

Oral Health Status in Children with Renal Disorders

Subramaniam P * / Gupta M ** / Mehta A ***

Advances in pediatric nephrology have resulted in increased survival rates of children with renal disorders. Renal disease is characterized by multiple organ involvement, including soft and hard tissues of the oral cavity. Data regarding the oral health status of Indian children with renal disorders is scarce. Thus, the aim of this study was to assess the oral health status of children with renal disorders in Jaipur city, India. Thirty six children in the age-group of 4-14 years, diagnosed with renal disorders were selected. Data pertaining to demographics, medication history, body mass index and blood investigations were obtained from the hospital records. The World Health Organization (WHO) criteria were used to diagnose dental caries. Enamel defects were recorded according to Developmental Defects of Enamel index. Oral hygiene status, salivary pH and buffering capacity were also assessed. The mean blood hemoglobin value was 9.75gm/dl, blood urea nitrogen 43.06 gm/dl and serum creatinine 1.5 mg/dl. Enamel defects were seen in 58.3% of children. Their mean deft and DMFT scores were 1.5 and 0.5, respectively. The mean Oral Hygiene Index-Simplified (OHI-S) score was 1.56. Gingival overgrowth was not present. Mean salivary pH was 6.92 and buffering capacity of stimulated saliva was 9.86. It is necessary for pediatric dentists to follow preventive oral health regimens that are tailored to these patients.

Keywords: Renal disorder, Enamel Defects, Salivary pH, Dental caries.

J Clin Pediatr Dent 37(1): 89–94, 2012

INTRODUCTION

The estimated incidence of end-stage renal failure (ESRF) in childhood, either due to a congenital or acquired condition, is 10 to 12 per 1 million children, with a prevalence varying between 39 to 56 million children in the United States.¹

Some of the renal disorders seen in children are nephrotic syndrome (NS), chronic renal failure (CRF) and Glomerulonephritis (GN). Nephrotic syndrome is a chronic disorder characterized by alterations of permselectivity at the glomerular capillary wall, resulting in its inability to restrict the urinary loss of protein. The proteinuria in childhood nephrotic syndrome is relatively selective, constituted

primarily by albumin.² Estimates on the annual incidence of nephrotic syndrome range from 2-7 per 1,00,000 children and prevalence from 12-16 per 1,00,000 in the United States.³ There is epidemiological evidence of a higher incidence of nephrotic syndrome in children from south Asia than non-Asians.⁴

Chronic renal failure is characterized by a reduction in glomerular filtration rate either due to glomerular or renal interstitial disease.⁵ Chronic renal failure was defined as a serum creatinine (SCr) value persistently above 1.2 mg/dl and End Stage Renal Disease (ESRD) as the stage when renal replacement therapy by dialysis or transplantation was required.⁶ Glomerulonephritis is a renal disease characterized by inflammation and damage to the capillary loops of the kidneys.⁷

Renal disease involves multiple organs including the soft and hard tissues of the oral cavity. Oral manifestations of children with renal diseases include ammonia-like odor, dysgeusia (impaired taste), stomatitis, xerostomia, parotitis, decreased salivary flow, gingival enlargement secondary to drug therapy. Enamel opacities may be seen due to disturbed calcium and phosphate metabolism. Oral findings frequently include increased plaque accumulation, gingivitis, gingival overgrowth and enamel hypoplasia.⁸ The prevalence of dental caries is low in these children.⁹

Advances in pediatric nephrology during the last two decades have resulted in a marked increase in the survival rate of children with chronic renal failure, and on renal replacement therapy.⁸ Pediatric dentists are likely to see an increasing number of these children in their routine practice.

* Priya Subramaniam, MDS Professor and Head, Department of Pedodontics and Preventive Dentistry, The Oxford Dental College, Hospital and Research Centre, Karnataka, India.

** Megha Gupta, MDS, Former Postgraduate Student, Department of Pedodontics and Preventive Dentistry, The Oxford Dental College, Hospital and Research Centre, Karnataka, India.

*** Amarjeet Mehta, MD, Professor, In charge, Department of Pediatric Nephrology, SMS Medical College and Hospital, Jaipur, Rajasthan, India.

