Enamel Hypoplasia in Children with Renal Disease in a Fluoridated Area

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The aim of this study was to compare the frequency of enamel hypoplasia in children with renal disease and healthy children, all of whom live in a fluoridated area. A cross-sectional study was made in 42 children divided into 2 groups. To describe enamel changes, 3 diagnostic criteria were applied: TSIF Index to describe dental fluorosis, Jackson-Al-Alousi Index to describe enamel hypoplasia, and Russell criteria to differentiate mild forms of dental fluorosis and enamel hypoplasia. The frequency of enamel hypoplasia in patients with renal disease was 38.09%. This frequency is smaller than that seen in other studies. There was no difference in the frequency of dental fluorosis between patients with renal disease and patients without renal disease. However, the patients with renal disease presented more severe dental fluorosis than children without renal disease.
Key words: *enamel hypoplasia, renal disease, dental fluorosis*
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INTRODUCTION

During the last two decades, advances in pediatric nephrology have resulted in a significant increase in the number of children being treated for kidney disease. Many complications observed previously can now be prevented or effectively treated. This is not true in the case of their oral health problems.

Data on epidemiology are available in many countries. The reported incidence of patients with chronic renal failure is 337, 90, 107, and 95 per million population in the US, Australia, New Zealand, and the UK, respectively. Approximately 8 million people in the United States are affected with some type of renal disease.[1,2] The incidence increases with age, and males are more commonly affected than females. Incidence also varies with ethnicity.[3] In Mexico, renal diseases are the ninth cause of death in the general population, with a rate of 10.37per 100,000 inhabitants (SSA, 2000).

Various studies report that 90% of patients with renal disease present oral manifestations. These include ammonia-like smell, dysgeusia (impaired taste), stomatitis, decreased salivary flow, xerostomia, and parotitis.[4-9] Metabolic and pathophysiological changes in children with renal disease affect bone metabolism and reduce the production of vitamin D. Sequelae include demineralization of mandible and maxilla, loss of trabeculation and lamina dura, and metastatic calcification.[6,10] There may be dental pulp mineralization or gingival enlargement secondary to drug therapy.[11] The gingivae may be pale because of anemia or they may bleed easily.[12] There may be mucosal lesions, particularly white patches, ulcerations, or leukoplakia. Stomatitis may manifest as white, red, or grey areas. The prevalence of dental caries is low (due to the raised pH). Urea is present in the saliva, and there is greater concentration of salivary proteins, potassium, and sodium.[2,7,13-17] Anomalies of developing teeth such as enamel hypoplasia,[1,4,5,8,9,14,15,18-23] have been attributed to the production of poorly formed enamel as a result of ameloblast disruption and alteration of calcium and phosphorus metabolism. Enamel hypoplasia, defined as a deficiency in enamel formation, is the main abnormality in the development and mineralization of teeth, and is seen clinically as pits, grooves, or lack of surface enamel. The lesions usually acquire yellow or brown discoloration from deposition of extrinsic pigments.[18] Among the local or systemic factors causing defects or irregularities in the enamel surface should be included hypoplasia related to renal disease and that caused by fluoride.

On the other hand, there is a direct relationship between the level of fluoride ingestion during amelogenesis and dental fluorosis. The prevalence of enamel opacities increases with increasing levels of fluoride ingestion. Many indexes have been used in the

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classification of dental fluorosis. Some problems exist, such as the risk of observer bias and variations in the illumination and dryness of the dental surface, in recording opacities in fluoridated and nonfluoridated areas.[24] Some of these problems have been solved by using specific indexes of fluorosis[25-28] and indexes and criteria to differentiate between fluorotic and nonfluorotic opacities.[25,29,30]

The aim of this study was to compare the frequency of enamel hypoplasia in children with renal disease and healthy children, all of whom live in a fluoridated area.

MATERIAL AND METHODS

This study was approved by the Institutional Review Board of San Luis Potosi University. The study was explained to the legal caregiver, and written informed consent was obtained. A cross-sectional study was made of 42 patients who were divided into 2 groups: group A (21 patients) who had been diagnosed with renal disease and Group B (21 patients) who did not have renal disease and who were brothers of the patients in group A. The inclusion criteria were as follows: medical diagnosis of renal disease, either gender, and aged between 7 and 15 years. Diagnosis of renal disease was made by members of the Department of Pediatric Nephrology, and serum creatinine concentrations were measured in each patient. To describe enamel changes, the following diagnostic criteria were applied: TSIF Index[27] to describe dental fluorosis, Jackson-Al-Alousi Index29 to describe hypoplasia. Type A had white areas less than 2 mm in diameter; type B, white areas equal to or greater than 2 mm in diameter; type C, colored (brown) areas less than 2 mm in diameter, irrespective of any white areas; type D, colored (brown) areas equal to or greater than, 2 mm in diameter, irrespective of any white areas; type E, horizontal white lines, irrespective of any white nonlinear areas; type F, colored (brown) or white areas or lines associated with pits or hypoplastic areas); and Russell criteria[30] to differentiate mild forms of dental fluorosis and enamel hypoplasia.

