ORIGINAL ARTICLE

Frequency of developmental defects of enamel and dentin in patients coming to tertiary care clinic of Karachi

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Abstract:

Objective: The objective of the study was to find out the frequency of enamel and dentine developmental defects in patients presenting in tertiary care facility.

Study design: Retrospective descriptive cross-sectional study

Setting & Duration: Data of 10,125 patients coming to oral medicine/diagnosis outpatientdepartment of Sindh Institute of Oral Health Sciences from November 2016 to November 2017 was reviewed.

Material and Methods: Retrospective analysis of data of 10,125 patients coming to oral medicine, diagnostic out patient department was done. Sampling technique was convenient. Data of 13 patients who were diagnosed on basis of clinical criteria were included. Statistical analysis was done through spss17.

Results: Data of 10,125 patients was reviewed, out of which 13 patients diagnosed with developmental disorders of enamel and dentine were included in this study. 10 patients were diagnosed with Amelogenesis imperfecta, fluorosis was found in 2 and 1 case of Dentinogenesis Imperfecta was found.

Conclusion: Amelogenesis imperfecta and fluorosis are more common than Dentinogenesis imperfecta in our region in comparison to western countries. A more comprehensive study on the severity of these defects in Pakistani population and relation to the etiology of these defects are required as due to difference in ethnic background and inter-familial marriages, inheritable diseases are relatively more common in our society.

Keywords: Disorders, enamel, dentin, amelogenesis imperfecta, dentinogenesis Imperfecta

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Introduction:

The abnormalities that affect the formation of enamel and dentine are termed as developmental defect. It consists of fluorosis, amelogenesis imperfecta, dentinogenesis imperfecta and molar incisor hypo mineralization.[1] The etiology varies according to different conditions. Possible causes include genetics (Amelogenesis imperfecta and Dentinogenesis imperfecta), environmental factors (fluoride and strontium), malnutrition (vitamin-D deficiency), low birth weight, premature birth and infectious diseases i.e. chicken pox, measles, mumps, scarlet fever, Tuberculosis, Pneumonia, diphtheria, whooping cough, otitis media and bulbar polio with encephalitis.[2]

These defects vary in discoloration to loss, disfigured enamel, dentin structure affecting both primary and permanent dentition. The prevalence of amelogenesis imperfecta has been estimated in range of 1.4:1000 to 1:160004 and for Dentinogenesis imperfecta is 1:6000-1:8000.[4] These defects affect in pair that is if left side tooth is affected then the contralateral tooth will also be affected. A combination of defects can also be found in same individuals.[5]

Enamel defects are also part of syndrome affecting skin, hair and nail. Some syndromes are listed in (table1). Amelogenesis Imperfecta is caused

Table-1: Syndrome affecting the formation of Enamel and Dentine.[4]

Syndrome	Symptoms
1. Congenital erythropoietic porphyria	Hemolytic anemia, photosensitivity, skin fragility, hypertrichosis and red-brown porphyrin pigmentation of bone and discolored and hypoplastic teeth
2. Ectodermal dysplasia	Two or more ectodermally derived structures are formed abnormally such as skin, hair, teeth, nails and mucous membrane.
3. Epidermolysis bullosa	Blistering of skin, fine to coarse pitting defects along with thin and uneven enamel, delayed or failure of eruption of teeth
4. Tuberous sclerosis	Pitting in teeth, intraoral fibromas, benign tumors may grow in brain and other vital organs.
5. Kindler syndrome	Varying level of enamel defect
6.Tricho-dento-osseus syndrome	Severe hypo mineralization of enamel, taurodontism, abnormalities in bone and hair.
7.Kenney-Caffrey Syndrome	Hypoparathyroidism, hyperopia, dystrophy syndrome, microphthalmia, micrognathia, enamel and dentine abnormalities.
8. DiGeorge Syndrome	Enamel hypo mineralization and hypoplasia
9. Autoimmune polyendocrinopathy candidiasis ectodermal dystrophy syndrome	Hypoparathyroidism, chronic mucocutaneous candidiasis and adrenocortical failure along with thin enamel due to hypoplasia.

