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Amelogenesis Imperfecta of the Combination Type–Hypoplastic and Hypocalcified: A Rare Case Report

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ABSTRACT:

'Amelogenesis imperfecta' is the term for a collection of developmental disorders that have a genetic basis and impact the structure and clinical appearance of enamel in all or almost all the teeth in a roughly equal way. Amelogenesis imperfecta is a significant issue that lowers the quality of life associated with dental health and results in some physiological issues. The patients typically present significant challenges for clinicians when it comes to whole dentition rehabilitation. According to reports, skeletal and dental abnormalities are linked to the majority of cases of this condition, which causes extreme sensitivity of the tooth tissues. This case report describes a rare instance of the combination type of amelogenesis imperfecta of a female who was 19 years old. This condition affected her self-confidence since childhood and finally wants cosmetic rehabilitation for the same.

Introduction

Enamel is thought to be the toughest and most mineralized tissue in the human body, with hydroxyapatite crystals encasing 85% of its volume.[1, 2] The direction and orientation of enamel rods, the compositional microstructure, and the structural arrangement of the mineral constituent of the tissue are all directly related to the physiological morphology.[3, 4] Enamel undergoes transformation throughout the process of organogeny, changing from a soft, pliable structure to a hard, calcified form that is protein-free.[5] The ultimate structural design is a reflection of the different cellular and developmental processes that take place throughout morphogenesis.[4] Amelogenesis imperfecta is caused by any deviation from normal organogenesis and might show up in the mouth cavity. Amelogenesis imperfecta is a genetic condition that affects a person's structure, appearance, and clinical status. It can be demoralizing for the patient and their sense of self. These are exceptional cases that happen rarely. Because enamel is hard, malleable, and chips readily, it must be handled with utmost care,

attention, and precision.

Amelogenesis imperfecta is characterized by extensive abnormalities in enamel development in both primary and permanent dentition. There are four types of inherited tooth malformations: X-linked, autosomal dominant, autosomal recessive, and sporadic. The

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dysfunction of enamel-forming proteins is specifically linked to mutation or altered expression of the enamelin (ENAM), amelogenin (AMEL), matrixmetalloproteinaise-20 (MMP20), kallikrein-4 (KLK4), and FAM83H genes.[6] There hasn't been any information of a connectionto a systematic or universal disorder.

There are four forms of amelogenesis imperfecta in the clinical manifestation.[7] Type I -which is typified by a hypoplastic structure and less enamel, is the most prevalent phenotype. Reduced enamel thickness, a rough surface, and different expansions of flaws are visible in the teeth.[8] Because of faulty protein maturation inside the enamel matrix. Type II, also known as hypomaturation, exhibits speckled and softer enamel. Furthermore, there is enamel chipping from the dentin.[6] Enamel thickness in amelogenesis imperfecta type II is normal. The ameloblasts secrete normally, however throughout the maturation phase, the secreted enamel matrix proteins are not normally reabsorbable. As a result, the enamel still contains a significant amount of organic stuff.[9] Type III(hypocalcification) is associated with defects in calcification and appears in enamel with normal thickness at the time of eruption. Because of the poor mineralization, the enamel rapidly wears down and X-rays show less opacity. Type IV manifests as a mixed appearance of hypoplasticity–hypomaturation combined with taurodontism.[6,10]

There have been reports of plaque buildup and unattractive appearance due to the structure of enamel hypersensitivity.[11] Multidisciplinary patient care is advised toavoid dental cavities, gingival irritation, open bites, and loss of vertical dimension. For effective dental rehabilitation, conservative, prosthetic, and orthodontic treatment are especially important. Different treatment approaches have been outlined based on the severity of the deformity, the patient's age, and their socioeconomic status.[12] While composite restorations, stainless steel crowns, and strip crowns are frequently used in primary dentition, maintaining the dentition during growth presents a challenge in adolescents with mixed and permanent dentition.[13] Adults are better off with ceramic crowns and veneers, but adolescents can benefit from high-quality restorations made possible by CAD/CAM composites. Less chair time and the potential for intraoral repairs in the event of material fractures are the benefits of this strategy. In addition to the dental troubles brought on by genetic enamel abnormalities, low aesthetics has been linked to lower oral health-related quality of life and issues with self- confidence.[14,15]

Case Report

A 19-year-old Indian, female patient, reported to the Department of Oral Medicine and Radiology with a chief complaint of yellowish discoloration, edentulous spaces in the posterior teeth region, and poor visual appearance of herteeth. This condition existed since she got her first deciduous tooth. The patient requested aesthetic rehabilitation of her mouth. The patient was a student and unmarried. She was accompanied to the department with her mother, theydiscussed the purpose of requesting for rehabilitation as her daughter had to get married and wanted her teeth to have a normal visual appearance.