Studies were completed to assess the reproducibility of recording indexes, and the Kappa value was calculated. Fifty full-arch tooth blocks were examined by the main examiner and another pediatric dentist to assess interexaminer agreement. The Kappa values for interexaminer agreement were 0.85 and 0.89 for the TSIF Index and Jackson-Al-Alousi Index, respectively.

For the statistical analysis, the data were analyzed by nonparametric statistical tests. The chi-square and Wilcoxon tests were used where appropriate. A probability value of <0.05 was considered statistically significant. The JMP IN v. 4.0 statistical program was used to analyze the data.[31]

RESULTS

The population sample of 42 patients included 25 males and 17 females ranging in age from 7 to 15 years with means of 10.23 for group A and 11.76 for group B. The patients´ weight ranged from 18.5 to 74.0 kg, with means of 31.2 for group A and 39.76 for group B. Height ranged from 110 to 178 cm with means of 130.5 for group A and 142.85 cm for group B. Serum creatinine concentrations were 2.65 ± 3.91 for group A and 0.99 ± 1.13 for group B (P <0.05).

None of the patients without renal disease presented enamel hypoplasia, compared with 8 patients (38.09%) with renal disease, who presented with enamel hypoplasia (P <0.05) (Table 1). Of those, 4 patients (50%) presented with type B (white areas equal to or greater than 2 mm in diameter), according to the Jackson Al-Alousi Index (Table 2). There was no difference in the frequency of dental fluorosis between patients with renal disease and patients without, but significantly more patients without renal disease had dental fluorosis (Table 3). Also, children with renal disease presented a more severe dental fluorosis (P <0.05, Wilcoxon) (Table 4). There was no statistically significant difference in frequency between males and females.

Further, the group with renal disease was subdivided into 2 subgroups: patients with chronic renal failure (n = 11) and those with other renal pathologies (n = 10). Of the patients with chronic renal failure, 6 (54.55%) presented with enamel hypoplasia as well as a greater number of affected teeth, in contrast to the patients with other renal pathologies; in the latter subgroup only 2 (20%) presented with enamel hypoplasia. Also, the patients with other renal disease presented with a higher frequency of dental fluorosis compared with patients with chronic renal failure.

Table 1: Frequency and percentage of enamel hypoplasia in patients with renal disease and without renal disease

Enamel hypoplasia	Patients with renal disease		Patients without renal disease	
	Frequency	Percent	Frequency	Percent
No	13	61.91	21	100.00
Yes	8	38.09	-	-
Total	21	100.00	21	100.00

P <0.05; X^2

Grade	Frequency	Percent
A	2	25.00
B	4	50.00
C	1	12.50
D	1	12.50
Total	8	100.00

Table 2: Frequency and percentage of type of hypoplasia in patients with renal disease (Jackson-Al-Alousi Index)

Table 3: Frequency and percentage of dental fluorosis in patients with renal disease and patients without renal disease

Dental fluorosis	Patients with renal disease		Patients without renal disease	
	Frequency	Percent	Frequency	Percent
No	11	52.38	8	38.09
Yes	10	47.62	13	61.91
Total	21	100.00	21	100.00

P <0.05; X^2

Table 4: Severity of dental fluorosis in patients with renal disease and patients without renal disease

Dental fluorosis	Patients with renal disease		Patients without renal disease	
	Mean	Standard deviation	Mean	Standard deviation
TSIF Index	2.75	± 1.70	1.83	± 1.16

P <0.05; X^2 Wilcoxon

DISCUSSION

Enamel hypoplasia is the most common abnormality in the development and mineralization of human teeth.[20] This study demonstrates that renal disease in children is associated with enamel hypoplasia. This might be due to a disturbance in dental development. Enamel hypoplasia, frequently seen in patients with renal disease, has been attributed to the production of poorly formed enamel as a result of ameloblast disruption. Factors causing this disruption include hypocalcemia, decreased serum levels of 1.25-dihydroxycholecalciferol, and increased serum levels of inorganic phosphate and serum parathyroid hormone.[32] The hypoplasia is manifested as diffuse or demarcated enamel opacities or enamel hypomineralization. This is seen clinically as pits, grooves, or generalized lack of surface enamel. Enamel hypoplasia is important because it can result in caries, tooth sensitivity, and poor esthetics. It involves a burden of care usually requiring the techniques of esthetic dentistry. Its pathogenesis is that severe metabolic upset causes a permanent defect in the developing tooth. The position and extent of the defect indicates the timing, duration, and to some extent, the severity of the underlying metabolic upset.