Table-2 Criteria for diagnosis of Developmental defects of Enamel and Dentine

	Amelogenesis imperfecta[9]	Dentinogenesis imperfecta[10]	Fluorosis[11]	Molar incisor hypo mineralization
Clinical feature	Type 1: hypoplastic can be white, yellow or brown and thin in thickness.	Type 1: amber translucent in color and attrition	1.Very mild fluorosis: small opaque white areas irregularly over the tooth.	a. Large demarcated opacities, whitish-cream or yellow- brown in color
	Type 2: Hypo maturation is yellow or brown and with normal thickness	Type 2: bulbous crowns	2.Mild fluorosis: white opaque paper-thin areas scatter over the but not involving as much as 25% of tooth.	b. May or may not be associated with post eruption enamel tooth break down
	Type 3: Hypocalcified can be white yellow or brown, soft.	Type 3: similar to type 1 and 2	3.Moderate fluorosis: white opaque areas more extensive but not involving as much as 50%. Brown stains.	c. Hypersensitivity
			4.Severe fluorosis: All enamel surfaces are affected as well as hypoplasia. Brown stains are wide spread.	d. Difficult to anesthetize
				e. Rapid caries progression.
Radiographic features	Type 1: decrease in thickness of enamel	Type 1: pulpal obliteration.	Osteosclerosis of bones, joints and ligaments.	Not required.
	Type 2: normal thickness of enamel.	Type 2: cervical constriction.		
	Type 2: normal thickness of enamel.	Type 3: shell like teeth		

by gene mutation in ENAM, AMELX, KLKS, MMP20, AMELOTIN and FAM83H.[4] Early diagnosis is important for treatment planning as amelogenesis and Dentinogenesis imperfecta increases the susceptibility of these teeth to caries and thus cavitationand tooth loss.[6] Also, the structure of enamel and dentin affected decreases the adhesion, retention and durability of restorations. While the fluorosis makes the teeth brittle. Discoloration requires veneers or crown. Proper history and evaluation to detect the cause may prevent tooth loss resulting in edentulousness. The main complaint is related to esthetics, caries, sensitivity and tooth discoloration. Treatment and prognosis depend upon age, biochemical and morphological characteristics of the defects.[7,8] The aim of this study is to find out the frequency of developmental defects in Pakistani society and corelate it with the western society.

Table-3 Comparison of Prevalence of Enamel and Dentine Defects (DDE)

Type of DDE	Country	Prevalence	Frequency in current study over 1 year out of 10125 cases
Amelogenesis Imperfecta	USA4	1:14000-1:16000	10 cases
	Israel[4]	1:8000	
	Sweden[4]	1.4:1000	
	Turkey[13]	43:10000	
	Pakistan[17]	16/345 patients	
Dentinogenesis Imperfecta	USA[4]	1:6000-1:8000	1 case
	Pakistan[17]	0	
Fluorosis	Pakistan[16]	Affects 53% of population of Gadap (Karachi)	2 cases

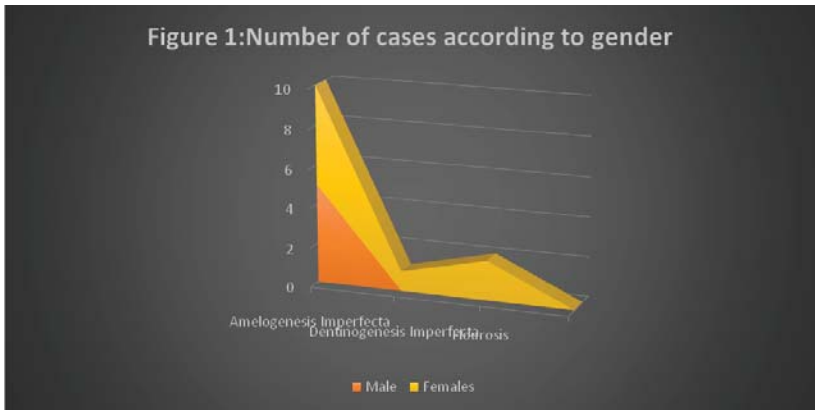

Fig. 1:

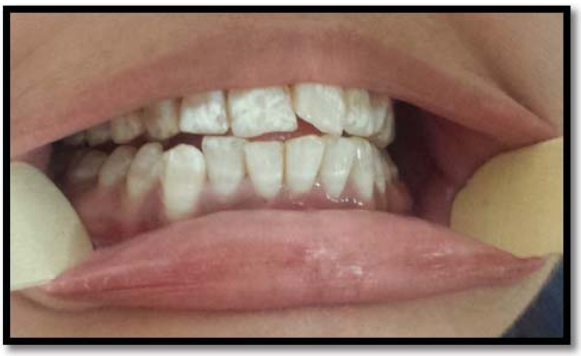

Fig. 2: clinical picture of a female patient with Amelogenesis Imperfecta showing pitting and opacity in incisal edge of all teeth

Material and Methods:

This retrospective descriptive cross-sectional study was conducted from the data of special cases registered at Oral Medicine and Diagnostic centre as regular part of departmental record keeping. Record of diagnosed cases from November 2016- November 2017 around 10,125 forms were analyzed to check the frequency of developmental defects of enemal defects. Convenience sampling was done as they are rare defects with chances of 1.4:1000 to 1:16000 for Amelogenesis imperfecta and Dentinogenesis imperfecta is 1:6000-1:8000.[4]

Cases were included based on recorded history and examination. X-ray record from radiology were retrieved. Patients records which full filled the clinical criteria [table-2] of diagnosis of the developmental disorders of dentine and enamel were included and incomplete forms or missing information were excluded. Performa specially designed for the above objectives was used for this purpose stating age, gender and diagnosis. Data confidentiality was maintained by not mentioning patient specific details such as name, form number and contact details and restricting the access of the data to investigators only. Permission from IRB board (Ref: JSMU/IRB/2018/-121) and administration of the institute was taken. Results were analyzed using SPSS version 17.

Results:

A total of 10,125 patients came to oral diagnosis department of Sindh Institute of Oral Health Sciences during the time period of one year from November 2016 to November 2017 out of which 13 fulfilled the criteria of being diagnosed as developmental disorder of dentine and enamel.

Figure 1 shows that among the cases Amelogenesis imperfecta had the highest percentage as having 10 cases with Fluorosis having the second commonest problem(2 cases) and only 1 case of Dentinogenesis Imperfecta with predominance of female gender. Female to male ratio was 1.6:1. Patients varied over a wide range of age from 7 years to 35 years of age. The commonest reason for seeking dental treatment was dental caries in Amelogenesis and Dentinogenesis Imperfecta cases and Esthetics in Fluorosis cases.

Discussion:

During the time period of our study, a total 10,125 patients came to visit the department for different dental problems out of which only 13 diagnosed as developmental defects. (figure 1)

Major number of those 13 cases included in our study were Amelogenesis Imperfecta (10 cases) followed by Fluorosis (2 cases) and Dentinogenesis Imperfecta (1). Following table 3 compares the prevalence of Developmental Defects globally.

The results show a higher predilection of amelogenesis Imperfecta in our population than USA, Sweden and Israel but less than Turkey. The high rate of inter-familial marriages is responsible for passing on of the autosomal dominant gene affecting all teeth in permanent dentition. The data available of local studies is either on one area of Karachi covering Fluorosis only or the other study does not show the timeline of study that is when data was collected and duration of the study.[16,17] While only 1 case per 10,125 cases of Dentinogenesis Imperfecta show lower prevalence than other countries compared to the study by HR Sukhia which does not show any patients diagnosed with Dentinogenesis imperfecta out of 345 patients.[17]

Amelogenesis imperfecta is divided into four types that is hypoplastic, hypocalcified, hypo maturation, hypoplastic with taurodontism. In clinical photograph 3, pitting and opacity in enamel is evident near incisal edges of all teeth affected symmetrically [Figure 2].

The water content of different areas of Karachi are high in Fluoride responsible for fluorosis of teeth. Areas such as Gadap shows more affected children and adults than other areas.[17] In comparison we had only two patients coming for esthetic problems. They were referred for veneers, micro abrasion.

A multi-disciplinary approach involving pediatric dentistry, orthodontics, prosthodontics along with psychiatry.[7] Direct restoration are preferred in young adults to avoid extensive tooth structure loss during cavity preparation with preferred material being resin based composites.[17] Effective etching with self-etching primers cannot be achieved in these cases thus problems of adhesion of dental restorative material is common.[7] Resin based composite thus have less than 5 year survival in these patients.[17] Susceptibility to caries in Amelogenesis Imperfecta also causes failure.[17] In contrast the Indirect restorations have predictable success rate.[17] The Long term follow-up is required for the success of treatment.

Conclusion:
Amelogenesis imperfecta and fluorosis are more common than Dentinogenesis imperfecta in our region in comparison to western countries. A more comprehensive study on the severity of these defects in Pakistani population and relation to the etiology of these defects are required as due to difference in ethnic background and inter-familial marriages, inheritable diseases are relatively more common in our society.

Limitations: The limitations of this study are that it being retrospective, we cannot compare the severity and etiology behind these defects.

Conflict of interest: None

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Role and contribution of authors:
Dr. Sanaa Ahmed, collected the data, references and did the initial writeup

Dr. Maria Naz, helped in collecting the data and also helped in introduction writing

Dr. Batool Bibi, helped in collecting hte references and also helped in methodology writing.

Dr. Hira Tariq, helped in collecting the data and discussion writing.

Dr. Aisha Naureen, helped in collecting the data and tablulation of the result and conclusion writing.

Dr. Sadaf Talha, critically review the article and made final chanages

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