The patient also gives a history of itching in the gums in the posterior teeth regionsince one month. Amongst the family members, none of the siblings or relatives presented with a similar dental condition. The mother gave history of deciduous teeth looking similar and only a few deciduous teeth erupted in the oral cavity in the anterior region with no history of dental extractions, similar condition to the permanent dentition. No history of tobacco or any other deleterious habits. No relevant medical history was associated. No pain was associated while chewing or at rest. The patient was conscious, well-oriented, and co-operative. An extraoral

examination revealed no temporomandibular joint issues and normal mouth opening. The patient had no co-morbidities. No facial asymmetry, swellings, or palpable lymph nodes. The hair and skin of the patient appeared normal. Intraoral examination revealed presence of fourteen teeth clinically. The maxillashows presence of 11, 13, 55, 21, 22, and 23 (Figure 1). The mandible shows presence of 31, 32, 33, 35, 41, 42, 43, and 85 (Figure 2).

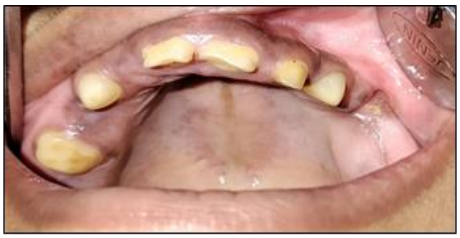

Figure 1: Intraoral photograph of the maxilla showing

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presence of 11, 13, 55,
21, 22, and 23.

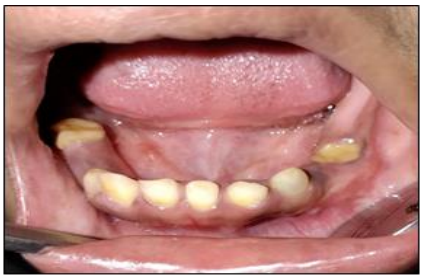

Figure 2: Intraoral photograph of the mandible showing presence of 31, 32, 33,
35, 41, 42, 43, and 85.

The maxillary and mandibular anterior teeth show yellowish discoloration.

Clinically, the maxillary anterior teeth appear intruded, and square in shape. Theshape of the maxillary anterior teeth appeared abnormal, with more mesio-distal width in the cervical aspect instead of the incisal aspect. Clinically, it appeared that the region of the enamel was lost, giving the teeth a yellowish appearance. Evident spacing is seen between all teeth.

On palpation, the surface appeared rough in texture, with minor pitting seen on the exposed tooth surfaces, with the buccal and labial surfaces showing the most pitting. No tenderness on palpation when tested with a probe, horizontally or vertically. Generalized blunting of the cusps noted. The periodontal status of the mouth revealed, the generalized color of the gingiva appearing to be pale pink, along with the absence of stippling or oedematous gingiva. Melanin pigmentation was seen on the gingiva, hard palate, and tongue. The gingiva appeared to be hyperplastic in the areas where the tooth is present, surrounding the cement-enamel junction of the tooth. In the areas where the tooth is not there, the gingiva appeared normal. The lips were competent and functional. The buccal mucosa, maxillary or mandibular muco-buccal fold, hard palate, soft palate, floor of the mouth, and tongue showed no abnormality. Based on the clinical findings, the provisional diagnosis was amelogenesis imperfecta – combination type of hypoplastic and hypocalcified, along with hereditary gingival fibromatosis. Differential diagnosis: amelogenesis imperfecta, oligodontia, non-acquired dental fluorosis, dentinogenesis imperfecta, molar incisor hypomineralization, Based on the clinical findings, for radiographic investigations, intraoral periapical radiographs of maxillary and mandibular anterior teeth, along with an orthopantomogram were advised for the final diagnosis. For further treatment and management, a cone-beam computed tomography was taken.

