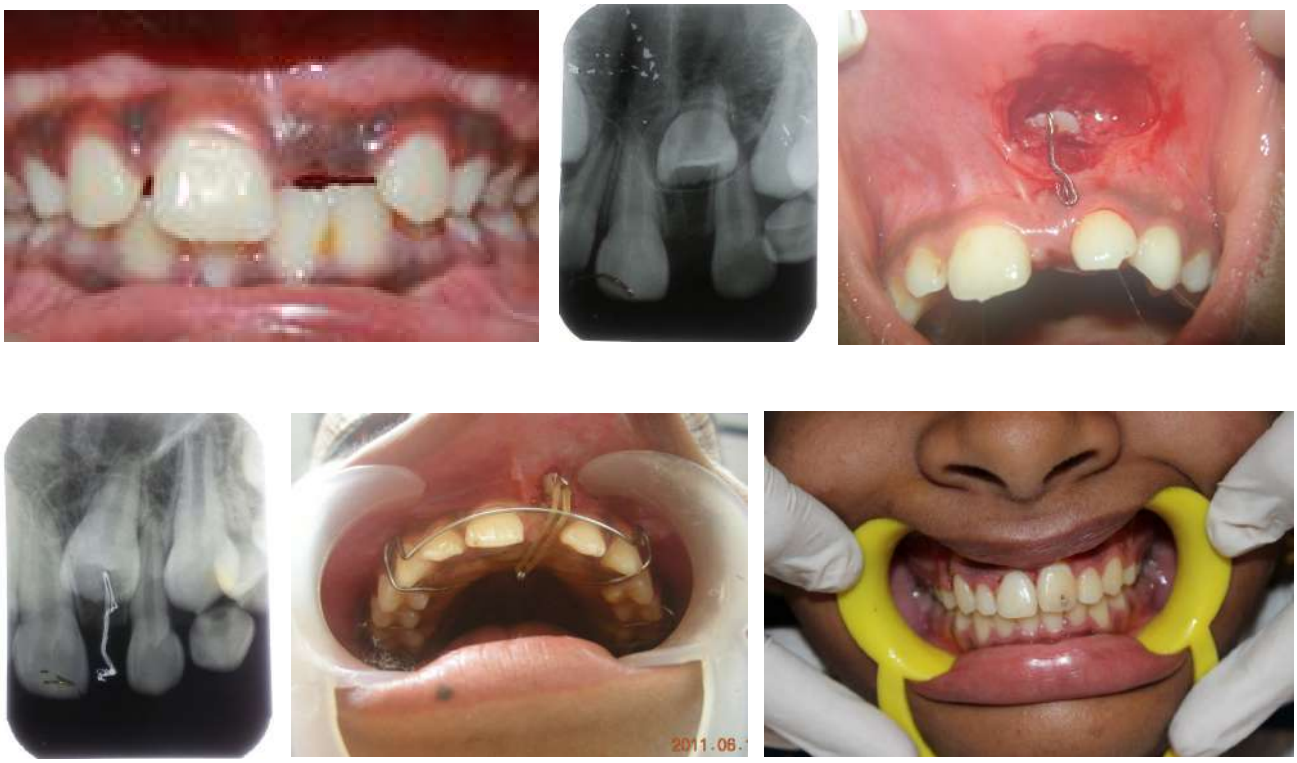
Case history.

A 9.5 year-old girl child reported to our department with an impacted left maxillary central incisor. The chief complaint was unerupted incisor (Fig 1). The child was in good health and had no history of medical or dental trauma.

Diagnosis and treatment planning

The child had an apparently symmetrical face with competent lips. Intraoral examination revealed early mixed dentition and an Angle's class I molar relationship. Clinical examination showed missing maxillary permanent left central incisor (Fig 1). There was no apparent arch length discrepancy in both maxillary and mandibular arches. An intraoral x ray of upper anterior region was taken which showed the presence of an horizontally impacted left central incisor (fig 2).

The possible treatment options were explained to the parents, including extraction of the impacted central



incisor, closure of the space, and alignment of the lateral incisor in place of the central incisor; extraction of the impacted central incisor and restoration with a bridge or an implant; and orthodontic traction of the impacted tooth. The patient and her parent opted for the non-extraction treatment; hence it was finally decided to expose the tooth and align it into the arch orthodontically.

Treatment progress

After achieving profound anesthesia, circular excision was made in oral mucosa overlying the impacted tooth. The alveolar bone overlying the impacted tooth was removed carefully to expose a small portion of the tooth. Since the tooth was quite deep and positioned in labio-palatal direction, the attachment could not be bonded. A small hole was drilled on the incisal edge with a fine tapering fissure bur. Ligature wire was passed through the hole and twisted in the form of a hook. The free end of the hook lay suspended in the labial surface of gingiva. A periodontal dressing was given for 24 hrs after which the pack was removed and yellow elastics were introduced. One end of the elastic was engaged in the modified hook and the other end on the hook place in the rugae region of Hawley's appliance. Patient was instructed to change elastics once in 2 days and reviewed periodically. After almost 3 months, the tooth became visible in the oral cavity. The ligature wire was replaced with a button bonded on the labial surface. The impacted tooth gradually moved towards the occlusal plane in a period of 9 months. The hole on the labial surface was restored.

Results

The impacted left maxillary central incisor was successfully aligned in proper position. The repositioned incisor had an acceptable gingival contour and width of attached gingiva. The post treatment radiograph showed no root resorption or periodontal bone loss. At follow-up, two years after the exposure of teeth, the tooth remained vital.

Discussion

Impacted maxillary permanent central incisor has serious impact on esthetics, phonetics, mastication, and psychology in young patients. Surgical exposure and moving the impacted tooth into normal occlusion with light force orthodontic traction is well accepted treatment modality.

Three accepted ways of surgical exposure have been suggested by Becker (1998) as:

a. Circular excision of the oral mucosa immediately overlying the impacted tooth.

b. Apically repositioning of the raised flap that incorporates the attached gingiva overlying the impacted tooth.

c. Closed eruption technique in which the raised flap that incorporates attached gingiva is fully replaced back in its former position after an attachment has been bonded to the impacted teeth.

According to Lin, 1999; Uematsu et al.,2004; Paola et al., 2005, many clinicians favour the closed eruption technique as they claim that the aesthetic and periodontal outcome is far more superior when compared with the circular excision and apically positioned flap technique. In the presented case, however circular excision was performed. The repositioned incisor did not exhibit any relapse or gingival recession at its labial margin, and it maintained the same clinical crown height as the right central incisor.

Uematsu et al. reported that extrusion forces greater than 50 grams should not be applied as it may cause nonvitality. Also ,the chances of nonvitality are naturally much lower when the treatment is initiated at a younger age due to the presence of a wide apical foramen. In the present case, which had a duration of 9 months, very light extrusion force was applied and also the patient was young (9.5 yr) at the time of initiation of treatment. This may have accounted for the little difference in the clinical crown length and maintenance of vitality of the impacted tooth post-alignment

Conclusion:

Surgical exposure and orthodontic correction offers a simplified treatment for impacted incisors. The advantages are immediate esthetic improvement; use of a single, simple surgical procedure; simple and short orthodontic therapy; normal gingival margins.

References

1. Becker A. The orthodontic treatment of impacted teeth.Mosby; 1998. p. 53-85
2. Uematsu S, Uematsu T, Furusawa K, Deguchi T, Kurihara S. Orthodontic treatment of an impacted dilacerated maxillary central incisor combined with surgical exposure and apicoectomy. Angle Orthod.2004; 74: 132-136
3. Paola C, Alessandra M, Roberta C. Orthodontic treatment of impacted dilacerated maxillary incisor. J. Clin. Pediatr.Dent. 2005; 30: 93-97
4. Tanaka E, Watanabe M, Nagaoka K, Yamaguchi K, Tanne K. Orthodontic traction of an impacted maxillary central incisor. J Clin Orthod 2001;35:375-8.
5. Crawford LB. Impacted maxillary central incisor in mixed dentition treatment. Am J Orthod Dentofac Orthop 1997;112:1-7.
6. Macias E, Carlos F, Cobo J. Posttraumatic impaction of both maxillary central incisors. Am J Orthod Dentofac Orthop 2003;124:331-8.
7. Kuvvetli SS, Seymen F, Gencay K. Management of an unerupted dilacerated maxillary central incisor: A case report. Dent Traumatol 2007;23:257-61.
8. Giampietro F, Cinzia M, Davide F. Orthodontic movement of a dilacerated maxillary incisor in mixed dentition treatment. Dent Traumatol 2009;25:451-6
9. Bayram M, Özer M, S¸ener I. Bilaterally Impacted Maxillary Central Incisors: Surgical Exposure and Orthodontic Treatment: A Case Report. J Contemp Dent Pract. 2006; 4:098-105.

Generalised florid cemento-osseous dysplasia: a rare case report

Twinkle S Prasad [a], S. Mohan [b], Bobby John [c], Anush. R [d], Annapoorani [e]

Abstract

Florid cemento-osseous dysplasia (FCOD) is a "fibro-osseous lesion" that characteristically affects the jaw bones of the middle-aged with multi-quadrant radiopaque cementum-like masses. The condition clearly appears to be a form of bone and cemental dysplasia that is limited to jaws. Patients do not have laboratory or radiologic evidence of bone disease in other parts of the skeleton. The diagnosis of FCOD is made on the basis of typical clinico-radiological features. Biopsy is usually not recommended due to the risk of postoperative infection. This paper reports a rare case of FCOD affecting maxilla as well as mandible bilaterally in a fifty-two years old woman.

