

International Journal of Dental Science and Innovative Research (IJDSIR)

International Journal of Denial Science and Innovative Research (IJDSIR : Dental Publication Service Available Online at: www.ijdsir.com Volume – 2, Issue – 1, January - February - 2019, Page No. : 181 - 188

Evaluation and detection of inter relationship between caries index and physiological factors of saliva in uncontrolled type 1 diabetic children

Hariharavel VP¹, Arun Prasad Rao V², Venu Gopal Reddy N³, Mohan G⁴, Sugumaran DK⁴, Shankar P⁵ ¹Associate Professor, Department of Dentistry, Andaman and Nicobar Islands Institute of Medical Science, Port Blair ²Professor and HOD, Department of Pedodontics and Preventive Dentistry, Mahatma Gandhi Post Graduate Institute of

Dental Sciences, Pondicherry- 605006

³Professor and HOD, Department of Pedodontics and Preventive Dentistry, Mamata Dental College and Hospital, Khammam -507002

⁴Professor, Department of Pedodontics and Preventive Dentistry, Rajah Muthiah Dental College and Hospital,

Chidambaram

⁵Reader, Department of Pedodontics and Preventive Dentistry, Sree Balaji Dental College and Hospital, Chennai **Corresponding Author:** Dr. Hariharavel VP, Associate Professor, Department of Dentistry, Andaman and Nicobar Islands Institute of Medical Science, GB Pant Hospital, Port Blair, India.

Type of Publication: Original Research Paper

Conflicts of Interest: Nil

Abstract

Background: As prevalence of diabetes in children increases, so does its oral complications. The association between dental caries and diabetes has received lesser attention compared to periodontal diseases. This study was carried out to evaluate and detect the relationship between dental caries and physiological factors of saliva in children with uncontrolled type 1 diabetes mellitus.

Methods: This age and gender matched case control study was done on 45 children with uncontrolled type 1 diabetes mellitus and 45 unaffected school going children selected by simple random sampling. Type III clinical examination was carried out for DMFT status. Unstimulated whole saliva was collected and flow rate and pH was measured. Stimulated whole saliva was collected after the subject chewed paraffin wax and buffering capacity of saliva was calculated according to manufacturer's instruction. **Results** :Mean DMFT score was higher in uncontrolled type 1 diabetic children but unstimulated salivary flow rate, stimulated salivary flow rate, salivary pH and buffering capacity of saliva was lower than unaffected school going children and statistically significant.

Conclusions: High caries experience in this study population could be mainly related to changes in saliva, such as reduced salivary flow rate, buffering effect and pH, induced by poorly controlled diabetes.

Keywords: Diabetic children, Dental caries, Saliva, Type 1 diabetes mellitus

Introduction :Saliva is the main biological fluid bathing the oral microbiota in the oral cavity and it is the medium of the salivary sediment system. Glucose substrate is rapidly cleared because of saliva. In vivo, clearance is enhanced in those oral sites where saliva is continually being replenished. Excessive fermentation and deficient saliva are the two overall factors that can work together to

affect the stability of the salivary pH. The buffering capacity of saliva is a property that has often shown an inverse correlation with caries prevalence and is largely due to salivary bicarbonate.[1]

Diabetes mellitus (DM) is a common, chronic, metabolic syndrome characterized by hyperglycemia as a cardinal biochemical feature. Nutritional treatment alone or in combination with appropriate insulin therapy averts or relieves symptoms of hyperglycemia in diabetic patients. Moreover, nutritional practices may influence the development of long term complications of diabetes.[2] In the diabetic patients the dietary recommendations concerning carbohydrates stress the omission of sucrose in the diet. Therefore, one could expect the dietary habits of diabetics not to favour the multiplying of cariogenic bacteria and the subsequent development of carious lesions.[3] This is surprising in view of the fact that the lower intake of refined carbohydrates, especially sucrose, and high protein content of the diet make the dietary habits of diabetic subjects clearly less cariogenic than among non-diabetics.[4] Diabetic adults usually present altered salivary secretion that can cause disorders of hard and soft tissues of the mouth. However, in children there was no agreement in results that relate to alterations of salivary chemical composition and oral health.[5]

In 1989 and 1992, Twetman et al found an association of metabolic control in children with type 1 DM, with elevated rate of dental caries. Twetman et al observed that uncontrolled levels of blood glucose levels affects salivary factors such as flow rate, buffer capacity, glucose content and levels of acidogenic bacteria.[6] It was not generally agreed that diabetics are at greater risk of developing caries than healthy individuals. Results of cross-sectional studies, in which diabetics had been compared with healthy controls had demonstrated higher, similar, and lower caries prevalence in diabetics than in controls.[7] **Objectives:** This study was carried out to evaluate and detect the relationship between caries index and physiological factor (salivary flow rate, pH, and buffering capacity) in children with uncontrolled type 1 DM.