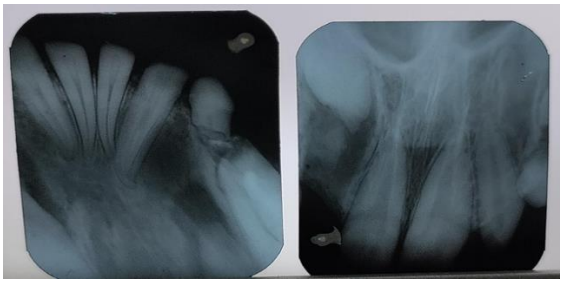

Figure 3: Two IOPAs showing maxillary and mandibular anterior teeth

An intraoral periapical radiograph of the upper and lower anterior of the mouth was taken showing 11, 21, 31,41,42,43 completely and 12, 23, 44 partially.

(Figure 3)

Crowns in the maxillary teeth show the presence of irregular radiolucency in the enamel. The crowns show radiopaque dentin and radiolucent pulp chambers. 11, 21, 22 and 43 show loss of tooth structure in the region of enamel. There is

absence of enamel in the incisal and lateral aspect of maxillary anterior teeth. The roots show presence of periodontal ligament space and lamina dura in the cervical, middle and apical third. Presence of periapical radiopacity in 42 suggestive of condensing osteitis. It also shows dilaceration of the root. 43 shows a horizontal line in the middle third of the root suggestive of a transverse tooth fracture. Advised radiograph in a different angle for ruling out any artifact. All rootsshow tapering and they are shortened. A trabaculated pattern is seen in the surrounding bone.

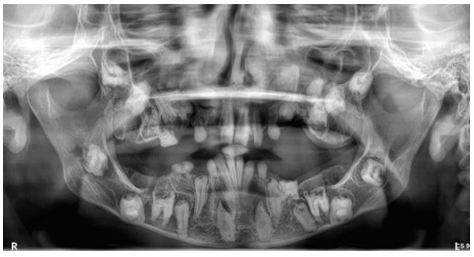

Figure 4: Orthopantomogram showing maxilla, mandible and associateddentoalveolar structures**.**

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This is an orthopantomogram showing dentoalveolar and associated

maxillofacial structures, taken in Kodak CS9600 machine under 73kvp & 8mA current. The OPG shows all permanent teeth. The permanent teeth impacted, which are impacted are 12, 14, 15, 17, 24, 25, 26, 27, 34, 36, 37. The deciduous teeth which are impacted are 53, 54, 43, 44, 33, 35, 43, 44. The inferior alveolar nerve canal is depressed on both the sides. (Figure 4).

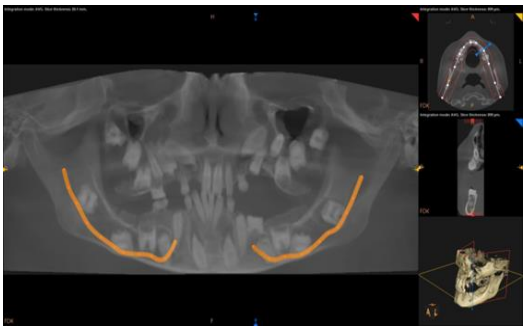

Figure 5: CBCT - panoramic section showing multiple permanent anddeciduous impacted teeth.

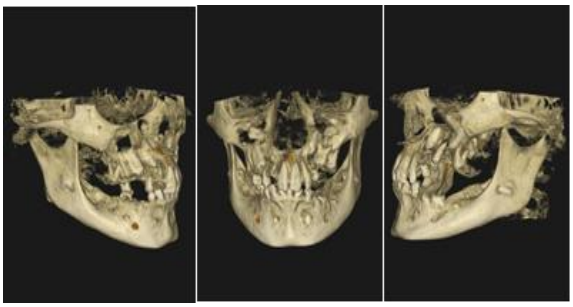

Figure 6: CBCT 3D reconstructed image of the hard tissues.

The CBCT shows similar findings like the OPG along with the tracing of inferior alveolar nerve canal and nasopalatine canal. The CBCT was taken to check the root canal morphologies for endodontic and restorative treatments, location of the impactions and status for orthodontic corrections along with implant planning measurements for post extraction full mouth rehabilitation. Hence, the final dental diagnosis is amelogenesis imperfecta of the combination of the hypoplastic and hypocalcified type. The final periodontal diagnosis is

hereditary gingival fibromatosis.