Key words: Florid cemento-osseous dysplasia, fibro-osseous lesion, cemental dysplasia.

Address for correspondence:
[a] Associate Professor, Dept of Oral Medicine & Radiology
[b] Professor, [c] Assistant Professor,
[d] Senior Resident, [e] Junior Resident,
Dept of Oral & Maxillofacial Surgery,
Govt Dental College, Kottayam

Introduction

Cemento-osseous dysplasias are a group of disorders known to originate from periodontal ligament tissues. They are usually classified depending on their extent and radiographic appearances into three main groups: periapical (surrounds the periapical region of teeth and are bilateral), florid (sclerotic symmetrical masses) and focal (single lesion) cemental dysplasias[1]

Florid cemento-osseous dysplasia (FCOD), described by Melrose *et al.* for the first time in 1976; refers to a group of fibro-osseous (cemental) exuberant lesions that involve multiple quadrants of jaw bones.[2,3] Although FCOD is commonly seen in middle aged black females, the same is not uncommon in Caucasians and Asians.[4,5,6]

These lesions are clinically asymptomatic and may be found as incidental radiological finding presenting as multiple radiopaque masses within peripheral radiolucent rim located in two or more quadrants usually in tooth-bearing areas.[7]

Case report

A 55 year old female patient presented to the department of Oral Medicine & Radiology, Govt Dental College, Kottayam with a chief complaint of slowly growing upper and lower jaw. Patient had no other relevant medical history.

On inspection patient was completely edentulous with extensive expansion of maxillary and mandibular residual arch. The expansion was severe enough to cause difficulty in lip closure but not extended to the basal bone. It seemed to be restricted to the alveolar bone in both the jaws and the mucosa over the swelling was normal. The mouth opening was within normal limits. (Figure 1,2)

On palpation the swelling was non tender and bony hard involving the entire maxillary and mandibular arch

CT scan showed extensive sclerotic masses in maxilla extending into the sinus on the right side and mandible (Figure 3)

Diagnosis was made on the basis of clinical and radiological presentation of the lesion. Surgical recontouring was performed under GA to ensure lip competence and to enable the patient for prosthetic rehabilitation (Figure 4,5). There was no intra operative or post operative complications. Patient is under follow

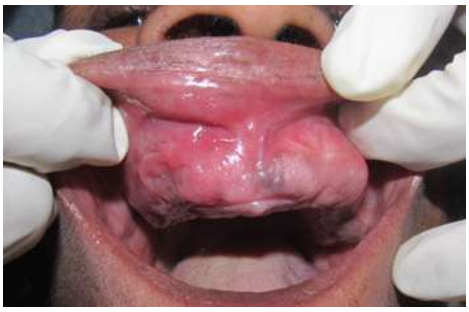


Fig 1: Pre operative picture showing maxillary arch enlargement

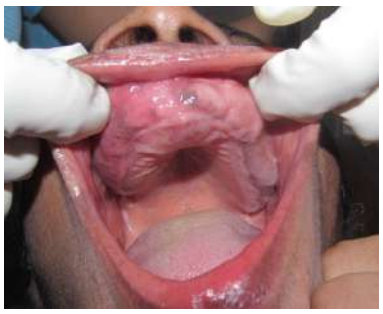


Fig 2: Occlusal view showing entire maxillary arch involvement

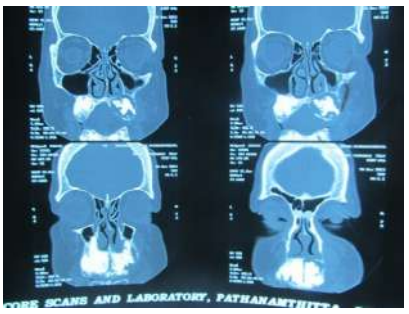


Fig 3: Coronal CT section showing sclerotic masses in maxilla

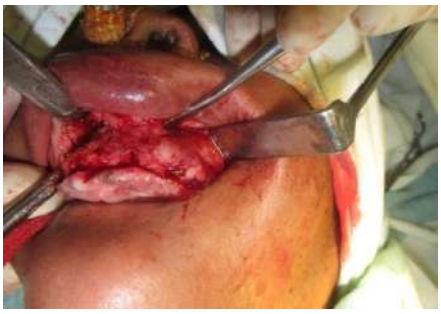


Fig 4: Intra operative picture showing surgical exposure of the lesion involving maxilla

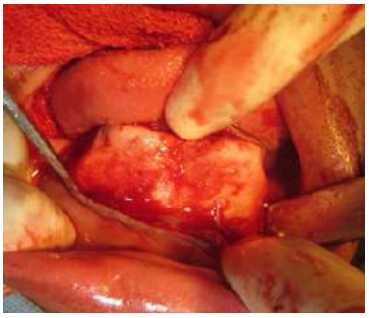


Fig 5: Intra operative picture showing surgical exposure of the lesion involving mandible

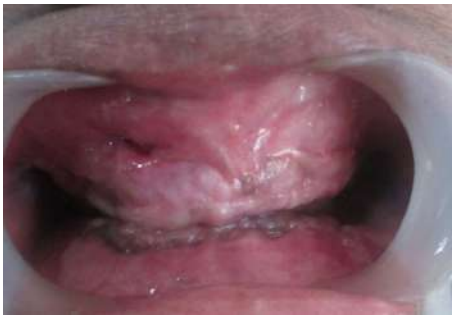


Fig 6: Post operative picture showing recontoured maxillary and mandibular arches

up since one and half year (Figure 6). There was no sign of recurrence till date.

Discussion:

FCOD refers to a set of radiolucent-radiopaque periapical and inter - radicular lesions involving the mandible bilaterally and sometimes the maxilla. FCOD occurs predominantly in black females (90%) with a mean age of 42 years at diagnosis. An intermediate frequency among East Asian population has also been described.[8]

FCOD is basically an extended form of periapical cementoosseous dysplasia. These lesions are also asymptomatic dysmorphic bone-cementum complexes. Radiographs show large, radiolucent, mixed, or most often dense radiopaque masses, limited to the periapical alveolar bone. They do not involve the inferior border except through direct focal extension and do not occur in the rami[9]. The present case was a severe form of FCOD involving all four quadrants including the angle and the basal bone in some areas of the mandible.

FCOD should be differentiated from Paget's disease, chronic diffuse osteomyelitis, and Gardner's syndrome. FCOD has no other skeletal changes, skin tumors or dental anomalies. Thus FCOD can be differentiated from Gardner's syndrome. Paget's disease is polyostotic and shows raised alkaline phosphatase level which is not a consistent feature of FCOD. Chronic diffuse sclerosing osteomyelitis is not confined to tooth-bearing areas. It is a primary inflammatory condition of the mandible with cyclic episodes of unilateral pain and swelling. The affected lesion of the mandible exhibits a diffuse opacity with poorly defined borders [10]

FCOD affecting multiple family members appears to be quite uncommon. There are only a few reports in which the hereditary nature of the lesion could be demonstrated[11,12,13]. Unlike the sporadic cases, the familial form is characterized by more expansile lesions, which may recur after surgery, and it tends to occur in younger subjects. In all of the familial cases reported, FCOD appears to be inherited as an autosomal dominant trait with variable phenotypic expression[11,13,14]. Toffanin et al[15] reported a case of FCOD affecting multiple family members. Some of the affected subjects had multiple impacted teeth, and one also had marked expansion of the symphyseal region. That patient underwent resection and reconstruction of the mandibular body with a free osteomyocutaneous fibula graft. In the present case, no familial aspects of the disease could be established.

Normally, the diagnosis of FCOD in the jaws is made through the clinical and radiographic features[16]. In the asymptomatic patient, it is probably wise to keep the patient under observation without surgical intervention. Due to the risk of osteomyelitis, biopsy is not required to confirm the diagnosis as this is usually established radiographically. It is not normally justified to surgically remove these lesions, as the surgery involved can be extensive. Instead, follow up and recontouring are recommended when cortical expansion occurs.[17] Whenever surgical treatment is planned, the lack of vascularity of the lesion and increased risk of osteomyelitis should be considered. The affected area undergoes changes from normal vascular bone into an avascular cementum-like lesion. Furthermore, complete removal of necrotic tissue may result in a large discontinuity defect[18]. However, in lesions causing pain and disturbance, surgery and the risks it entails might be necessary for adequate treatment. However, recontouring should be the treatment of choice where there is only cortical expansion and mucosal perforation due to the cementoosseous lesions[19].

Conclusion:

Asymptomatic cementoosseous lesion does not generally need surgical management. The present case was compromising the patient's aesthetics, lip competence and prosthetic rehabilitation. Hence surgical recontouring was done.