Methods

Study setting and study population: This study was carried out as case control study among 90 school going children in (area) for a period of months/years). The participants were divided into 45 cases who were uncontrolled type 1 diabetic children aged 12-18 years old children with capillary glucose value >130 mg/ml were selected.[8] The remaining 45 children were controls, who were normal school going children in the same age group. Sample size and sampling: All the type 1 diabetes mellitus children enrolled in our tertiary care hospital were included in the study as cases. They were age and gender matched with school going children in the schools attached to the field practice area of our hospital. Since 45 type 1 diabetes mellitus children were enrolled, 45 age and gender matched controls were selected from the school by simple random sampling.

Selection criteria: Children with absence of active disease, no history of drug treatment or therapy within previous months and no history of diabetes were selected as controls. The exclusion criteria used for both groups were the presence of systemic conditions that could influence salivary gland physiology, psychotropic drugs use, smokers, illicit drugs use, and alcohol use.[8]

Ethical approval and informed consent: Approval was obtained from the Institutional Ethics Committe prior to the commencement of the study. Each participant and their parent/ guardian were explained in detail about the study and informed consent was obtained from both the participant and their parent prior to the data collection.

Data collection: The examination for dental caries was carried out by single trained examiner using DMFT

index.[9] Type III oral examination was conducted, under natural light. Radiograph was not obtained. The data was entered on a structured interview schedule. Saliva samples were collected from all the groups by spitting method at least 2 hours after breakfast between 10am to 2pm.[10-12] Salivary properties estimated were:

- 1. Flow rate
- 2. pH
- 3. Buffering capacity

Unstimulated whole saliva was collected and the flow rate was calculated.[10] Immediately after collection, pH was measured by a manual pH meter.[3] Buffering capacity of saliva was determined by Saliva-Check Buffer as per manufacturer's instruction.

Data analysis: Data was entered and analyzed using SPSS Inc.;Chicago, IL, USA, Version 17.0.The results obtained were tabulated and statistically analysed by Mann – Whitney Test.

Results: Results obtained and estimated values are as follows (Table 1, 2 and 3). The mean DMFT score for uncontrolled type 1 diabetic children was 2.82 and for unaffected school going children was 1.46 respectively. The mean DMFT score for uncontrolled type 1 diabetic children was higher when compared with unaffected school going children and statistically significant. (Table 3)

The mean stimulated salivary flow rate for uncontrolled type 1 diabetic children and unaffected school going children was 3.92ml/5min and 5.00ml/5min respectively; and the mean unstimulated salivary flow rate was 1.33ml/5min and 1.90ml/5min respectively. Both the values were statistically significant. (Table 3)

The mean pH for uncontrolled type 1 diabetic children and unaffected school going children was 6.38 and 6.88 respectively. The value was statistically significant. (Table In the present study, 18 uncontrolled type 1 diabetic children had very low buffering capacity and 27 had low buffering capacity and 27 unaffected school going children had normal/high buffering capacity and 18 had low buffering capacity respectively. The mean buffering capacity for uncontrolled type 1 diabetic children was 5.73 and for unaffected school going children was 9.80 respectively. The value was statistically significant. (Table 3)

Discussion: The virulence of Streptococcus mutans to initiate dental caries depends on its ability to produce adhesive extracellular polysaccharides and to release organic acids which demineralize tooth substance. The organic acids involved are end products of the metabolism of fermentable substrates, especially sucrose, by the bacteria.[4]

The more frequent the intake of sucrose, with the consequent production of acid, the more disastrous is the effect upon tooth substance. To avoid hypoglycemic shock, however, the diabetics may have to resort on occasions to the frequent use of sucrose, thus, creating an oral environment which facilitates the demineralization of tooth substance commonly referred to as dental caries. In other words, diabetic patients may have more frequent meals than normal subjects and repeated intakes of even small amounts of carbohydrates may be cariogenic.[4]

It is well known that oral diseases can be caused by a number of factors of the oral cavity, with microorganisms being one of the main factors. However, diabetes may cause alterations in physiochemical properties of saliva, which may contribute to an increased pathogenic bacterial number. A slow flow rate also affects the oral flora and alters saliva composition.[5]

In the present study, uncontrolled type 1 diabetic children had higher DMFT values than the unaffected school going children and the observations were statistically significant.