Send all correspondence to: Dr Priya Subramaniam, Department of Pedodontics and Preventive Dentistry, The Oxford Dental College, Hospital and Research Centre, Bommanahalli, Hosur Road, Bangalore-560068, Karnataka, India.

Phone: + 91 9844225624

Fax : + 080 91 25734656

E-mail: drpriyapedo@yahoo.com

Thus, it is essential for them to know and understand the general health parameters of these children. In India, there appears to be paucity in literature with regard to the oral health conditions of children with renal disorders. Therefore, the aim of this study was to assess the oral health status in children with renal disorders.

MATERIALS AND METHOD

The study was carried out at the Department of Pediatric Nephrology, SMS Medical College and Hospital, after obtaining prior permission from the concerned hospital authorities. Ethical clearance was obtained from the institutional review board and written consent was also obtained from the parents. Initial screening of 68 children with renal disorders was carried out. The children comprised of both boys and girls in the age group of 4–15 years.

The exclusion criteria included children with acute renal disease (renal failure due to malaria, acute glomerulonephritis, acute tubular necrosis, vasculitis) uncooperative children, those children with any other systemic disorder in addition to renal impairment and children who underwent dialysis on the day of examination. Thus, the study group comprised of 21 children suffering from nephrotic syndrome; 8 children with chronic renal failure and 7 children suffering from glomerulonephritis. There were 28 boys (77.8%) and 8 girls (22.2%) aged between 4–14 years, with a mean age of 7.92 years. Information pertaining to demographics, medication history (Table 1), height, weight and blood investigations was taken from the hospital records.

The Body Mass Index (BMI) for age was assessed as per the Center for Disease Control (CDC, 2000) growth charts, for boys and girls.¹⁰ After recording the individual height and weight of each child, the BMI for age was calculated as weight in kilograms/ height in meters. The value obtained was plotted on the gender and age specific weight for height curves on the charts, in order to obtain a percentile. According to these curves, the children were classified as underweight, normal, risk of overweight and overweight.¹⁰

Oral examination was carried out at the hospital in natural daylight using sterile mouth mirrors with good reflecting surfaces, and disposable sterile gloves. Examination did not reveal soft tissue pallor or gingival enlargement. The WHO criteria was used for the diagnosis and recording of

Table 1. Medication History

Nephrotic syndrome	Corticosteroids (Prednisolone) Immune - modulator (Levamisole) Immunosuppressant (Cyclophosphamide)
Chronic renal failure	Combination of antihypertensives (Amlodipine, Enalapril, Atenolol, Clonidine, Frusemide) Corticosteroids (Prednisolone) Antibiotics (Cephalosporins),
Glomerulonephritis	Antihypertensives , Diuretics

Supplements which included iron, calcium and multivitamins were given to all the children.

dental caries using a Community Periodontal Index (CPI) probe.¹¹ Individual deft and DMFT scores were given for each child. Teeth were examined for enamel defects and recorded according to the DDE (Developmental Defects of Enamel) index¹² (Table 2A, 2B & 2C). Oral hygiene was assessed using the Oral Hygiene Index-Simplified¹³ and for deciduous teeth the modified index given by Miglani *et al*¹⁴ was followed.

Salivary pH and buffering capacity were assessed using GC Saliva check Kit (GC Asia). As per the manufacturer's instructions, the children were asked to refrain from eating and drinking 2 hours prior to the test. In order to assess salivary pH, unstimulated saliva was used. The children were instructed to expectorate any pooled saliva into a collection cup. A drop of the saliva sample was then placed on a pH

Table 2A. Type of Defect

Type of defect	Code	
Normal	0	A
Opacity (white/cream)	1	B
Opacity (yellow/brown)	2	C
Hypoplasia (pits)	3	D
Hypoplasia (grooves: horizontal)	4	E
Hypoplasia (grooves: vertical)	5	F
Hypoplasia (missing enamel)	6	G
Discolored enamel	7	H
Other defects	8	I

Table 2B. Number and Demarcation

Number and demarcation of defect	Code	
	Permanent teeth	Deciduous teeth
Single	1	A
Multiple	2	B
Diffuse (fine white lines)	3	C
Diffuse (patchy)	4	D