References:

1. Florid cemento-osseous dysplasia: A case reportSaadettinDaðistan, ÜmmühanTozoðlu, Mustafa Göregen, BinaliÇakur,Med Oral Patol Oral Cir Bucal 2007;12:E348-50.
2. Mangala M, Ramesh DN, Surekha PS, Santosh P. Florid cemento-osseous dysplasia: Review and report of two cases. Indian J Dent Res. 2006;17:131–4.
3. Kim JH, Song BC, Kim SH, Park YS. Clinical, radiographic, and histological findings of florid cemento-osseous dysplasia: A case report. Imaging Sci Dent. 2011;41:139–42.
4. Gonçalves M, Píspico R, Alves F de A, Lugão CE, Gonçalves A. Clinical, radiographic, biochemical and histological findings of florid cemento-osseous dysplasia and report of a case. BrazDent J. 2005;16:247–50.
5. Pitak-Arnnop P, Dhanuthai K, Chaine A, Bertrand JC, Bertolus C. Florid osseous dysplasia: Report of a case presenting acute cellulitis. Med Oral Patol Oral CirBucal. 2009;14:e461–4.
6. Mac Donald-Jankowski DS. Florid cemento-osseous dysplasia: A systematic review. DentomaxillofacRadiol. 2003;32:141–9.
7. Babaria U, Patel H, Pandya H, Dewan H, Bhavasar B, Thakkar D, et al. Florid osseous dysplasia: A case report. J Dent Sci. 2011;2:10–1.
8. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacialpathology, 3rd ed. Philadelphia: Saunders, 2009:613e77,701e40.
9. R. E. Marx and D. Stern, *Oral and Maxillofacial Pathology: A Rationale for Diagnosis and Treatment*, Quintessence, London, UK, 2004.
10. J.H. Kim, B. C. Song, S. H. Kim, and Y. S. Park, "Clinical, radiographic and histological findings of florid cemento-osseous dysplasia: a case report," *Imaging Science in Dentistry*, vol. 41, pp. 139–142, 2011
11. S. K. Young, N. R. Markowitz, S. Sullivan, T. W. Seale, and R. Hirschi, "Familial gigantiformcementoma: classification and presentation of a large pedigree," Oral Surgery Oral Medicine and Oral Pathology, vol. 68, no. 6, pp. 740–747, 1989.
12. H. O. Sedano, R. Kuba, and R. J. Gorlin, "Autosomal dominant cemental dysplasia," Oral Surgery Oral Medicine and Oral Pathology, vol. 54, no. 6, pp. 642–646, 1982.
13. H. Coleman, M. Altini, J. Kieser, and M. Nissenbaum, "Familial florid cemento-osseous dysplasia–a case report and review of the literature," The Journal of the Dental Association of South Africa, vol. 51, no. 12, pp. 766–770, 1996.
14. J. S. Cannon, E. E. Keller, and D. C. Dahlin, "Gigantiformcementoma: report of two cases (mother and son)," *Journal of Oral Surgery*, vol. 38, no. 1, pp. 65–70, 1980.
15. A. Toffanin, R. Benetti, and R. Manconi, "Familial florid cemento-osseous dysplasia: a case report," *Journal of Oral and Maxillofacial Surgery*, vol. 58, no. 12, pp. 1440–1446, 2000.
16. D. J. D. S. Sarmento, B. V. de BritoMonteiro, A. M. C. de Medeiros, and E. J. D. da Silveira, "Severe florid cemento-osseous dysplasia: a case report treated conservatively and literature review," *Oral and Maxillofacial Surgery*, vol. 17, no. 1, pp. 43–46, 2013
17. W. Jerjes, B. Banu, B. Swinson, and C. Hopper, "Florid cemento-osseous dysplasia in a young Indian woman. A case report," *British Dental Journal*, vol. 198, no. 8, pp. 477–478, 2005.
18. S. Bencharit, D. Schardt-Sacco, J. R. Zuniga, and G. E. Minsley, "Surgical and prosthodontic rehabilitation for a patient with aggressive florid cemento-osseous dysplasia: a clinical report," *Journal of Prosthetic Dentistry*, vol. 90, no. 3, pp. 220–224, 2003.
19. 22.S. KayaaltýÖzarslan, H. H. Ýylmaz, M. Ç. Aksoy, and T. Baykul, "Florid cemento-osseous dysplasia: a case report," Journal of Dental Faculty of Atatürk University, vol. 4, supplement, pp. 128–132, 2011.

Diagnostic approach to the dental patient with bleeding disorders: Step 1-evaluation

S Mohan [a], Twinkle S Prasad [b]

Abstract

Easy bruising and excessive bleeding may be ends of a spectrum of altered haemostatic apparatus, but may innocuously lead to a life threatening situation in a matter of minutes. The key to management lies in an adroit history, clinical and laboratory evaluation. Once the patient with bleeding problem has been identified, steps can be initiated for reduction of risks.

Address for correspondence:

[a] Professor & Head of Department, Department of Maxillofacial surgery, [b] Associate Professor, Department of Oral Medicine & Radiology, Government Dental College, Kottayam.

Introduction

While bruising is a confluent purplish discoloration of the skin due to extravasations of blood, excessive bleeding is bleeding beyond physiologic expectations. Spontaneous hemorrhage is always abnormal, except for occasional self-limiting epistaxis & menstrual bleeding. It must be recognized, however, that the adequacy of hemostasis is only relative, and despite the presence of normal vessels, platelets and coagulation factors, bleeding can occur as a result of localized pathologic processes, especially inflammation. A stepwise clinical approach is mandatory, starting with detailed history and key questions, which provide valuable clues as to whether the abnormality resides in the vessels, platelets or the coagulation process, and whether the disorder is inherited or acquired. Physical evaluation that follows may reveal characteristic lesions of the skin and oral mucosa. Results of the evaluation will lead to a differential diagnostic template, followed by a rational laboratory evaluation to supplement and confirmation of diagnosis by sieving the template. This review stresses on the art of diagnosis in this special setting.

Clinical approach

Acquired bleeding disorders are common. They complicate well defined clinical disorders which can be detected by history and examination. Inherited bleeding disorders are uncommon, but can be detected by careful clinical assessment, including family history.

History should include nature & severity of persistent bleeding from shaving wounds, menstrual flow, bleeding episodes requiring medical attention, iron deficiency anemias, blood transfusions, episodes of epistaxis, gum bleeds & bruising, family history of bleeding disorder, drugs & herbal medications, liver disease, bleeding after trauma & surgery, spontaneous hemarthrosis, palatal petechiae, palpable purpuras and joint deformities.

Causes of bleeding

- *Local* Infection.
  Local irritants
  Post surgical/ post traumatic/iatrogenic
  Bleeding ulcers, rupture of bulla
  Congenital malformation (HHT, Hemangiomas)
- *Systemic*

1) Non-thrombocytopenic purpuras (vascular wall disorders)

a) *Inherited*- HHT
Disorders of collagen matrix (Marfan's syndrome, Ehlers Danlos syndrome)

b) *Acquired*- scurvy, infections, allergy, chemicals

2) Platelet disorders

a) Number: Thrombocytopenias
b) Function: *Hereditary*-Bernard-Soulier syndrome, von Willebrand's disease
Acquired · Drugs-NSAIDs, Alcohol, Aspirin
· Allergy

- Autoimmune disease
- Uremia
- Liver disease
- Multiple myeloma

3) Coagulation disorders

a*) Inherited*- Hemophilia A, Hemophilia B, von Willebrand disease

b) *Acquired*

- Liver disease,
- Vitamin deficiency,
- Anticoagulant drugs,
- DIC
- Primary fibrinogenolysis

Interpreting a Hemorrhagic History

The hemophilias follow a pattern of X linked recessive inheritance. However up to 30% of cases of hemophilia A are spontaneous mutations with no family history. Any patient, who has had major surgery, a tonsillectomy or dental extractions without unusual bleeding, has had the best evaluation of their coagulation system possible. A bleeding abnormality manifests as moderate bleeding over a prolonged period, not as bleeding at an excessive rate. Bleeding from skin and mucous membranes tends to occur with platelet disorders, while bleeding in joints and muscles tends to occur with the haemophilias. History of medical disease should include liver disease, renal disease, hematological malignancy (leukemias), myeloproliferative disease, Vitamin K deficiency, solid organ malignancies of prostate, lung & colon.

The mode of enquiry

The age of onset and sex is specifically asked for, since hereditary disease manifests in early age. For elderly, an internal malignancy may be suspected or senile purpura and purpura simplex may be looked for. Hemophilia A & B occur in males only. Spontaneous bleed occur in moderate to severe hemophilia. Prolonged bleed only on trauma or following surgery indicate mild coagulation defect. Bleeding from a single site usually indicate local cause, while multiple sites of bleeding warrants ruling out systemic cause. History of transfusions indicates severe disease. A diet history deficient in leafy vegetables or a recent antibiotic usage indicates vitamin K deficiency.

Clinical examination

General examination should specifically include anemia, jaundice, clubbing, cushinoid / myedematous features. Skin should be examined for telangiectasias, elasticity of skin, hemangiomas and palpable purpuras. Abdomen should be examined for organomegaly, venous engorgements and ascites. Joints need to be looked for swelling, tenderness & deformity[1]. Clinical signs & symptoms are arbitrarily divided in to those seen more in platelet & vessel disorders and those more common in coagulation disorders. Table 1 summarizes the clinical distinction.[2]

Gastrointestinal bleeding and/or epistaxis as isolated problems are unlikely to be due to an inherited bleeding disorder. Mucosal surfaces should always be inspected for the characteristic lesions of hereditary hemorrhagic telangiectasia. Telangiectasias on the face, lips, oral or nasal mucosa, and tips of the fingers and toes in a patient

Clinical signs/ symptoms	*Coagulation disorders (purpuric disorders)*	*Disorders of platelets or vessels*
Petechiae	Rare	Characteristic
Deep, dissecting hematomas	Characteristic	Rare
Superficial ecchymoses	Common: usually large & solitary	Characteristic: usually large & multiple
Hemarthrosis	Characteristic	Rare
Delayed bleeding	Common	Rare
Bleeding from superficial cuts/ scratches	Minimal	Persistent, often profuse
Sex of patient	80-90% of inherited forms occur only in males	Relatively more common in females
Positive family history	Common	Rare (except VWD)

Table 1

with a positive family history of excessive bleeding is likely hereditary hemorrhagic telangiectasia. Bleeding from superficial sites, including skin and mucous membranes, suggests a quantitative or qualitative defect in platelets or a defect in blood vessels (eg, amyloidosis). Bleeding into deep tissues (eg, hemarthroses, muscle hematomas, retroperitoneal hemorrhages) suggest a defect in coagulation. Bleeding in a patient who is pregnant or has recently delivered, who is in shock, or who has a serious infection suggests disseminated intravascular coagulation (DIC). Patients with known alcohol abuse or liver disease may have coagulopathy, splenomegaly, or thrombocytopenia. In patients with a history of i/v drug abuse, HIV infection should be considered.[3]

Retro orbital masses, bone tumors and tumors at different sites are seen in acute leukemias, myeloma and CML. Bone pain caused by increased pressure of the hyperplastic marrow is seen in acute leukemias, CML, Multiple myeloma and hemoglobinopathies. Dysphagia and koilonychias is frequent in iron deficiency states. Conjunctival congestion and plethoric appearance is seen in polycythemia. Chronic leg ulcers are seen in hemolytic anemia.[4] Table 2 summarizes the differential diagnosis of easy bruisability.