Discussion

'Amelogenesis imperfecta' is a developmental, often inherited disorder, affecting dental enamel. It is characterized by a variety of phenotypic entities and typically manifests without systemic characteristics.[4] The majority of affected individuals will exhibit a mixed phenotype, which is enamel hypoplasia (enamel that is thin and seems to be correctly mineralized), hypomineralization (which is separated into hypomaturation and hypocalcification), or both. The trait of ameloegensis imperfecta can be transmitted by an autosomal-dominant, autosomal-recessive, or X-linked mode of inheritance.[16,17] Amelogenesis imperfecta affects both the deciduous and permanent dentition, however it affects permanent teeth more frequently than primary teeth, and it affects incisor teeth and first molars in both the upper and lower jaws more frequently. Numerous non-enamel anomalies, such as delayed tooth eruption, congenitally missing teeth, anterior open bite, taurodontism, pulpal calcifications, dentin dysplasias, hypercementosis, root malformations, and root resorption, have been linked to amelogenesis imperfecta, according to numerous studies on the subject. Additionally, malocclusion and gingivitis have been linked toamelogenesis imperfecta.

Witkop et al, first observed that only males were impacted by hypomaturationamelogenesis imperfecta in two families. The adjective "recessive" in his description of "X-linked recessive amelogenesis imperfecta" may have been used because it was believed that the females were unaffected. He was then able to re-examine the females of one of the two original families under suitable lighting conditions, prompted by the presence of a third proband, and found that there were "alternating vertical bands of mottled white hypomature enamel separated by normal-appearing enamel."[7]

The examples described by McLarty et al.[19] and Haug and Ferguson[20] do havesome resemblance to this look, which is an apparent hypomineralization

accompanied by vertical hypoplasia of the enamel. Amelogenesis imperfecta was classified into hypoplastic and hypocalcified varieties by Weinmann et al.[7];this division has been maintained in the majority of subsequent classifications. With its focus on perceived phenotype, this divergence may be more grounded in

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clinical opinion than in histology facts. It could also explain some of the

terminology variations that appear in the literature about X-linked amelogenesisimperfecta.

Until now, linkage studies[19] have assigned the disease to the distal short arm of the X chromosome. However, with additional recombinant DNA research, we will be able to differentiate between three forms of the disease: an apparent simple hypoplastic form, a hypoplastic and hypomineralized form, and a pure hypomaturation type.[20,21] Amelogenesis imperfecta may be caused by a few of the genes encoding the particular enamel proteins. This theory has been validated by mutational investigations conducted within the examined families. Amelogenin mRNAs have been recently demonstrated to express at a low level in odontoblasts, but the tooth-specific amelogenin gene is expressed in pre-ameloblasts, ameloblasts, and the remnants of the epithelial root sheath. There are now 14 amelogenesis imperfecta mutations linked to AMELX.[22]

The enamelin (ENAM) gene is unique to teeth and is primarily expressed by the enamel organ as well as, to a lesser extent, by odontoblasts. The chromosome 4 (4q13.3) is where the human ENAM gene is located. A 4Mb area on 4q21 has been associated with autosomal-dominant amelogenesis imperfecta (ADAI), one autosomal-inherited variant of amelogeneis imperfecta. ENAM mutations may have dose-dependent enamel phenotypes, where localized enamel pitting segregates as a dominant trait and generalized hypoplastic amelogenesis imperfecta segregates as a recessive trait.[23] Ameloblasts express the ameloblastin (AMBN) gene at high levels, but odontoblasts and pre-odontoblasts express it at low levels. Hertwig's epithelial root sheath and odontogenic tumors, like ameloblastomas, also exhibit intermediate expression of this gene.[24] The ultimate classification will be based on the genetic mutation and the resulting biochemical abnormalities in each family, much like in other genetic disorders. Several researchers have proposed a phenotypic and pedigree-based classification scheme that incorporates molecular genetics, biochemical techniques, and scanning electron microscopy. Histological analysis revealed extremely thin enamel in a ground section of the affected teeth, which was made up of laminations of imperfectly organized enamel prisms.[25] The autosomal recessive rough hypoplastic amelogenesis imperfecta case study including the excised deciduous teeth revealed an exposed outer enamel surface with irregularly formed globular protrusions. The enamel areas at the cervical region of the crown had a pattern of parallel, wavy ridges. The mottled and fibrillar texture of the cementum area and its tendency to overlap the ridged coronal structure along the cervical line made it easy to identify from the more coronal region. There was some unusual prism development and a high organic content in the enamel. Because the enamel matrix had a more uniform look than the dentin due to its array of collagen fibrils, the dentin-enamel junction was well-defined and easily recognized.[26]

