

Histopathological Features of Shell Teeth: A Case Report

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ABSTRACT

Shell Teeth are a type of radiographic appearance of teeth seen in Dentinogenesis Imperfecta type II (DGI II). Radiographically, they are characterised by an enlarged pulp chamber enclosed by a thin layer of dentin. The clinical appearance of these teeth ranges from yellow to bluish-grey in colour. The histological features of shell teeth have not been updated in the literature, unlike clinical and radiographic appearance. These features are not available in the literature compared to other dental anomalies. Here, the authors present a case of an 18-year-old female who came with the complaint of poor aesthetic dentition. The dentition aligned with the clinical and radiographic features of shell teeth. Histopathological features of the present case included an irregular arrangement of the dentinal matrix and a few cellular inclusions. The final diagnosis was given as DGI type II. This developmental disorder requires prevention and early intervention of its dental implications, such as dental caries and attrition. The treatment usually requires a well-planned multidisciplinary protocol involving endodontic and prosthodontic approaches. Histopathological features of shell teeth are required to aid in the identification of the precise pathogenesis and genetic mechanism behind this structural disorder of dentin. It also helps in the modification of certain treatment procedures to accommodate the structural abnormality. The case report aimed to bridge the literature gap on the histopathological features of shell teeth.

Keywords: Dentin defect, Dentinogenesis imperfecta, Developmental anomaly

CASE REPORT

An 18-year-old female patient presented to the Institute with complaints of decayed teeth and poor aesthetics. Her medical history was non contributory. Two years ago, she underwent extraction due to caries. She reported normal exfoliation of milk teeth. Upon intraoral examination of her dentition, generalised yellowish discolouration and attrition of teeth were observed [Table/Fig-1]. Missing teeth noted were at 14, 16, 26, and 46. A carious tooth was grossly present at 47, and root stumps were identified at 11, 12, 13, 21, 22, 23, 24, 25, and 36.



[Table/Fig-1]: Clinical photograph shows generalised yellowish discolouration along with attrition of teeth.

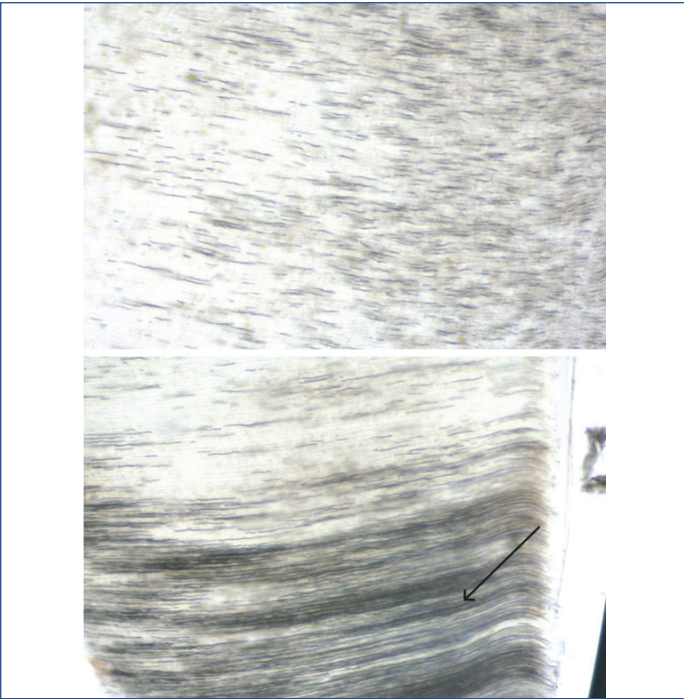
The Orthopantomogram (OPG) examination revealed a generalised absence of enamel on teeth, along with a generalised widening of pulp chambers and root canals surrounded by a thin layer of dentin, suggestive of shell teeth. Ill-defined periapical radiolucencies were observed in relation to 43, 42, 41, 31, 32, and 33. Impacted mandibular second molars were noted on the right and left-sides [Table/Fig-2]. Based on the clinical history, intraoral and radiographic findings, a provisional diagnosis of DGI Type II was provided. The patient was advised to undergo extraction of tooth 11, and the tooth specimen was sent for histopathological examination. The specimen

was sectioned longitudinally into two halves, with one half subjected to ground sectioning and the other processed for decalcification.