An inherited bleeding disorder is suggested by onset of bleeding symptoms in infancy and child hood, a positive family history and laboratory evidence of a single coagulation factor aberrance or deficiency. A negative family history is of no value in excluding an inherited coagulation disorder in an individual patient, as 30-40% of patients with hemophilia A with autosomal recessive traits and in consanguinity, has negative family history.[5] In acquired bleeding disorders, bleeding manifestations are usually less severe than inherited forms, and clinical picture is dominated by underlying disease than bleeding.

Steps of hemostasis

Primary Hemostasis

- Platelet Plug Formation, Dependent on normal platelet number & function

Secondary Hemostasis

- Activation of Clotting Cascade

Deposition & Stabilization of Fibrin

Tertiary Hemostasis

- Dissolution of Fibrin Clot

Dependent on Plasminogen Activation

Whenever a vessel is severed or reputed hemostasis is achieved mainly through four mechanisms.

i. Vascular spasm.

ii. Formation of platelet plug.

iii. Blood coagulation

iv. Eventual growth of fibrous tissue to close the defect in the vessel permanently

Immediately after a vessel is ruptured, the stimulus of the traumatized vessels causes the wall of the vessel to contract; this instantaneously reduces the flow of blood from the ruptured vessel. The contraction results from nervous reflexes, local myogenic spasm and local humoral factor from the traumatized tissues and blood platelets. For smaller vessels, the platelets are responsible for much of the vasoconstriction by clearing the vasoconstrictor substance "Thromboxane A_2".

When platelets come in contact with a damaged vascular surface such as the collagen, fibres in the vascular wall or even damaged endothelial cells, they immediately change their characteristics drastically. They

Diagnosis	*Manifestation*
Hereditary hemorrhagic telangiectasia	Mucocutaneous telangiectasia appearing in third decade
Ehlers danlos syndrome	Skin hyper extensibility and joint hyper mobility
Osteogenesis imperfecta	Retinal angioid streaks and yellow cutaneous plaques in flexural sites
Pseudoxanthoma elasticum	Retinal angioid streaks and yellow cutaneous plaques in flexural sites
Scurvy	Perifollicular bleed
Multiple myeloma & amyloid disease	Periorbital and pinch purpura
Steroid excess purpura	Purpura associated with red brown skin pigmentations
Small vessel vasculitis	Purpura associated with red brown skin pigmentations
Psychogenic purpura	Bruising associated with menstrual cycle
Senile purpura	Purpura associated with shearing strain sites like specs frame

Table 2

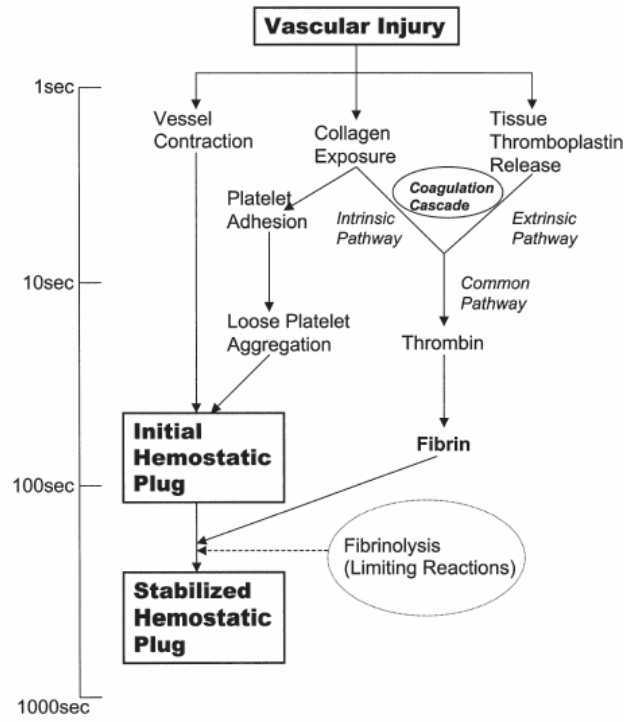


Fig 1: The Hemostatic Mechanism

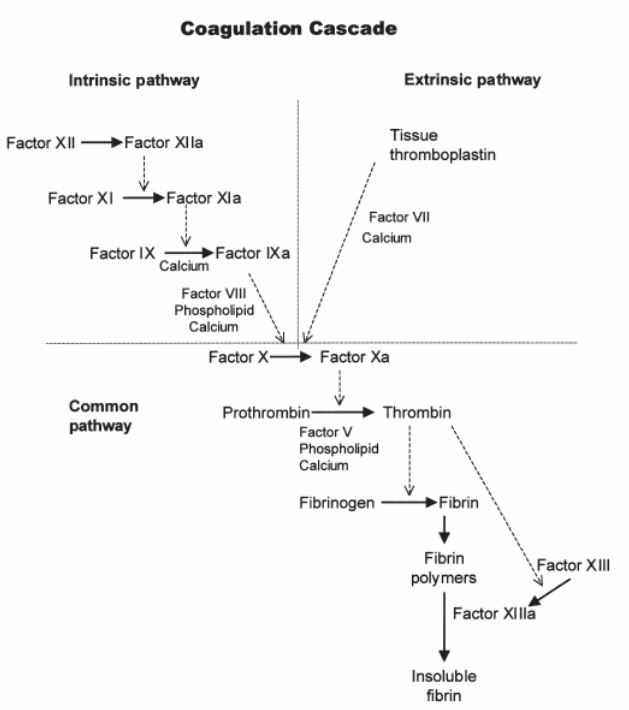


Fig 2: The Coagulation Cascade

begin to swell, and assume irregular forms with numerous radiating processes protruding from their surfaces, their contractile proteins contract forcefully and cause the release of granules containing multiple active factors, they become sticky so that they stick to the collagen fibres, secrete large quantities of ADP, and their enzymes form thromboxane A_2, which is also secreted into the blood. The ADP and thromboxane A_2 in turn act on nearby platelets to activate them as well, and the stickness of these additional platelets cause them to adhere to the originally activated platelets thus forming the platelet plug. At first it is a loose plug, later the fibrin threads become attached to it forming a tight and unyielding plug.

The third mechanism for hemostasis is the formation of blood clot. The clot begins to develop in 15 to 20 sec if the trauma of the vascular wall has been severe and in 1 to 2 min if the trauma has been minor. Activator substances both from the traumatized vascular wall and from platelets and blood proteins adhering to the traumatized vascular wall initiate the clotting process. Within 3 to 6 min after rupture of a vessel, if the vessel opening is not too large, the entire openings become filled with clot. After 20 min to an hour, the clot retracts and this closes the vessel further.

Once the blood clot has formed it can follow two separate courses. It can become invaded by fibroblasts which subsequently form connective tissue all throughout the clot or it can dissolve. The usual course for a clot that forms in a small hole of a vessel wall is invasion by fibroblasts, beginning within a few hours after the clot is formed and continuing to complete organization of the clot into fibrous tissue within approximately 1 to 2 weeks. On the other hand, when blood coagulates to form a larger clot, such as blood that has leaked into tissues, special substances within the clot itself become activated and these then function as enzymes to dissolve the clot itself. (Figures 1 & 2)

Laboratory investigations

No single test is suitable for the laboratory evaluation of the overall process of hemostasis. In a patient with a suspicion of bleeding disorder, the dentist can order the screening tests. Patients with positive screening tests are to be evaluated further by a hematologist, who arrives at a diagnosis and gives recommendations for the treatment.

Three tests are recommended for initial screening: activated partial thromboplastin time (aPTT), prothrombin time(PT), and platelet count.[6,7] If no clues

are evident, two additional tests can be added; platelet function analyser (PFA 1000) and thrombin time (TT).[8] The patient with positive screening tests are further evaluated for specific factor deficiency and for presence of inhibitors.

Platelet disorders are further evaluated by platelet aggregation tests, ristocetin induced platelet aggregation, ristocetin cofactor activity, immunoassay of vWF, multimeric analysis of vWF and specific factor VIII assays.[7, 8]

Disorders of the intrinsic pathway

Screening tests show prolonged aPTT, normal PT and normal platelet count. The next step is to mix (mixing tests) the patient's blood with a sample of pooled plasma and repeat the aPTT. If this test is normal, the specific missing factor is identified by specific assays. If the test is abnormal, inhibitor activity tests (antibodies to the factor) are performed.