Table 2C. Location of Defect

Location of defect	Code
No defect	0
Gingival one – half	1
Incisal one-half	2
Gingival and incisal halves	3
Occlusal	4
Cuspal	5
Whole surface	6
Other combinations	7

strip and observed for any color change following 10 seconds. This was compared with the color coded pH indicator chart, which also gave individual pH scores. The pH ranging from 5 - 5.8 was considered as highly acidic, 6.0 - 6.6 - moderately acidic and 6.8 - 7.8 - healthy saliva (according to the test chart provided by the manufacturer).

Stimulated saliva was used to assess the buffering capacity. The children were asked to chew on a paraffin pellet for 30 seconds following which they had to expectorate into a graduated cup. This was done every 30 seconds, over a period of 5 minutes. At the same time, a buffer test strip was removed from the foil package and placed onto an absorbent tissue with its test side up. With the help of a micropipette, one drop each of saliva was dispensed on the three individual test pads. The strip was then immediately turned 90 degrees to soak up any excess saliva from swelling on the test pad and affecting the accuracy of the result. The test pad changed color immediately and after 2 minutes the final color was noted. Points were given for each pad as follows: green - 4, green / blue - 3, blue - 2, red / blue - 1 and red was given 0. The total point value of the three pads gave the final buffering score which ranged from very low buffering capacity (0-5), low buffering capacity (6-9), and high buffering capacity (10-12).

The data obtained was analyzed using SPSS version 15. Two tailed Pearson correlation test was used to find out the

significance between variables. Significance for all the statistical tests was predetermined at a 'p' value of 0.01 or less.

RESULTS

The mean general health parameters are given in Table 3. The mean Body Mass Index score of the study group was 20. According to CDC growth charts (2000), for both boys and girls, 44.44% were underweight, 27.78% were normal, 11.11% were at a risk of overweight and 16.67% were overweight.

The soft tissue including the gingiva appeared to be normal in all children. The mean OHI-S score was 1.56. The mean deft and DMFT scores were 1.50 and 0.5, respectively. Salivary pH showed a mean value of 6.92 and for salivary buffering capacity, it was 9.86. The mean values of oral health parameters obtained for each type of renal disorder are given in Table 4. Salivary pH and buffering capacity was correlated with the mean deft score. This inverse correlation was -0.636 for pH and -0.600 for buffering capacity, which were highly significant ($p \leq 0.01$). On correlating the salivary pH and buffering capacity with the mean DMFT score, a significant correlation of -0.247 was observed only with buffering capacity.

Enamel defects were seen in 58.3% of children. In children with nephrotic syndrome, occlusal surfaces of primary molars showed white/cream and yellow/brown opacities that were diffuse and patchy. Hypoplasia of the primary maxillary anterior teeth occurred as a single defect, with enamel missing in the gingival half. In both chronic renal failure and glomerulonephritis, white/cream, diffuse and patchy opaci-

Table 3. Mean values of general health parameters

GENERAL HEALTH PARAMETERS	MEAN VALUE	STANDARD DEVIATION
Blood Hemoglobin (gm / dl)	9.75	1.214
Blood Urea Nitrogen (mg /dl)	43.06	21.310
Serum Creatinine (mg /dl)	1.549	2.1343
Height (meters)	1.107	.2800
Weight (kgs)	24.4583	10.94491
BMI	20.00	6.621

Table 4. Mean values of oral health parameters according to type of renal disorder

TYPE OF RENAL DISORDER	Salivary pH	Salivary Buffering Capacity	Deft	DMFT	OHI-S
Nephrotic syndrome	6.85	9.9	1.6	0.4	0.87
Chronic renal failure	6.95	9.75	0.8	0.5	0.77
Glomerulonephritis	7.02	9.85	2.0	1.0	2.04

Table 5. Distribution of enamel defects

Percentage of children affected	Total no. of teeth	Percentage of teeth affected	Percentage of primary teeth affected				Percentage of permanent teeth affected
			incisor	canine	first molar	second molar	
Nephrotic syndrome 76.19% (16/21)	443	15.80% (70/443)	11.43% (8/70)	8.57% (6/70)	15.71% (11/70)	48.57% (34/70)	15.71% (11/70)
Chronic renal failure 25% (2/8)	192	4.17% (8/192)	-	-	-	-	100% (8/8)
Glomerulo-nephritis 28.57% (2/7)	164	9.76% (16/164)					100% (16/16)

ties were seen on the occlusal surfaces of permanent molars. The distribution of enamel defects is given in Table 5.