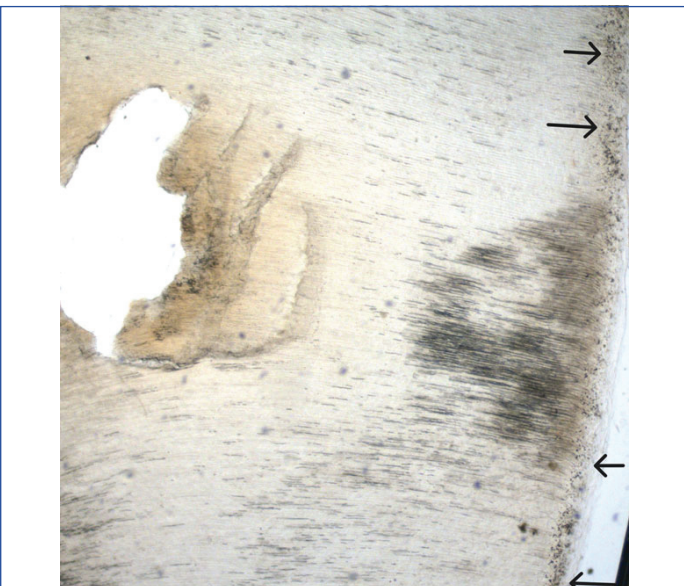


[Table/Fig-2]: OPG shows generalised widening of pulp chambers and root canals surrounded by thin layer of dentin suggestive of shell teeth.

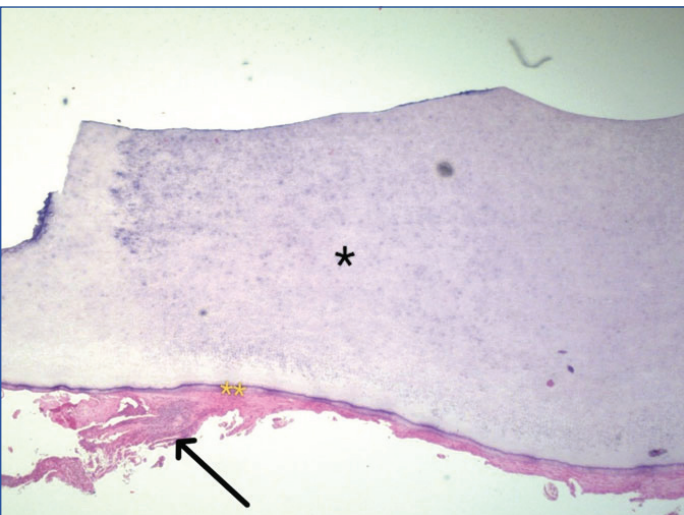
The ground section of the tooth specimen revealed the absence of enamel and the presence of various structures within the cementum and dentin. The dentin exhibited an irregular arrangement of dentinal tubules, with dead tracts observed in a few areas [Table/Fig-3]. The Tomes granular layer was also present adjacent to the cementum [Table/Fig-4]. The decalcified section of the tooth specimen, stained with Haematoxylin and Eosin (H&E) showed the presence of dentinal matrix, cemental matrix, and fibrocellular stroma [Table/Fig-5]. The dentinal matrix contained dentinal tubules appearing as circular structures with enlarged diameters and odontoblast spaces [Table/Fig-6]. Cellular inclusions were noted in some dentinal tubules, along with an irregular arrangement of the dentinal matrix [Table/Fig-7]. The cemental matrix appeared as an intense basophilic layer with incremental lines adjacent to the dentinal matrix. An area of fibrocellular stroma next to the cemental matrix, suggestive of the periodontal ligament, was also observed [Table/Fig-5]. The histopathological results indicated a dentinal defect, leading to the final diagnosis of DGI Type II was provided.