Disorders of the extrinsic pathway

A normal aPTT & prolonged PT suggest factor VII deficiency, or inhibitors to factor VII. Factor VII deficiency is confirmed by specific assay, while mixing studies is used to rule out factor VII inhibitors.[9]

Disorders of the common pathway

A prolonged aPTT and a prolonged PT in a patient with congenital bleeding disorder indicate a common pathway factor deficiency. Congenital deficiency of factors V and X, prothrombin or fibrinogen is rare. When both these tests are prolonged, an acquired common pathway factor deficiency is indicated.[10]

Disorders of fibrinogen and fibrin products

In patients with prolonged aPTT, PT and TT, the defect involves the last stage of common pathway, which is the activation of fibrinogen to form fibrin, to stabilize the clot. The plasma level of fibrinogen is determined, and if within normal limits, tests for fibrinolysis are performed, viz staphylococcal clumping assay, agglutination of latex particles coated with antifibrinogen antibody and euglobulin clot lysis time.[11, 12]

Disorders with normal primary screening tests

Patients with vascular abnormalities may not show positive screening tests. BT is the only test that might be abnormal in these patients. Three known defects in the coagulation system do not affect PT, aPTT or TT. These are factor XIII deficiency, alpha-2 plasmin inhibitor deficiency and PAI-1 deficiency (major inhibitor of plasminogen activators) These patients require additional testing, such as use of 5M urea.[13, 14] Table 3 summarizes the profile of screening tests in hemostasis.

The interpretation of Laboratory results

PT and aPTT evaluate the coagulation phase of hemostasis. PT evaluates the extrinsic pathway and is commonly elevated in patients taking Coumadin. A prothrombin time within normal range indicates that the patient has normal amounts of clotting factors VII and X (extrinsic and common pathways of coagulation). The PT is reported as the international normalized ratio (INR), which reflects the ratio of the patient's PT to the laboratory's control value; the INR controls for differences in reagents among different laboratories. The aPTT tests for deficiencies of all (intrinsic) clotting factors except factor VII (measured by the PT) and factor XIII and is elevated in hemoplilia A, B, C and vWD. A normal result indicates that at least 30% of all coagulation factors in the pathway are present in the plasma. Patients taking heparin are evaluated with APTT.

PT	*APTT*	*platelet Count*	*Differential Diagnosis*
increased	normal	normal	Acquired factor VII deficiency (early liver disease, early vitamin K deficiency , early warfarin therapy)
normal	increased	normal	Deficiency or inhibitor of factors VIII,IX or XI; vWD; Heparin
increased	increased	normal	Vit K deficiency, liver disease, warfarin, heparin
increased	increased	decreased	DIC, Liver disease
normal	normal	decreased	Increased platelet destruction, hypersplenism, hemodilution,
normal	normal	increased	inherited platelet disorders, myeloproliferative disorders
normal	normal	normal	Mild vWD, uremia, fibrinolytic disorders, factor XIII deficiency

Table 3

The platelet count evaluates the platelet phase of coagulation and indicates the number of platelets present. It does not evaluate qualitative defects but only quantitative ones.[15] The bleeding time evaluates the platelet phase and vascular phase, especially the patient's ability to clot small vessels up to 50 microns in diameter. It depends largely on the rate of formation of platelet plug and is mostly independent of fibrin forming coagulation mechanisms. An abnormal aPTT or PT may lead to evaluation of factor assays. The normal range of individual factor assays is between 50-150%.[16] Patients who develop inhibitors to factor VIII are evaluated by the Bethesda assay with the Bethesda unit (BU). The patients with inhibitors are divided in to low responders (less than 20 BU) who make up 25% and high responders (greater than 20 BU at least once) who make up the rest 75%.[17]

A patient with suspected factor VIII (hemophilia A) or IX deficiency will have a prolonged aPTT, normal PT & TT. All other individual clotting factors are normal. For a factor XI deficiency, APTT will be prolonged, with normal PT & TT, but factor XI will not decrease severely and factors VIII & IX will be normal. In factor XII, High molecular weight kininogen & prekallikrein deficiency, APTT will be prolonged with normal PT & TT, along with a decrease of these factors, but there will be no clinical bleeding. In inhibitor deficiencies, there will be lack of correction of aPTT after incubation of a mixture of one half normal plasma and one half patients' plasma and increase of factors VIII or IX with a lack of shortening of aPTT after infusion of clotting factor concentrate.

Von Willebrand's Disease type I is a quantitative defect of VWF, with a prolonged aPTT & PT, abnormal ristocetin induced platelet aggregation & ristocetin co factor, decrease in factor VIII & VW antigen and a decrease in all multimers of the molecule. In type II VWD, there is a selective deficiency of plasma high molecular weight VWF multimers, decrease in VWF antigen and a decrease in factor VIII. The type III VWD is rare, but most severe with very low levels of VWF and factor VIII.

In liver disease acquired clotting defects, a prolonged PT, decrease in factors V (most specific), VII, II, X, IX , I & antithrombin, normal factor VIII and evidence of activated fibrinolysis or DIC will be seen, along with thrombocytopenia. In vitamin K deficiency or in the event of K inhibitors, PT will be prolonged with normal TT and a decrease in factors VII (most sensitive), X, II, IX, proteins C & S with normal factors V & VIII.[18]

Agent	*Monitoring*	*Complications*
STANDARD HEPARIN HIGH DOSE	APTT 1.5-2 times control	Bleeding, thrombocytopenia
STANDARD HEPARIN LOW DOSE	None	Bleeding, thrombocytopenia
WARFARIN	INR 2.0-3.0	Bleeding, Intolerance:rash, skin necrosis, alopecia
LOW MOLECULAR WEIGHT HEPARIN	None	Bleeding, thrombocytopenia, fever, peripheral odema
ENOXAPARIN (LMWH)	Oral warfarin started within 72 hours	bleeding
SYNTHETIC HEPARINS	none	bleeding
DIRECT THROMBIN INHIBITORS	APTT 1.5-2.5 times lab normal test time	Bleeding, allergy, anaphylaxis
ASPIRIN	Usually none but bleeding time can be used	GI bleeding, tinnitus, urticaria, bronchospasm
NSAIDs	none	GI bleeding, tinnitus, urticaria, bronchospasm
ADP INHIBITORS:CLOPIDOGREL	CBC every two weeks	GI bleeding, thrombocytopenia, diarrhea
FIBRINOGEN RECEPTOR INHIBITORS	none	GI bleeding, thrombocytopenia, neutropenia, rash

Table 4

Antithrombotic therapy

Excess activation of coagulation or inhibition of anticoagulant mechanisms may result in hypercoagulability and thrombosis. Patients should be considered for evaluation of inherited thrombotic disease if they are younger than 45 years of age and have recurrent thrombosis. The currently used antithrombotic drugs and monitoring tests required are summarized in Table 4.[19]

Routine preoperative INR, BT & PT are not very helpful in predicting operative and post operative bleeding. A history of erratic INR values in the past is significant. If values are supra therapeutic (INR > 3), the risk of postoperative bleeding increases significantly.[20]

Conclusion

Any type of bleeding is a severe symptom that needs prompt professional medical diagnosis. Patients with coagulopathies and those patients who are anticoagulated are difficult management problems when surgical intervention is planned. Proper evaluation is the first step of management of these patients. A diagnostic approach to the bleeder patient is reviewed here. The next step is to effect management strategies.

References

1. Pai R. Bleeding In: Symptom analysis for medical students.1st ed.p.246-48.
2. Biggs R. Human blood coagulation, Hemostasis and Thrombosis. Oxford: Blackwell Scientific, 1976.
3. Wahlberg T,et al. Application of indicators, predictors and diagnostic indices in coagulation disorders. Meth Inform Med 1980; 19:194.
4. Rochems CA. Approach to the bleeding patient. Hematol Oncol Clin North Am 6: 983-989,1992.
5. Biggs R, MacFarlane RG. Hemophilia and related conditions: A survey of 187 cases. Br J Hematol 1958; 4:1.
6. Rodgers GM, Bithell TC. The diagnostic approach to bleeding disorders. In Lee GR, Foerster J et al(eds). Wintrobes Clinical Hematology,10th ed. Philadelphia, Lippincott Williams & Wilkins, 1999.pp 1557-1578.
7. Rand JH, Senzel L. Laboratory evaluation of hemostatic disorders. In Hoffman R, Benz EJJ, et al(eds). Hematology:Basic Principles and Practices, 4th ed. Philadelphia, Elsevier-Churchill Livingstone, 2005, pp 2001-2011.
8. Watzhe HH. Evaluation of the acutely bleeding patient. In Young NS, Gerson SL (eds). Clinical Hematology. St Louis, Elsevier-Mosby, 2006, pp 1169-1179.
9. Plow EF. The molecular basis for platelet function. In Hoffman R, Benz EJJ(eds) Hematology:Basic Principles and Practices, 4th ed. Philadelphia, Elsevier-Churchill Livingstone, 2005, pp 1881-1889.
10. Furie B, Furie BC. Molecular basis of blood coagulation. In Hoffman R, Benz EJJ(eds) Hematology:Basic Principles and Practices, 4th ed. Philadelphia, Elsevier-Churchill Livingstone, 2005, pp 1931-1955.
11. Greenberg CS, Orthner CL. Blood coagulation and fibrinolysis. In Lee GR, Foerster J et al(eds). Wintrobes Clinical Hematology,10th ed. Philadelphia, Lippincott Williams & Wilkins, 1999.pp 684-765.
12. Handin RL. Disorders of coagulation and thrombosis. In Kasper DL, Braunwald E, Fauci AS, et al(eds). Harrison's Principles of Internal Medicine. New York, NY, McGraw-Hill, 2005, pp 680-687.
13. McVey JH. Coagulation factors. In Young NS, Gerson SL (eds). Clinical Hematology. St Louis, Elsevier-Mosby, 2006, pp 103-123.
14. Handin RL. Bleeding and thrombosis. In Kasper DL, Braunwald E, Fauci AS, et al(eds). Harrison's Online Principles of Medicine, 16th ed. New York, NY,McGraw-Hill, 2005, pp 337-343.
15. Redding SW. Oral bleeding. In: Montgomery MT, Redding SW (eds). Orofacial emergencies. Chicago: Federation of Special Care Organizations in Dentistry; 1994, p 103.
16. Olive JA. Disorders of hemostasis. In Tullman MJ, Redding SW, editors. Systemic disease in dental treatment. New York: Appleton Century Crofts; 1982, p 195.
17. Redding SW, Stiegler KE. Dental management of the classic hemophiliac with inhibitors. Oral Surg Oral Med Oral Pathol 1983: 56(1); 145-8.
18. Meehan S, Schmidt MC, Mitchell PF. The international normalized ratio as a measure of anticoagulation: significance for the management of the dental outpatient. Special care in Dentistry 1997; 17(3): 94-6.
19. Francis CW, Kaplan KL. Venous and arterial thrombosis. In Young NS, Gerson SL (eds). Clinical Hematology. St Louis, Elsevier-Mosby, 2006, pp1089-1106.
20. Goyal M, Rajagopal A. Anticoagulants and dentistry. Dental Practice: Mar-April 2012, Vol 10, No 6, pp 42-44.