This prevalence has been reported in various studies. In patients with renal disease, it varies from 31% to 83%, depending on the racial, ethnic, nutritional, or socioeconomic status of the child, type of classification system used, and method of examination. Badger reported enamel hypoplasia in 22% of 55 children 1 year six months to 11 years in age with no differences in sex; however, the criteria used for the diagnosis were not mentioned.[33] Slayton et al, examining 698 children, found that 44 (6%) had enamel hypoplasia on at least 1 primary tooth, 3% had 1 permanent tooth affected, 2% had 2 teeth affected, and less than 1% had 3 teeth affected. In their study, fluorosis and nonfluoritic opacities were differentiated using Russell´s criteria and features of the developmental defects of enamel index.[34] Llarena and Madrigal found enamel hypoplasia in 15.4% of 91 patients having various nephropathies.[35] These data contrast with our study wherein 38.09% of patients with renal disease presented enamel hypoplasia. Nunn *et al* reported enamel defects having an unusual pattern in children with renal disease. They had a much higher prevalence of diffuse opacities and enamel hypoplasia than did normal children (83% and 22%, respectively). They attributed this increased prevalence to disordered calcium and phosphate metabolism.[36] Enamel opacities were assessed in the permanent dentition using the Developmental Defects of Enamel Index. Nowaiser et al, using the same methods observed enamel defects in 57% of the chronic renal failure children compared with 33% of the controls.[37]

The results obtained in this study differ from others. The reason could be the criteria used for diagnosing enamel hypoplasia, because it is possible to confuse enamel hypoplasia with other defects such as dental fluorosis. In this respect, if we added the frequency of enamel hypoplasia and the frequency of dental fluorosis in children with renal disease we would come up with 85.7% of the patients having enamel defects; this result is similar to others published recently.

Dental fluorosis is a specific disturbance in tooth formation, and is defined as a chronic, fluoride-induced condition in which enamel development is disrupted and the enamel is hypomineralized. Clinically, dental fluorosis is seen as white spots or opaque white lines (striations), or the tooth surface may have a white parchment-like appearance. At higher concentrations of fluoride, discrete or confluent pitting of the enamel surface is seen, accompanied by extrinsic stains.[38] Dental fluorosis is symmetrically distributed, and the severity varies among the different types of teeth. Teeth that develop and mineralize later in life have a higher prevalence of dental fluorosis. The effects of fluoride on enamel formation in man are cumulative, rather than requiring a specific threshold dose. These effects depend on the total fluoride intake from all sources. The principal source of fluoride is drinking water; many cities have large amounts of natural fluoride in their drinking water, putting the population at risk of developing dental fluorosis. The city of San Luis Potosi (SLP), Mexico, is located in an area where drinking water contains excessive quantities of natural fluoride. Results showed that 61% of tap water samples collected in SLP had fluoride levels between 0.7-1.2 ppm.[39] Also, the high prevalence of dental fluorosis in SLP cannot be attributed only to the levels of fluoride in drinking water; other risk factors must be considered.[40-42]

On the other hand, studies have shown that excessive fluoride can affect certain body systems such as the urinary system. High concentrations of fluoride produce renal toxicity.[43] Warady *et al* reported that patients with chronic renal failure have high plasma fluoride levels. Since the kidneys have an important function in the removal of inorganic fluoride, this malfunction might be an etiological factor in the prevalence of enamel defects.[44] In our study, 61.9% of patients without renal damage presented with fluorosis, in contrast with 47.61% of patients with renal damage. In the present study, dental fluorosis was differentiated from enamel hypoplasia using the criteria of Russell. These criteria were used to differentiate slight forms of enamel hypoplasia from dental fluorosis based on such characteristics as affected area, shape of lesion, demarcation, color and teeth affected. In some studies, the Developmental Defects of Enamel Index has been used; however, this index is a subjective method in the perception of color; in addition, the analysis is complicated. Perhaps the use of this index is the reason the prevalence of enamel hypoplasia might be overestimated. This confirms the necessity of being familiar with the criteria for diagnosing enamel hypoplasia to differentiate it from other opacities in patients living in communities having chronic endemic dental fluorosis.

CONCLUSION

Children with renal disease presented a frequency of enamel hypoplasia of 38.09%, which is less than that found in other studies. There was no difference in the frequency of dental fluorosis

between patients with renal disease and those without renal disease. However, patients with renal disease presented a more severe form of dental fluorosis than children without renal disease.

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