3)

The findings obtained in the present study was similar to that of Ivana Maria Saes Busato et al[8] and Maria Elena Lopez et al[5] but Azza Tagelsir et al[13] reported that diabetic children had higher DMFS/DMFT values than the non-diabetic controls although the difference was not statistically significant.

Increased blood glucose level causes micro vascular changes which in turn produces basement membrane alteration in salivary gland leading to increased leakage of glucose from ductal cells which results in salivary and crevicular fluid hyperglycemia. Increased glucose in crevicular fluid causes decreased fibroblastic activity which in turn produces increased plaque accumulation which causes dental decay and periodontal diseases.[6]

Previous studies showed that the rate of decay in diabetics was higher than the healthy persons, which was due to high consumption of carbohydrates and lack of cooperation during treatment at younger age.[14] Increase in incidence of dental caries has been found in association with uncontrolled or poorly controlled DM in both humans and experimental animals.[15] The level of metabolic control and traditional caries risk markers were important factors for caries development in type 1 DM children.[16]

Restriction of pure carbohydrate consumption among diabetics had a favourable effect in promoting a lower caries rate/frequency.[17,18] However other studies report that the rate of decay was not lower than the control group, due to poor compliance of diabetic patients regarding their treatment and diet.[14] If patients with type 1 DM had adequate control of blood glucose levels, their salivary and caries data does not differ from that of healthy controls.[19]

In the present study mean unstimulated salivary flow rate for cases and controls was 0.26 ml/min and 0.38 ml/min respectively and was statistically significant similar to J

© 2019 IJDSIR, All Rights Reserved

Siudikiene et al.[20] Range of unstimulated salivary flow rate varies from 0.15 to 0.79 ml/min for DM and 0.25 to 1.06 ml/min for normal children.[3,5,7,8,12,19-21] Stimulated salivary flow rate for cases and controls obtained from the present study was 0.78 ml/min and 1.00 ml/min and it was statistically significant. This value was in accordance with Ivana Maria Saes Busato et al.[8] Range of stimulated salivary flow rate varies from 0.86 to 1.47 ml/min for DM and 1.22 to 1.62 ml/min for normal children. [3,5,7,8,12,19-21]

Salivary hypofunction in diabetic patients may be a response to inadequate metabolic control of the diabetes.[8] In diabetic subjects, both the subjective sensation of dry mouth and the quantification of hyposalivation associated with diabetic were neuropathy.[22] Elevated fasting blood glucose concentrations were associated with significant reductions in resting salivary flow rates and high salivary glucose.[12,22]

In the present study pH of saliva was 6.3 and 6.88 for cases and controls respectively and it was statistically significant. The value was in accordance with study done by Gamze Aren et al.[21] Studies reported pH ranges from 6.43 to 7.3 for DM and 6.9 to 7.4 for normal children.[3,5,7,19,21]

Buffering capacity of saliva of the cases was significantly lower than controls and it was in accordance with study done by Gamze Aren et al[21] who reported statistically significant value between normal and diabetic group with regards to buffering capacity of saliva. Buffer capacity ranges from 4.21 to 4.92 for DM and 4.55 to 5.1 for normal.

DM individuals have lower pH and less buffering capacity, because more glucose is released into the saliva and metabolized to lactic acid by plaque.[21] Salivary hyperglycemia produces increased acidogenic salivary

flora which results in decreased salivary pH. This increases acidophilic bacteria which cause increased dental decay and periodontal diseases.[6] The acidic pH in diabetic children may be associated either to microbial activity or to a decrease of bicarbonate with flow rate. Salivary glands act as filters of blood glucose that would be altered by hormonal or neural regulation.[21]

Long term glucose leakage into saliva is likely to increase the metabolic activity of the oral microflora. Because of an increased acidurity such changes in the natural balance of the dental biofilm may play an important role in preparing the local micro environment for caries.[20] Furthermore, metabolic control of DM had been shown to be related to factors modifying caries occurrence, such as reduced salivary flow, growth of oral yeasts, counts of mutans streptococci and of lactobacilli. DM as well as dental caries are chronic diseases and require constant, effective daily self-care.[23]