Unusual morphology of permanent maxillary central incisor- a case report

Shiney Dominic [a], S Mohan [b], Bobby John [c], Dibin R [d]

Abstract

Dilacerations - The term refers to an angulation, which can occur anywhere along the length of the tooth—ie, its crown, cementoenamel junction, along the root, or only involving the apex of the root resulting in disruption of the normal axial relationship of the tooth. Dilaceration of the permanent teeth usually occurs because of trauma to their deciduous predecessors. Dilaceration in the crown is rare compared with that in the root and is more common in the maxillary incisors followed by mandibular anterior teeth. Generally, surgical exposure of the tooth followed by orthodontic traction or extraction of the tooth is the treatment of choice. This article highlights a rare case of crown dilaceration of permanent maxillary central incisor.

Key words: dilacerations, maxillary incisors, surgical removal

Address for correspondence:

[a] Associate Professor, [b] Professor & HOD, [c] Assistant Professor, [d] Junior Resident, Department of Oral & Maxillofacial Surgery, Government Dental College, Kottayam

Introduction

Dilaceration is an angulation or sharp bend in the root or crown of a formed tooth[1]. Stewart[2] compared tooth dilacerations with "the hand of a traffic policeman." Dilaceration refers to anangulation, which can occur anywhere along the length of the tooth—ie, its crown, cementoenamel junction, along the root, or only involving the apex of the root resulting in disruption of the normal axial relationship of the tooth[15]. It is more common in the maxillary central incisors followed by mandibular central and lateral incisors[3] and is usually caused by trauma occurring during the developmental stages of tooth formation [4].

Developmental disturbances in the permanent dentition following an injury to the primary dentition were reported in 12%–69% of cases in various clinical studies[11–14]. Possible morphologic variations include; discoloration of the enamel, enamel hypoplasia, odontoma like malformations, crown or root dilacerations, root duplications, and partial or complete cessation of the root formation[12]. The severity of dilaceration depends on various factors like the direction and intensity of the force involved the stage of development of the permanent tooth at the time of trauma, and the relationship of the permanent tooth to the roots of primary teeth[4]. It can also occur because of premature extraction of the deciduous predecessors[8]. It can also originate from nontraumatic causes[3–5]. The frequency of occurrence of crown dilaceration is 3% [9]. It usually occurs because of trauma between 1 and 5 years of age.[3] Bilateral dilaceration had also been reported [10].

The crown of a permanent tooth has to be one half developed at the time of trauma[6]. Deviation of the crown varies according to the location of the tooth. The maxillary incisors show palatal deviation, whereas the lower incisors are inclined labially[6]. The treatment of a dilacerated anterior tooth is challenging for the clinician, because of its difficult position and the abnormality of the root. Treatment often involves surgical removal with subsequent orthodontic methods to either close the space or keep it open until the patient reaches an age when implants or prosthetic treatment can be performed. Both methods have associated problems.

This case report describes a permanent central incisor having a 90 degrees bent at the cementoenamel junction being managed surgically.

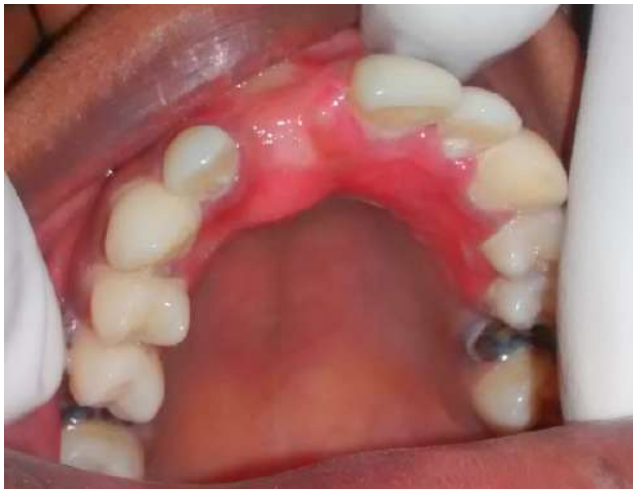


Fig 1: A preoperative photograph. (An intraoral view showing an edentulous region in the right maxillary central incisor region with an elevation in the alveolus in the labial and palatal surface).

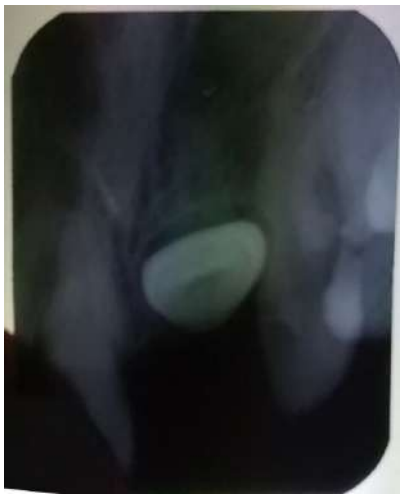


Fig 2: An intraoral periapical radiograph (11 region) revealing an unerupted right maxillary central incisor with crown dilaceration without any periapical radiolucency and a rounded radiolucent area at the point of dilaceration giving a ''bull's-eye'' appearance.

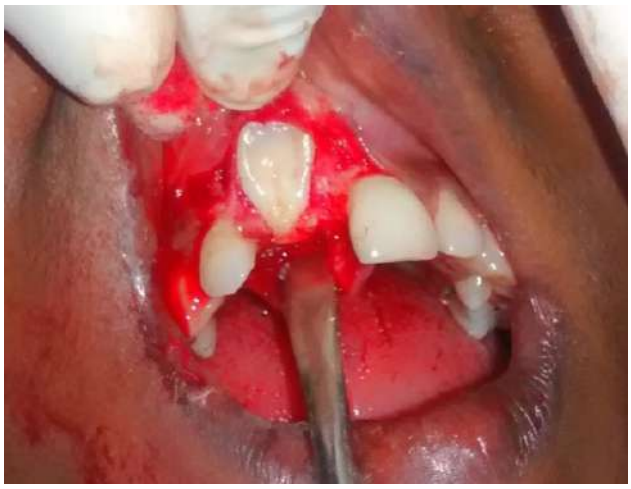


Fig 3: An intraoperative photograph revealing the dilacerated crown portion.

Fig 4: Post operative photograph with the whole tooth intact.

Case report

A 22 year old female patient reported to the department of Oral and Maxillofacial Surgery Govt Dental College Kottayam with a chief complaint of pain in the upper front tooth region since 1 month. History revealed trauma in the anterior tooth region during 4 years of age. Her right maxillary central incisor was avulsed and an RPD space maintainer was advised by a dentist and no further follow up was done for the same. The clinical investigation revealed an edentulous region with mild elevation in the alveolus on the labial side in the region of the incisor (figure1)..

An intraoral periapical radiograph revealed an unerupted right maxillary central incisor with crown dilaceration without any periapical radiolucency and also a rounded radiolucent area at the point of dilaceration giving a "bull's-eye" appearance (figure 2). Based on the clinical and radiographic findings, a diagnosis of an impacted right maxillary central incisor with crown dilaceration was made

The treatment planned was surgical removal of the impacted tooth followed by prosthetic replacement. The patient was issued an informed consent form and agreed to it after being made aware of alternative treatment

options. The surgical site was anesthetized with infiltration using 2% lignocaine with 1:80,000 adrenaline .Using a number 15 Bard Parker blade, a crestal incision followed by a crevicular incision was placed and an envelope flap was raised and the tooth was exposed followed by removal of the dilacerated tooth. (figure 3 and 4). The surgical site was irrigated with saline and closure done with interrupted suture using 3-0 silk. The RPD was then used as a temporary splint.

Discussion

Dilaceration is an abnormal angulation or bend in the root or, less frequently, the crown of a tooth. Although most examples are idiopathic, a number of teeth with dilaceration appear to arise after an injury that displaces the calcified portion of the tooth germ, and the remainder of tooth is formed at an abnormal angle. The damage frequently follows avulsion or intrusion of the overlying primary predecessor, an event that usually occurs before 4 years of age.[15] Histologically, it can be explained by the displacement of enamel epithelium along with the mineralized portion of the tooth in relation to the dental papilla and cervical loops[4].

In the above reported case the trauma reported during the age of 4 years followed by the use of space maintainer could be the probable reason for the dilaceration of the crown of the permanent successor.