DISCUSSION

In our study, the age of the children ranged from 4-14 years, hence it was not possible to refer to the WHO BMI charts wherein, the lower age limit is 5 years. Similarly, the growth charts for Indian children do not include children below 5 years of age.¹⁵ It has been reported that these percentiles are higher for 5th to 95th percentiles as compared to the CDC growth charts. This is due to under-nutrition which is more prevalent in Indian children.¹⁵ However, BMI is a derived number (a ratio) and is therefore unlikely to be altered significantly by minor errors. In fact, this is why BMI is an effective epidemiological tool for use in the community.¹⁶

Some of the children with nephrotic syndrome appeared to be cherubic due to generalized edema resulting in cushingoid facies.⁷ Children with CRF were also advised to restrict the intake of fruits and juices due to their high potassium content and this could lead to hyperkalemia.⁸ A high number of children with CRF and glomerulonephritis were found to be underweight.⁸ This was probably because of the low protein, high carbohydrate diet followed in order to reduce the renal workload.

The systemic condition of children with renal disease exhibits oral manifestations and it has specific implications for dental treatment. Since children in our study were anemic (mainly due to hematuria), they were given regular iron supplements. Thus pallor of the skin and oral tissues could not be well appreciated. Increased levels of blood urea nitrogen could have been responsible for their high salivary pH and buffering capacity values. The salivary pH was measured using pH strips contained in the Saliva Check kit. This is a relatively easy, quick and convenient method in which unstimulated saliva is evaluated according to the color change observed on the pH test strips. Buffering capacity is distinguished from pH per se in that, whilst pH of saliva is a labile parameter highly influenced by the types and timing of food intake as well as oral hygiene habits, buffering capacity is a more useful measure of an individual's innate ability to maintain a neutral or slightly alkaline pH in saliva.¹⁷ The bicarbonate system of saliva is the main mechanism of buffering action of saliva. The bicarbonate concentration is very low in unstimulated saliva and such saliva is poorly buffered.¹⁸ Therefore, stimulated saliva was used to assess buffering capacity, using buffer strips. The colorimetric method was followed because it is a simple, non invasive, chair side test which could be easily carried out in a hospital setting. It was also possible to discriminate between high, medium and low buffering capacity.

The gingival overgrowth seen in ESRD is believed to be related to an alteration of the fibroblast metabolism by cyclosporine and/or its metabolites, increasing protein synthesis, collagen, antihypertensive medication and extracellular matrix formation.⁸ In an earlier study, presence of gingival hyperplasia in children with renal disease did not

show any relationship with the use of immunosuppressant therapy.⁹ Absence of gingival overgrowth in our study is possibly due to the variable drug regimen and combination of antihypertensive medication, which in turn could have masked the effects of any individual drug.

Children with CRF are more susceptible to increased plaque and calculus formation. This may be due to the presence of high salivary urea.¹⁹ The high calcium and phosphate supplements often given to these children might increase the salivary concentration of these ions.²⁰ Regular brushing and rinsing after meals, practiced by these children could have been the reason for their fair oral hygiene.