Maintaining metabolic control of type 1 DM in adolescents is a significant challenge for health professionals. Glycemic control has a modifying effect on the relationship between dental caries and salivary factors in young patients. Stimulated salivary flow reduction had been observed in type 1 DM patients and was considered to be a risk factor for dental caries. Poor metabolic control prevalence in type 1 DM adolescents was 76%, demonstrating that maintaining metabolic control during adolescence was complex.[8] There was association between poor metabolic control and dental caries.[16,23,24] The sample size might have interfered in the results, making it difficult to indicate an association between poor metabolic control and dental caries experience.[8] Normal salivary function is essential to the preservation of the integrity of teeth and oral soft tissues. When treating patients with diabetes, practitioners should be alert to complaints of dry mouth and signs of decreased salivary function. Hyposalivation may be indicative of poor glycemic control in some of these patients.[22]

Patients with DM appear to lack important knowledge about oral health complications associated with the disease. Children and adolescents with less good controlled type 1 DM may exhibit uncontrolled caries development in spite of extensive preventive efforts.[16] It was also known, and worth mentioning here, that the prevalence and clinical features of DM vary from one ethnic group to another. The conflict of the results referred to myth, therefore, be more apparent than real, the variability being related to both genetic and environmental factors such as over nutrition.[4]

Dental management strategies for patients with diabetes should be individualized with respect to patient needs and should possibly include more frequent visits to a dentist and oral hygienist, comprehensive medical and drug-use histories, dietary evaluations and counseling, smoking cessation recommendations, topical fluoride application, and instructions for proper oral hygiene.[22]

Conclusion

The present study suggested that high caries experience in this study population could be mainly related to changes in saliva, such as reduced salivary flow rate, buffering effect and pH, induced by poorly controlled DM. In particular, present study indicated that children with poorly controlled diabetes should take better care of their oral hygiene. No Indian studies were available for comparison.

By extending the study to other regions of the country, the impact of DM on the dental health of diabetic children could be studied with additional confounding factors entering the data analysis, such as differences in social background of the population, fluoride levels in drinking water, etc.

© 2019 IJDSIR, All Rights Reserved

References

- Kleinberg I. A mixed-bacteria ecological approach to understanding the role of the oral bacteria in dental caries causation: an alternative to streptococcus mutans and the specific-plaque hypothesis. Crit Rev Oral Biol Med 2002;13(2):108-25.
- Ramin Alemzadeh, David T Wyatt. Diabetes Mellitus in Children. In: Kliegman, Stanton, St Geme, Schor, Behrman, editors. Nelson Textbook of paediatrics, 19th edition. Elsevier, Saunders; 2011. p.1946-67.
- Tenovuo J, Alanen P, Larjava H, Viikari J, Lehtonen OP. Oral health of patients with insulin-dependent diabetes mellitus. Scand J Dent Res 1986 Aug;94(4):338-46.
- Al-Khateeb TL, Al-Amoudi NH, Fatani HH, Mira SA, Ardawi MS. Periodontal diseases and caries experience of diabetic patients in an Arabian community. Saudi Dent J 1990; 2: 91-5.
- López ME, Colloca ME, Páez RG, Schallmach JN, Koss MA, Chervonagura A. Salivary characteristics of diabetic children. Braz Dent J 2003; 14: 26-31.
- Amna Qureshi, Ambrina Qureshi, Hina Qureshi, Ayyaz A Khan. Blood glucose level, salivary pH and oral bacterial count in type 1 diabetic Children. Infect Dis J 2007; 16: 45-8.
- Karjalainen KM, Knuuttila ML, Käär ML. Relationship between caries and level of metabolic balance in children and adolescents with insulindependent diabetes mellitus. Caries Res 1997; 31: 13-18.
- Saes Busato IM, Bittencourt MS, Machado MA, 8. Grégio AM. Azevedo-Alanis LR. Association between metabolic control and oral health in adolescents with type 1 diabetes mellitus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2010; 109:e51-6.