The treatment options available are extraction of the dilacerated tooth followed by prosthetic replacement, surgical resection of crown followed by root canal therapy and cast post supported prosthesis[16], surgical exposure followed by orthodontic extrusion and alignment of the tooth[17,18].

The above case was managed by surgical removal of the tooth followed by prosthetic replacement to prevent any midline shift and to prevent the loss of alveolar height in the aesthetically critical region of the maxilla.

Conclusion

Traumatic injury of the primary teeth played a role in the formation of the abnormal tooth morphology in this case. Clinicians should consider the potential prognoses and complications of traumatic injuries to primary teeth and inform the patients and their guardians about the clinical importance of dental management and further periodic follow-up.

References

1. Shafer WG, Hine MK, Levy WM. A textbook of oral pathology. 4th edition. Philadelphia,PA: Saunders; 1983:40.
2. Stewart DJ. Dilacerated unerupted maxillary central incisors. Br Dent J 1978;145:229–33.
3. Andreasen JO, Ravn JJ. Epidemiology of traumatic dental injuries to primary andpermanent teeth in a Danish population sample. Int J Oral Surg 1971;21:235–9.
4. Andreasen JO, Sundstrom B, Ravn JJ. The effect of traumatic injuries to primaryteeth on their permanent successor. I. A clinical and histologic study of 117 injuredpermanent teeth. Scand J Dent Res 1971;79:219–83.
5. Andreasen JO, Ravn JJ. The effect of traumatic injuries to primary teeth on theirpermanent successor. II. A clinical and radiographic follow-up study of 213 teeth.Scand J Dent Res 1971;79. 281–29l.
6. Andreasen JO. The influence of traumatic intrusion of primary teeth on their permanentsuccessors. A radiographic and histological study in monkeys. Int J Oral Surg1976;5:207–19.
7. Da Silva Assunc¸ ~ao LR, Ferelle A, et al. Effects on permanent teeth after luxationinjuries to the primary predecessors: a study in children assisted at an emergencyservice. Dent Traumatol 2009;25:165–70.
8. Matsuoka T, Sobue S, Ooshima T. Crown dilaceration of a first premolar caused byextraction of its deciduous predecessor: a case report. Endod Dent Traumatol 2000;16:91–4.
9. Crescini A, Doldo T. Dilaceration and angulation in upper incisors consequent todental injuries in the primary dentition: orthodontic management. ProgOrthod2002;3:29–41.
10. Rengaswamy V. Bilateral dilaceration of maxillary central incisors. Oral Surg OralMed Oral Pathol Oral RadiolEndod 1979;47:200.
11. Wilson CF. Management of trauma to primary and developing teeth. Dent Clin NorthAm 1995;39:133–67.
12. Selliseth NE. The significance of traumatised primary incisors on the developmentand eruption of permenent teeth. Rep CongrEurOrthodSoc 1970;443–59
13. Ravn JJ. Sequelae of acute mechanical traumata in the primary dentition. A clinicalstudy. ASDC J Dent Child 1968;35:281–9.
14. Andersson L. Epidemiology of traumatic dental injuries. J Endod 2013;39(3 Suppl):S2–5.
15. Angela C. Chi, Douglas D. Damm, Brad W. Neville, Carl M. Allen, Jerry Bouquot. Oral and maxillofacial pathology third edition:89-90
16. Diab M, el Badrawy HE. Intrusion injuries of primary incisors. Part III: effects on thepermanent successors. Quintessence Int 2000;31:377–84.
17. Farronato G, Maspero C, Farronato D. Orthodontic movement of a dilaceratedmaxillary incisor in mixed dentition treatment. Dent Traumatol 2009;25:451–6.
18. McNamara T, Wolfe SN, McNamara CM. Orthodontic management of a dilacerated maxillary central incisor with unusual sequelae. J ClinOrthod 1998;32:293–7.

'Peri-apical cyst associated with deciduous mandibular first molar - a case report'

Digesh Balachandran [a] , Rahul K. [b] , Deepak D. Kammath [c]

Abstract

Periapical cysts though rare in primary dentition, can occur in young children associated with a carious exposed primary tooth. Usually periapical cysts arise from the apex of primary mandibular molars. Enucleation of the periapical cyst along with the extraction of the involved teeth is the recommended treatment. The present case report describes the successful management of a periapical cyst surgically in a 3 1/2 year old boy.

Key words: Periapical cyst, enucleation.

Address for correspondence:

[a] Assistant Professor, [b] Senior Resident, Department of Pedodontics, Govt Dental College, Trivandrum; [c] Senior Lecturer, Department of Pedodontics, Govt. Dental College, Kottayam.

Introduction:

Periapical cysts are relatively rare in deciduous dentition because of the distinct biological cycle of the primary teeth[1]. The frequency of radicular cysts in permanent dentition is about 7-54%, while in primary dentition it is approximately 0.5-3.3% of the total number of radicular cysts in both the primary and permanent dentition[2].

Case report:

A 3 1/2 year healthy boy was referred to the Department of Pedodontics, Government Dental College, Thiruvananthapuram with the complaint of a painless swelling on the right side of lower jaw. He had a history of recurrent episodes of pain in relation to the decayed right lower back tooth for approximately a month. Clinically there was a swelling in relation to 84 which was obliterating the vestibular sulcus. Periapical radiograph could not be obtained as the child did not cooperate in placing the film in the lingual vestibule. Mandibular occlusal view in the region revealed buccal cortical plate expansion with almost no involvement of the lingual cortical plate presenting a unilocular lesion.

A provisional diagnosis of peri-apical cyst was made from the history, clinical and radiographic picture. It was decided to extract 84 along with enucleation of the cystic lesion. Anesthesia was attained through the inferior alveolar nerve block and local infiltration in relation to 84 region. 84 was extracted and access to the cyst was made through the extracted wound. Lining of the cyst was curetted and the bony cavity irrigated with normal saline. Extracted wound was sutured and the lining sent for hsistopathological examination.

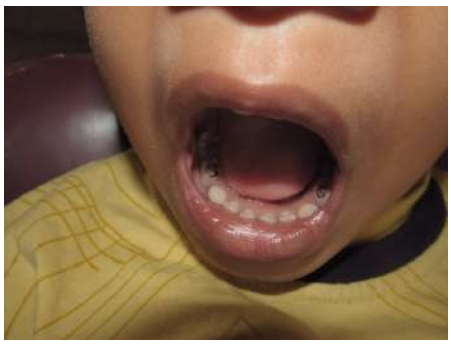
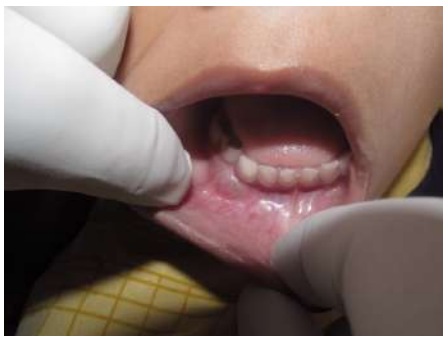
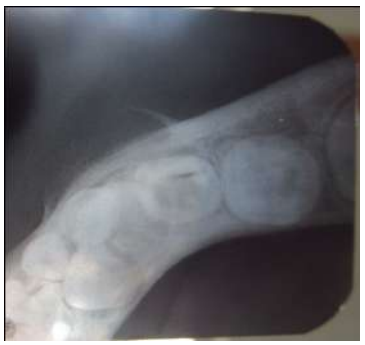
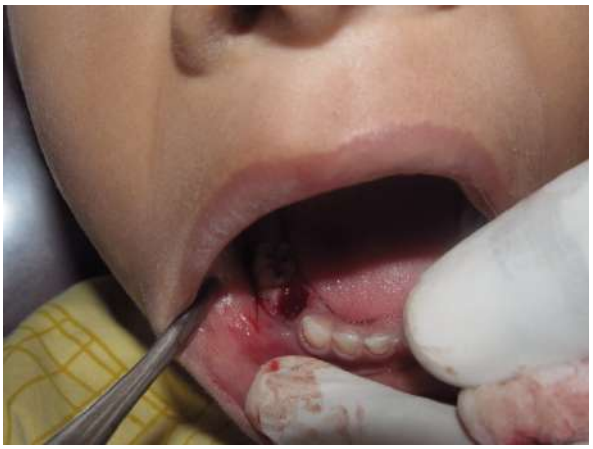
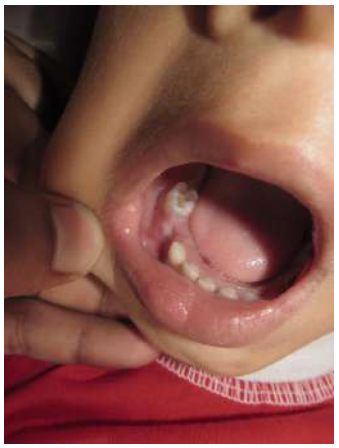
Histopathologically fragments of moderately collagenous connective tissue capsule seen with no epithelium. Focal collection of chronic inflammatory cell infiltrate chiefly lymphocytes; therefore suggestive of an inflamed cystic wall. The patient was recalled after one week for suture removal. The swelling decreased considerably within one week.

Discussion:

The present case of radicular cyst was in relation to the deciduous right mandibular first molar, which was in favour of earlier case studies[3]. Caries are the most common etiologic factorassociated with radicular cysts in the primary dentition[4]. Peri-apical cyst formation in children may cause bony expansion and resorption, delayed eruption, malposition, enamel defects or damaging of the developing permanent successors[3]. Radicular cysts arise from the epithelial remnants in the periodontal ligament as a result of inflammation[5].