According to Winter et al.'s description, the histology of autosomal dominant hypomaturation-hypoplasia type amelogenesis imperfecta with taurodontism consisted of regions with severe hypomineralization and pore volumes ranging from 1 to 25%. They described the enamel as having a typical prismatic structure, but with a significant amount of organic substance added after calcification and sporadic bands of globular flaws. There were also reports of dentin defects, including dilatations, cellular inclusions, a rise in intertubular dentin, and a decrease in tubules. In the radicular dentin, each of these observations was more pronounced. The pulp was regular but larger than usual.[27]

Dental professionals have a variety of clinical challenges in managing restorative cases due to the diverse etiology of amelogenesis imperfecta. Since these patients' function and appearance are both impaired, full coverage crowns, direct and indirect veneers, and bonded esthetic restorations are typically used in their treatment, depending on the patient's age and the state of each individual tooth. Rapid attrition results from the extremely thin or severely deficient enamel. As soon as it is feasible, these variations must have complete coverage. Delays in treatment result in a reduction in the length of the useful crown. In patients with insufficient crown lengths, complete dentures (or overdentures in some situations) frequently become the only satisfactory approach.

The cosmetic look is the primary objective, while the other varieties of amelogenesis imperfecta show slower

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rates of tooth loss. Sometimes veneers are not properly enameled, which prevents a long-lasting restoration. This shortcoming is frequently addressed by combining dentinal adhesives with glass ionomer cements. Restoring the smile in individuals with amelogenesis imperfecta deals with lotof accuracy, patience and skill along with comprehensive collaboration with various sectors of dental treatment. Multidisciplinary approach of treatment planning includes restoration of function, aesthetics and vertical dimension. The patient's age, dental hygiene, quality of life, periodontal issues, internal tooth architecture, remaining tooth structure, and orthodontic concerns are just a few of the many variables that must be taken into account when planning a course of therapy.[27,28]The treatment modalities may change depending on the various factors. Because of the structure of teeth, treating patients with amelogenesis imperfecta by root canal is thought to be challenging. Pulp space therapies coupled with restorative therapy modalities should always be evaluated. The teeth appear normal and the pulp does not become non-vital due to excessive dentin formation, despite the fact that the majority of the teeth affected by amelogenesis imperfecta have radiographic obliteration. Endodontic therapy is typically seen as required for patients with amelogenesis imperfecta. In these patients, elective endodontic therapy may result in higher long-term success rates and better treatment outcomes. For such patients, full coverage post-endodontic restorations are the ideal option since they preserve and repair the dental tissues from future damage.

Conclusion

Amelogenesis imperfecta patients present a continuing challenge to the clinician: complete reintegration necessitates extreme caution, accuracy, and a multidisciplinary approach with active collaboration from several dental specialties. The goal of the interdisciplinary approach to treating these disorders should be to preserve the patient's bodily, functional, and aesthetic well-being. In order to provide early intervention and balance, the decision between early intervention and the long-term life of the restorations, the dentist must diagnosethe issue as soon as feasible. Dentists should act to alleviate these patients' suffering while taking into account the societal ramifications for them. Therefore, the goal of this article is to increase the clinician's understanding of the clinical diagnosis and necessary action for this kind of illness.

References

1. Robinson C, Briggs HD, Atkinson PJ, Weatherell JA. Matrix and mineral changes in developing enamel. J Dent Res. 1979;58:871–82.
2. Simmer JP, Fincham AG. Molecular mechanisms of dental enamel formation. Crit Rev Oral Biol Med. 1995;6:84–108
3. Mahoney EK, Rohanizadeh R, Smail FSM, Kilpatrick NM, Swain MV. Mechanical properties and microstructure of hypomineralized enamel of permanent teeth. Biomaterials. 2003;25:5091–100.
4. Chaudhary M, Dixit S, Singh A, Kunte S. Amelogenesis imperfecta: report of a case and review of literature. J Oral Maxillofac Pathol. 2009;13(2):70–7.
5. Paine ML, White SN, Luo W, Fong H, Sarikaya M, Snead ML. Regulated expression dictates enamel structure and tooth function (review). Matrix Biol. 2001;20:273–92
6. Gadhia, K.; McDonald, S.; Arkutu, N.; Malik, K. Amelogenesis imperfecta: An introduction. *Br. Dent. J.* 2012, *212*, 377–379.
7. Witkop, C.J., Jr. Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: Problems in classification. *J. Oral Pathol.* 1988, *17*, 547–553.
8. Toupenay, S.; Fournier, B.P.; Maniere, M.C.; Ifi-Naulin, C.; Berdal, A.; de La Dure-Molla, M. Amelogenesis imperfecta: Therapeutic strategy from primary to permanent dentition across case reports. *BMC Oral Health* 2018, *18*, 108.
9. Crawford, P.J.; Aldred, M.; Bloch-Zupan, A. Amelogenesis imperfecta. *Orphanet J. Rare Dis.* 2007, *2*, 17.
10. Ohrvik, H.G.; Hjortsjo, C. Retrospective study of patients with amelogenesis imperfecta treated with different bonded restoration techniques. *Clin. Exp. Dent. Res.* 2020, *6*, 16–23.
11. Quandalle, C.; Boillot, A.; Fournier, B.; Garrec, P.; De La Dure-Molla, M.; Kerner, S. Gingival inflammation, enamel defects, and tooth sensitivity in children with amelogenesis imperfecta: A case-control study. *J. Appl. Oral Sci.* 2020, *28*, e20200170.
12. Ayers, K.M.; Drummond, B.K.; Harding, W.J.;