- Soben Peter. Survey procedures. In: Soben Peter, editor. Essentials of preventive and community dentistry, 2nd edition. New Delhi. Arya Publishing House; 2004. p.616-44.
- Navazesh M. Methods for collecting saliva. Ann N Y Acad Sci 1993; 20:72-7.
- Leon M Silverstone. Dental caries pathogenesis. In: Ray E.Stewart, Thomas K.Barber, Kenneth C.Troutman, Stephen H.Y.Wei, editors. Pediatric Dentistry: Scientific foundation and clinical practice. London. The C.V. Mosby Company; 1982. p.535.
- Karjalainen KM, Knuuttila ML, Käär ML. Salivary factors in children and adolescents with insulindependent diabetes mellitus. Pediatr Dent 1996; 18: 306-11.
- Tagelsir A, Cauwels R, van Aken S, Vanobbergen J, Martens LC. Dental caries and dental care level (restorative index) in children with diabetes mellitus type 1. Int J Paediatr Dent 2011; 21: 13-22.
- Alavi AA, Amirhakimi E, Karami B. The prevalence of dental caries in 5 – 18-year-old insulin-dependent diabetics of Fars Province, southern Iran. Arch Iran Med 2006; 9: 254–60.
- 15. Rees TD. The diabetic dental patient. Dent Clin North Am 1994; 38: 447-63.
- 16. Twetman S, Johansson I, Birkhed D, Nederfors T. Caries incidence in young type 1 diabetes mellitus patients in relation to metabolic control and cariesassociated risk factors. Caries Res 2002; 36: 31-5.
- Tavares M, Depaola P, Soparkar P, Joshipura K. The prevalence of root caries in a diabetic population. J Dent Res 1991; 70: 979-83.
- Usha C, Sathyanarayanan R. Dental caries A complete changeover (Part I). J Conserv Dent 2009; 12: 46-54.

- Swanljung O, Meurman JH, Torkko H, Sandholm L, Kaprio E, Mäenpää J. Caries and saliva in 12–18year-old diabetics and controls. Scand J Dent Res 1992; 100: 310-3.
- Siudikiene J, Machiulskiene V, Nyvad B, Tenovuo J, Nedzelskiene I. Dental caries increments and related factors in children with type 1 diabetes mellitus. Caries Res 2008; 42: 354-62.
- Aren G, Sepet E, Ozdemir D, Dinççağ N, Güvener B, Firatli E. Periodontal health, salivary status and metabolic control in children with type 1 diabetes mellitus. J Periodontol 2003; 74:1789-95.
- Moore PA, Guggenheimer J, Etzel KR, Weyant RJ, Orchard T. Type 1 diabetes mellitus, xerostomia, and salivary flow rates.
 Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001; 92: 281-91.
- 23. Bakhshandeh S, Murtomaa H, Vehkalahti MM, Mofid R, Suomalainen K. Dental findings in diabetic adults. Caries Res 2008; 42:14-8.
- 24. Miralles L, Silvestre FJ, Hernández-Mijares A, Bautista D, Llambes F, Grau D. Dental caries in type 1 diabetics: influence of systemic factors of the disease upon the development of dental caries. Med Oral Patol Oral Cir Bucal 2006; 11: E256-60.

Cases	Ν	N	lean	Std. Deviation	
	Statistic	Statistic	Std Error	Statistic	
DMFT	45	2.8222	0.37375	2.50716	
Unstimulated salivary					
flow rate per 5 min	45	0.3311	0.02910	0.19519	
Stimulated salivary	45	3.9267	0.07617	0.29124	
flow rate per 5 min					
pH of saliva	45	6.3867	0.04341	0.29124	
Buffer capacity of					
saliva	45	5.7333	0.22563	1.5137	

Table 2- Descriptive Statistics – Controls

Table 1- Descriptive Statistics – Cases

Tables & Figures

Cases	Ν	M	lean	Std. Deviation	
	Statistic	Statistic	Std Error	Statistic	
DMFT	45	1.4667	0.22608	1.51658	
Unstimulated salivary					
flow rate per 5 min	45	1.9067 0.03587		0.24064	
Stimulated salivary	45	5.000	0.07220	0.48430	
flow rate per 5 min					
pH of saliva	45	6.8800	0.02871	0.19259	
Buffer capacity of					
saliva	45	9.8000	0.23484	1.57538	

Test	Groups	N	Mean Rank	Sum of Ranks	Mann Whitney U	Z	Sig(2-talled)
DMFT	Diabetic Group Unaffected Group Total	45 45 90	52.23 38.77	2350.50 1744.50	709.500	-2.526	0.0001
Unstimulated salivary flow rate per 5 min	Diabetic Group Unaffected Group Total	45 45 90	25.00 66.00	1125.00 2970.00	90.000	-7.486	0.0001
Stimulated salivary flow rate per 5 min	Diabetic Group Unaffected Group Total	45 45 90	26.10 64.90	1174.50 2920.50	139.500	-7.069	0.0001
pH of saliva	Diabetic Group Unaffected Group Total	45 45 90	25.73 66.27	1158.00 2937.00	123.000	-7.227	0.0001
Buffer capacity of saliva	Diabetic Group Unaffected Group Total	45 45 90	24.93 66.07	1122.00 2973.00	87.000	-7.586	0.0001

Table 3 – Mann – Whitney Test

*p<0.05 statistically significant