The presence of enamel defects in 58% of children, involving both primary and permanent dentition was an interesting observation made in our study. Enamel defects were of unusual pattern with a higher prevalence of diffuse opacities and enamel hypoplasia.⁹ An early decline in renal function appears to be important for the development of enamel defects. Enamel defects of deciduous dentition indicate prenatal/early postnatal damage affecting ameloblasts/enamel maturation.⁶ Calcium, phosphorus and vitamin D metabolism is also disturbed in children with chronic renal failure. Intestinal absorption of calcium is diminished early in renal failure because the kidneys cannot convert vitamin D to its active form; there is also a corresponding retention of phosphate which ultimately leads to decreased serum calcium levels.²¹ A diet low in protein along with proteinuria is mainly responsible for defective matrix formation, leading to hypoplasia. None of the children were either preterm or of low birth weight, and so these factors could not be considered as a cause of their enamel hypoplasia. Although the children in our study had an increased exposure to dietary carbohydrates and hidden sugars in long-term medicated syrups, they showed low caries in both primary and permanent dentition. This is due to a combination of factors including their high salivary pH and buffering capacity, regular intake of calcium supplements and fair oral hygiene. Children with CRF have significantly less dental caries than healthy children due to the inhibitory effect of increased salivary urea levels.²² Salivary urea is metabolized by oral microflora to form ammonia and carbon – dioxide, which may raise the pH above the critical level for enamel demineralization. This would negate the effect of any acid formation resulting from sugar intake and neutralize the acidic pH in the oral environment.^{20, 23}

When salivary pH was evaluated independent of buffering capacity, a correlation between resting salivary pH and the dental caries experience was found.²⁴ Also, it was indicated that salivary pH is a poor to moderate risk factor for caries prevalence and incidence.²⁴ However in this study, a significant correlation was found between the unstimulated salivary pH and dental caries in the primary dentition. There is also no definitive consensus on whether or not the stimulation status per se of whole saliva pH is important in caries risk.²⁵

Nunn *et al* reported a mean DMF of 0.9 and dmf of 0.8 in

thirty children aged 2 to 16 years, suffering from various renal disorders. Fifty percent of the children never experienced decay.⁹ Nowaiser *et al*²⁶ reported mean salivary pH to be 6.4 in 4 to 13.6 year old children suffering from CRF. They also found a mean dmft of 1 and DMFT of 2.7 in these children. Yahya and Ali²⁷ reported a mean DMF score of 2.25 in 5-18 year old children with chronic renal failure in Tehran.

Immune deregulation in children with renal disorders is apparent by some degree of immunosuppression invariably seen in them. Moreover, corticosteroids form the first line of drugs in the treatment of nephrotic syndrome. These children are at a high risk of infection and are susceptible to bacteremia. Antibiotic prophylaxis is necessary prior to invasive dental treatment and it is essential to monitor drug dosage in these patients. Some drugs are nephrotoxic in themselves and the added strain these drugs exert on the already damaged kidney must be avoided.²¹ Timely referral and communication with the pediatric nephrologist is essential.

Effective preventive strategies should be employed to avoid aggressive treatment procedures. The prescription of additional fluorides (other than that received from fluoridated water and toothpastes) is contraindicated.⁹ Children have lower renal fluoride clearance rates and a moderate impairment of renal function could lead to increased retention of fluoride.²⁸ Several studies^{9,20,26,27} have shown that children with renal disorders have low caries. Thus, routine professional topical fluoride application may not be necessary for all children. In addition to nutritional assessment, tailored diet counseling combined with periodic dietary review would certainly be beneficial to oral health. Placement of sealants, restoration of hypoplastic teeth, and maintenance of regular oral hygiene is recommended.

CONCLUSION

From this study it was concluded that children with renal disorders have a high number of enamel defects and low caries. The saliva of these children showed a high pH and buffering capacity.