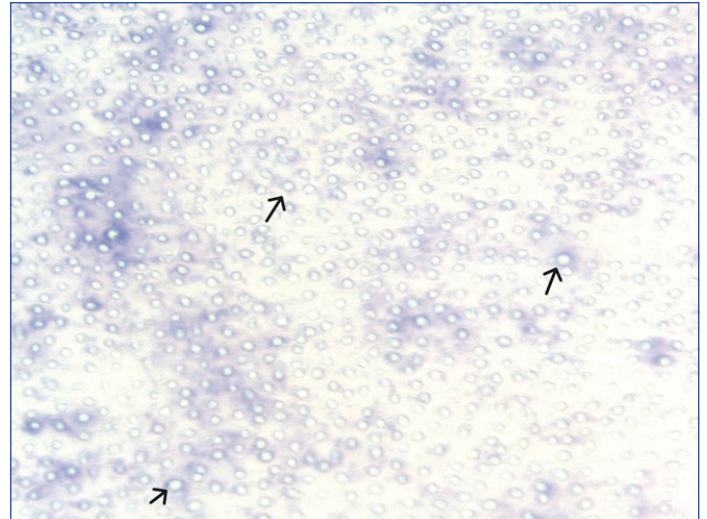
[Table/Fig-3]: Ground section under low power view (100x): Irregular arrangement of dentinal tubules in longitudinal cut section (Top); Presence of dead tracts indicated by black arrow (Bottom).



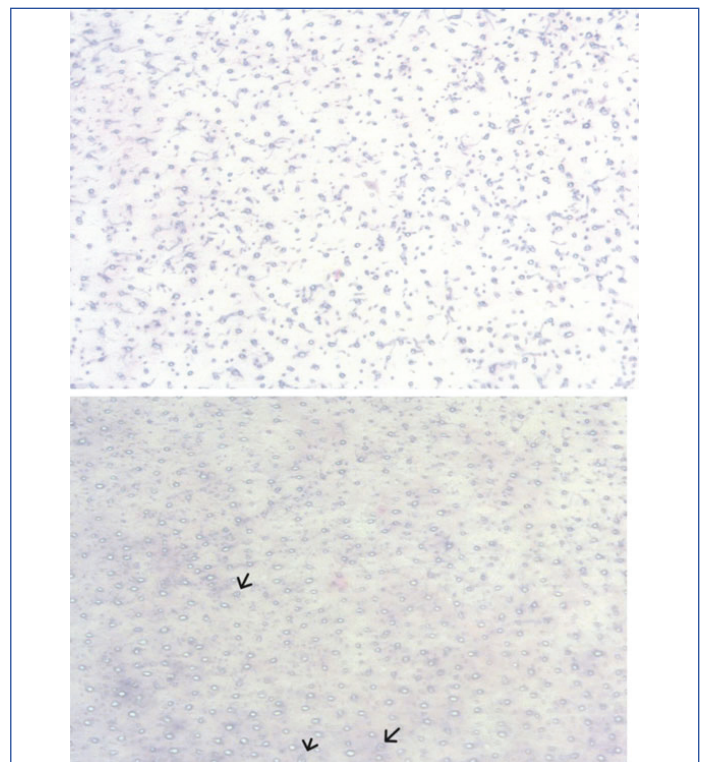
[Table/Fig-4]: Ground section under scanner view (40x): Presence of tomes granular layer (Black arrows).



[Table/Fig-5]: Decalcified section under scanner view. Dental matrix (black asterisk), Cemental matrix (Double yellow asterisk) and fibrocellular stroma suggestive of periodontal ligament (Black arrow). (H&E, 40x)



[Table/Fig-6]: Decalcified section under high power view. Dentinal tubules appearing as circular structures of enlarged diameters with odontoblast spaces. (H&E, 40x)



[Table/Fig-7]: Decalcified section under high power view. Irregular arrangement of dentinal tubules in transverse cut section (Top); Presence of cellular inclusions indicated by black arrows (Bottom). (H&E, 40x)

DISCUSSION

According to Shield in 1973, DGI was classified into three types: a) DGI Type I with Osteogenesis Imperfecta; b) DGI Type II; c) DGI Type III [1]. This classification became outdated as it did not incorporate the molecular etiologies of hereditary dentin defects that were later elucidated. The revised classification is based on the Mendelian Inheritance in Man (MIM) database. This new classification excludes DGI with OI and incorporates Type II and Type III of Shield's classification as Type I and Type II, respectively [2]. The molecular aetiology for DGI Type I and II (Revised classification) involves a mutation in the Dentin Sialophosphoprotein (DSPP) gene. The DSPP gene is located on chromosome 4q21 and is responsible for encoding the major non collagenous protein in dentin secreted by odontoblasts. Mutations in this gene lead to inadequate dentin mineralisation [3].