Journal of Chemical Health Risks
www.jchr.org
JCHR (2024) 14(3), 2537-2543 | ISSN:2251-6727

Salis, S.G.; Liston, P.N. Amelogenesis imperfecta--multidisciplinary management from eruption to adulthood. Review and case report. *N. Z. Dent. J.* 2004, *100*, 101–104.

13. American Academy on Pediatric Dentistry Council on Clinical Affairs. Guideline on oral heath care/dental management of heritable dental development anomalies. *Pediatr. Dent.* 2008, *30*, 196–201.
14. Visram, S.; McKaig, S. Amelogenesis imperfecta—Clinical presentation and management: A case report. *Dent. Update* 2006, *33*, 612–616.
15. Robinson, F.G.; Haubenreich, J.E. Oral rehabilitation of a young adult with hypoplastic amelogenesis imperfecta: A clinical report. *J. Prosthet. Dent.* 2006, *95*, 10–13.
16. Ooya K, Nalbandian J, Noikura T. Autosomal recessive rough hypoplastic amelogenesis imperfecta. A case report with clinical, light microscopic, radiographic and electron microscopic observations Oral Surg Oral Med Oral Pathol. 1988;65:449–58
17. Backman B, Holmgren G. Amelogenesis imperfecta: a genetic study Hum Hered. 1988;38:189–206
18. McLarty EL, Giansanti JS, Hibbard ED. X-linked hypomaturation type of amelogenesis imperfecta exhibiting lionization in females Oral Surg Oral Med Oral Pathol. 1973;36:678–85
19. Lagerstrom M, Dahl N, Iselius L, Backman B, Pettersson U. Mapping of the gene for X-linked amelogenesis imperfecta by linkage analysis Am J Hum Genet. 1990;46:120–5
20. Witkop CJ. Hereditary defects in enamel and dentin Acta Genet Med Gemollol (Roma). 1957;7:236–9
21. Witkop CJ Jr. Partial expression of sex-linked amelogenesis imperfecta in females compatible with the Lyon hypothesis Oral Surg Oral Med Oral Pathol. 1967;23:174–82
22. Kindelan S, Brook A, Gangemi L, Lench N, Wong F, Fearne J, et al Detection of a novel mutation in X-linked amelogenesis imperfecta J Dent Res. 2000;79:1978–82
23. Ozdemir D, Hart PS, Firatli E, Aren G, Ryu OH, Hart TC. Phenotype ofENAM mutations is dosage-dependent J Dent Res. 2005;84:1036–41
24. Toyosawa S, Fujiwara T, Shintani S, Sato A, Ogawa Y, Sobue S, et al Cloning and characterization of the human ameloblastin gene Gene. 2000;256:1–11
25. Ishikawa G, Waldron CA. Amelogenesis imperfecta Color atlas of Oral Pathology. 19991st Indian ed:15
26. Crawford PJ, Aldred MJ. Amelogenesis imperfecta: autosomal dominant hypomaturation – hypoplasia type with taurodontism Br Dent J. 1988;164:71–3
27. Sari T, Usumez A. Restoring function and esthetics in a patient withamelogenesis imperfecta: a clinical report. J Prosthet Dent. 2003b;90(6):522–5.
28. Shetty N, Joesph M, Basnet P, Dixit S. An integrated treatment approach: a case report for dentinogenesis imperfect Type II. Kathmandu Univ MedJ. 2007;5(2):230–3.