REFERENCES

1. Trivedi HS, Pang MM. Discrepancy in the epidemiology of non diabetic chronic renal insufficiency and end – stage renal disease in black and white Americans: The third National Health and Nutrition Examination Survey and United States Renal Data System. *Am J Nephrol*, 23: 448–457, 2003.
2. Bagga A, Mantan M. Nephrotic syndrome in children. *Ind J Med Res*, 122: 13–28, 2005.
3. Eddy AA, Symons JM. Nephrotic syndrome in children. *Lancet*, 362: 629–639, 2003.
4. Mc Kinney PA, Feltbower RG, Brocklebank JT, Fitzpatrick MM. Time trends and ethnic patterns of childhood nephrotic syndrome in Yorkshire, UK. *Pediatr Nephrol*, 16:1040–1044, 2001.
5. Riegden S. The management of chronic and end–stage renal failure. In: Webb N, Postlethwaite R. *Textbook of Clinical Pediatric Nephrology*. 3rd edition: Oxford Medical Publications GB: 427- 445, 2003.
6. Koch MJ, Buhner R, Pioch T, Scharer K. Enamel hypoplasia of primary teeth in chronic renal failure. *Pediatr Nephrol*, 13: 68–72, 1999.
7. Guyton & Hall. *Textbook of Medical Physiology*. Elsevier Publication, 402–418, 2005.
8. Davidovich E, Davidovits M, Eidelman E, Schwarz Z, Bimstein E. Pathophysiology, Therapy, and Oral Implications of Renal Failure in Children and Adolescents: An update. *Pediatr Dent*, 27: 298–105, 2005.
9. Nunn JH, Sharp J, Lambert HJ, Plant ND, Coulthard MG. Oral health in children with renal disease. *Pediatr Nephrol*, 14: 997–1001, 2000.
10. US DHHS. National Centre for Health Statistics 2000 CDC growth charts for the United States: Methods and development. *Vital Health Stat*, 11: 41–41, 2002.
11. WHO: Oral health Surveys–Basic methods. Geneva: 4th edition, 1997.
12. FDI commission on Oral Health, Research and Epidemiology. An epidemiological index of developmental defects of dental enamel (DDE index). *Int Dent J*, 32: 159–167, 1982.
13. Greene JC, Vermilion JR. The simplified oral hygiene index. *J Am Dent Assoc*, 68: 7, 1964.
14. Miglani DC, Beal JF, PMC Beans, Behari SA. The assessment of dental cleanliness status of the primary dentition using a modification of the simplified oral hygiene index. (OHIS - M). *Journal of Indian Dent Assoc*, Dec: 385–388, 1973.
15. Agarwal KN, Saxena A, Bansal AK, Agarwal DK. Physical growth assessment in adolescence. *Ind Pediatr*, 38: 1215–35, 2001.
16. Banerjee I, Ghia N, Bandopadhyay S, Sayed HN, Mukherjee D. Body mass index in Bengali adolescents. *Ind Pediatr*, 42: 262–267, 2005.
17. Dowd FJ: Saliva and dental caries. *Dent Clin North Am*, 43(4): 579–597, 1999.
18. Nikiforuk G: *Understanding Dental Caries*. Karger. Sydney, p. 239–247, 1985.
19. Shannon IL, Feller RP, Eknoyan G, Suddick RP. Human parotid saliva in renal failure and during dialysis. *Arch Oral Biol*, 22: 83–86, 1977.
20. Jaffe EC, Roberts GJ, Chantler C, Carter JE. Dental findings in chronic renal failure. *Br Dent J*, 160: 18–20, 1986.
21. De Rossi SS, Glick M. Dental considerations for the patient with renal disease receiving hemodialysis. *J Am Dent Assoc*, 127: 211–19, 1996.
22. Lucas VS, Roberts GJ. Oro-dental health in children with chronic renal failure and after renal transplant: a clinical review. *Peadiatr Nephrol*, (10): 1388–1394, 2005.
23. Greenberg MS, Glick M. *Burket's oral medicine diagnosis and treatment*. 10th edition. Philadelphia; p. 417–9, 2003.
24. Leone CW and Oppenheim FG: Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. *J Dent Educ*, 65(10): 1054–1062, 2001.
25. Alamoudi N, Farsi N, Faris J, Masoud I, Merdad K, Meisha D. Salivary characteristics of children and its relation to oral microorganism and lip mucosa dryness. *J Clin Pediatr Dent*, 28(3): 239–48, 2004 .
26. Al Nowaiser A, Roberts GJ, Trompeter RS, Wilson M, Lucas VS. Oral health in children with chronic renal failure. *Pediatr Nephrol*, 18: 39–45, 2003.
27. Yahya NB, Ali B. The dental and oral status of children with chronic renal failure. *J Indian Soc Pedod Prev Dent*, Mar: 7–9, 2007.
28. Spak CJ, Berg U, Ekstrand J. Renal clearance of fluoride in children and adolescents. *Pediatrics*, 75: 575–79, 1985