The DGI is also associated with syndromes other than osteogenesis imperfecta. These include Ehlers-Danlos Syndrome, Goldblatt Syndrome, and Schimke immuno-osseous dysplasia [4].

'Shell teeth,' the term used by many reports in the literature, refers to the radiographic appearance of teeth belonging to DGI Type II (revised classification). Shell teeth have a thin layer of dentin with an enlarged pulp chamber [5]. According to Sivapathasundharam B, the histopathology of shell teeth has not been adequately documented [6]. The first case documented as shell teeth was by Rushton MA in 1954. Rushton considered it as a new form of dentinal dysplasia but then discussed the differential diagnosis primarily regarding DGI. The microscopic features regarding this case were relatively normal initial 0.5-1.0 mm of dentin followed by dentin with fewer tubules and cellular inclusions and a zone of matrix adjacent to the pulp [7]. According to the scoping review by Garrocho-Rangel A et al., the histopathological features of shell teeth include: defective mineralisation, poor quality of organic matrix, dentin with an absence of tubules [8]. The present case showed a defect in the dentinal matrix of its irregular arrangement. Enlarged diameters of dentinal tubules also indicate the dentinal defect. According to Sapir S and Shapira J, the structure of the mantle dentin is normal, whereas the circumferential dentin shows a reduced number of coarse and branched dentinal tubules [9]. The presence of an atubular area in the dentin with reduced mineralisation and a reduced number of odontoblasts are common findings. Other findings include pulpal inclusions and interglobular dentin. Similar findings of pulpal inclusions were also seen in the present case.

CONCLUSION(S)

A proper diagnosis of shell teeth at an early stage helps in determining the prognosis and guiding the treatment. The treatment for shell teeth requires a calculated and multidisciplinary approach.

These teeth are more prone to attrition and periapical abscesses, as seen in the present case. Early intervention is necessary as a preventive measure for dental caries and attrition. Patients also present with malocclusion resulting in poor aesthetics, which requires intervention. Histopathology aids in confirming defects in the dentin that can influence the treatment plan.

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REFERENCES

- [1] Shields ED, Bixler D, El-Kafrawy AM. A proposed classification for heritable human dentine defects with a description of a new entity. *Arch Oral Biol.* 1973;18(4):543-53.
- [2] Garg SK, Bansal S, Mittal S, Bhathal MK. Dentinogenesis Imperfecta-Aetiology And Prosthodontic Management. *Indian J Dent Sci.* 2012;4(1):75-78.
- [3] Kim JW, Simmer JP. Hereditary dentin defects. *J Dent Res.* 2007;86(5):392-99.
- [4] Kantaputra PN. Dentinogenesis imperfecta associated syndromes. *Am J Med Genet.* 2001;104(1):75-78.
- [5] Kaur R, Karadwal A, Sharma D, Sandhu, Manpreet K. Dentinogenesis imperfecta type II: Diagnosis, functional and esthetic rehabilitation in mixed dentition. *J Oral Maxillofac Pathol.* 2021;25(Suppl 1):76-80.
- [6] Sivapathasundharam B. *Shafer's Textbook of Oral Pathology-E Book.* Elsevier Health Sciences; 2016.
- [7] Rushton MA. A new form of dentinal dysplasia: shell teeth. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 1954;7(5):543-49.
- [8] Garrocho-Rangel A, Dávila-Zapata I, Martínez-Rider R, Ruiz-Rodríguez S, Pozos-Guillén A. Dentinogenesis imperfecta type II in children: A scoping review. *J Clin Pediatr Dent.* 2019;43(3):147-54.
- [9] Sapir S, Shapira J. Dentinogenesis imperfecta: An early treatment strategy. *Pediatr Dent.* 2001;23(3):232-37.

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