Usually periapical radiolucencies related to primary teeth are neglected and in many cases as they resolve after tooth extraction4. They are comparatively rare in primary dentition as the pulp and inter-radicular infections in primary teeth have a tendency to drain more than permanent teeth[5].

Most of the authors prefer enucleation of the cyst along with the removal of involved tooth as the suitable treatment1. Marsupialisation of the cystic lesion and using a resin appliance with projection for

decompressing the lesion is a more conservative treatment modality[4]. Early diagnosis of such cystic lesions are very important and regular clinical and radiographic follow up after surgical removal are mandatory.

Conclusion:

Peri-apical cysts associated with primary teeth are relatively a rare clinical entity which can often be missed and can potentially affect the permanent successor tooth if it is not dealt with appropriately. Prompt diagnosis and removal of the cystic lesion along with the involved tooth often resolves the clinical progression of these rare cystic lesions.

References

1. Toomarian L, Moshref M, Mirkarimi M, Lofti A, Beheshti M. Radicular cyst associated with a primary first molar: A case report. Journal of Dentistry, Tehran University of Medical Sciences, Tehran, Iran 2011; 8(4).
2. Neville B, Damm D, Allen C, Bouquot J. Oral and Maxillofacial Pathology. 3rd ed. Elsevier; 2008. Chapter 3..
3. Mass E, Kaplan I, Hirshberg A. A clinical and histopathological study of radicular cysts associated with primary molars. J Oral Pathol Med 1995 Nov;24(10):458-461.
4. Delbem AC, Cunha RF. Conservative treatment of a radicular cyst in a 5-year-old child: a case report. Int J Paediatr Dent 2003 Nov;13(6):447-50.
5. Ramakrishna Y, Verma D. Radicular cyst associated with a deciduous molar: A case report with unusual clinical presentation. J Indian Soc Pedod Prev Dent 2006 Sep;24(3):158-60.

'Hemifacial microsomia - a case report'

Digesh Balachandran [a] , Deepak D. Kammath [b] , Aby J. Mathew [c]

Abstract

Unlike anomalies like Cleft lip and palate, several other conditions are often undiagnosed, especially if they are present in less severe forms. This case report is about Hemifacial microsomia, a rare congenitally occurring condition, diagnosed as a result of a dental consultation and identification of associated anomalies in a primary school going child, whose parents were totally unaware about the condition .It also discusses the management of such anomalies.

Address for correspondence:

[a] Assistant Professor, [b] Senior Lecturer, Department of Pedodontics, Govt Dental College, Kottayam; [c] Senior Lecturer, Department of Pedodontics, Govt. Dental College, Kottayam.

Hemifacial microsomia is a congenital malformation where deficiency in the amount of hard and soft tissue on one side of the face is seen. This Case report is about a 9 year old boy who had reported to the Department of pedodontics, Govt. Dental College, Trivandrum wanting correction of his malposed upper anterior teeth. Facial anomaly was evident on clinical examination itself. The parents of the child were unaware of the condition of the child and they are thinking that the child was having a dental irregularity only and which could be corrected by braces or any similar appliances

Clinical examination

Bilaterally asymmetry of the face was evident on initial clinical examination itself. Deviation of lower jaw towards left side shifting the midline of mandible also seen The chin was deviated to the left side. The corner of the mouth was deviated to the left side. Left side of the face appeared to be flat. On palpation the affected side was found to be having, smaller sized coronoid and condyles compared to unaffected side. Fullness of cheek was absent on left side and Muscles of Mastication appeared to be under developed . Other facial structures like eyes, nose, lips, neck movements and skin appeared unaffected. Intra oral examination showed high palatal vault. Maxillary left deciduous second molar was appeared to be congenitally missing and the first primary molar was smaller in size compared to unaffected side. Primary canines & Lateral incisors and permanent incisors appeared unaffected and similar in appearance Mesiodistal dimensions of mandibular left posterior teeth were similar as compared to unaffected right side. However there appear to be a delay in eruption of the first permanent molar on the affected side.

The medical examination of the child also revealed a hearing impairment of the affected side.

Radiographic examination

It was found that the maxillary first and second permanent molars & the second premolar were missing and the developing first premolar appeared to be malformed. Mandibular teeth did not show any differences between affected and unaffected sides. The third molar tooth buds appeared yet to be formed in all quadrants.

Radiographic examination in case of Hemifacial microsomia is of limited value because of superimposition of normal and abnormal bony structures. To understand the skeletal and soft-tissue findings of a patient with Hemifacial microsomia , modern imaging methods like three dimensional computerized tomography may be required.

Discussion

Hemifacial microsomia is primarily a syndrome of the first branchial arch, involving underdevelopment of the temporomandibular joint, mandibular ramus, masticatory muscles and the ear. The affected ear may have an external soft-tissue malformation in addition to being lower set than on the contra lateral side. Hearing loss may result from underdevelopment of the osseous components of the auditory system and a diminished or absent external auditory meatus. Occasionally, second branchial arch defects involving the facial nerve and facial muscles coexist with Hemifacial microsomia.

The general presentations of hemifacial microsomia — such as unilateral microtia, macrostomia, and hypoplasia of the mandibular ramus and condyle are well known, the effects on the teeth are not well-documented

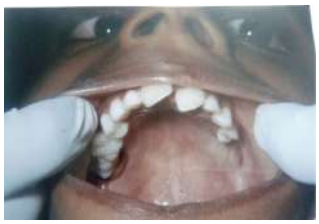


Fig 1: Clinical examination showing restriction of right eye in superior gaze

Fig 2: Profile view showing lack of fullness of cheek on the left side (affected side)

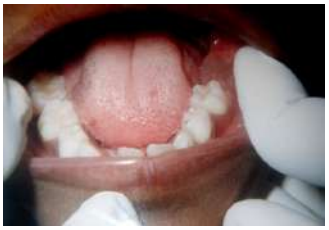


Fig 3: Mandibular arch showing delayed eruption of first permanent molar

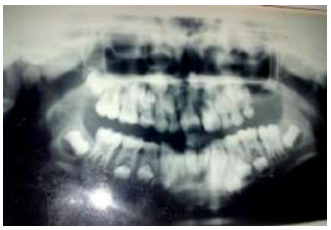


Fig 4: OPG shows missing maxillary first and second permanent molars & the second premolar

and studies have shown that the mesiodistal dimensions of the primary molar and permanent molar teeth on the affected side in hemifacial microsomia were significantly smaller or even missing compared. The most posterior tooth in each arch being the most severely affected and no effect being seen in the canines and the incisors. The dental lamina in hemifacial microsomia is affected, where pathogenesis involves an abnormality of the neural crest.

The differential diagnosis would include Pierre Robin syndrome, Treacher Collin Syndrome, ParryRomberg syndrome, Down's syndrome, hypoplasia of condyle on the left side and congenital unilateral ankylosis on the left side .

Treatment

Treatment is usually long term and may includes surgical and non surgical management. The aim of surgeries is to improve facial symmetry and functioning which may have been affected due to growth and development issues.

0-1 year : Vital functions - breathing, eating and sleeping must be assessed immediately, attention is paid to feeding to ensure that the infant can thrive.

2-5 years : Mildly affected children require no treatment during this time. In those cases where the mandible is severely underdeveloped, surgical reconstruction may be required using a rib bone graft or lengthening of the body of the mandible with bone distraction devices. Occlusal anomalies may require suitable corrections concomitantly .

6-9 Years: Cosmetic Reconstruction of the external ear may be required in patients who are not severely affected. Severe facial asymmetry may requires reconstruction of the jaw and cheekbone contour, delaying ear reconstruction.

10-12 Years : This is crucial time in the treatment program as the appearance becomes more and more of a concern for both the parent and the child. To create fullness of the cheek Allograftic procedures may be required like transferring soft tissue and its blood supply from another part of the body to the affected region and intraoral or extraoral appliances for correction of malocclusion

Teenage Years : Orthognathic surgery may be performed on those patients whose mild condition did not require it in early childhood. Mandibular growth that occurs in adolescence may require those severely affected to undergo further surgery. There may be also be a requirement of both pre- and post surgical orthodontic treatment .

Since this is not a progressive condition unlike hemifacial atrophy, timely and appropriate dental and medical interventions provide a stable and satisfactory treatment result and affected individuals mostly lead uneventful lives after the treatment.

Conclusion

Early diagnosis of this rare condition is required as treatment for severely affected individuals may require to be started at a very early age and may last till the early adulthood and will include dental and medical interventions. In the particular case reported here the parents were unaware of the condition of the child and hearing impairment was diagnosed only on a detailed medical examination which was advised to the parents after the dental consultation.

References

1. Rita SN, Sadat SM, Sitan KN, Khan MR. Management of Hemifacial microsomia - A review. Med Today 2011;23:106-8
2. Hemifacial microsomia: A case report- R Kapur, R Kapur, S Sheikh, S Jindal, S Kulkarni-JISPPD, Year : 2008
3. Munjal D. Hemifacial microsomia: A case report and review of literature. J Indian Assoc Oral Maxillofac Radiol 2006;18:64-9.
4. Kalsotra P, Chowdhary A, Bhagat DR, Parihar SS,Prabhakar R (2006) Craniofacial microsomia. J K Sci 8, 168-170.
5. Monahan R, Seder K, Patel P, Alder M, Grud S, O'Gara M. Hemifacial microsomia: Etiology, diagnosis and treatment. Am Dent Assoc 2001;132:1402-8.
6. Raymond WS, Angelisa MP, Samson L, Michael LC. Hemifacial microsmia in pediatric patients: Asymmetric abnormal development of the first and second branchial arche. AJR 2002;178:1523-30.