ORIGINAL RESEARCH

Comparison of Salivary Flow Rate, pH, Buffering Capacity, and Secretory Immunoglobulin A Levels between Children with Early Childhood Caries and Caries-free Children

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Abstract

Background and objectives: Early childhood caries (ECC) is the most common chronic disease of childhood in many developing countries, which is associated with local, systemic, psychological, and social consequences. Multiple variables are shown to be associated with an increased risk of ECC. The knowledge regarding the role of saliva in the pathophysiological process of ECC still remains controversial and unexplored. Scanty studies focused on probing the role of salivary flow rate, pH, buffering capacity, and secretory immunoglobulin A (sIgA) in unstimulated whole saliva of children with ECC and children without ECC.

Aim: To assess the salivary flow rate, pH, buffering capacity, and sIgA levels in children with ECC and caries-free children and to evaluate their role in caries risk assessment.

Materials and Methods: The study was carried out among 64 children aged between 24 and 71 months. Clinical examinations were carried out according to the criteria by the World Health Organization, and carious status was recorded. Subjects were categorized as group I with ECC (dmfs-Decayed, Missing or Filled Surfaces (Deciduous dentition) of ≥5), and group II included children without ECC (dmfs = 0). Unstimulated whole salivary samples were collected in a sterile vial and stored at −70°C by draining. Estimations of salivary flow rate, pH, buffering capacity, and sIgA levels were done. Digital pH meters were used for the estimation of pH and buffering capacity. A human IgA enzyme-linked immunosorbent assay (ELISA) kit was used to estimate sIgA levels. Statistical software IBM Statistical Package for the Social Sciences (SPSS) statistics 20.0 (IBM Corporation, Armonk, New York, United States of America) was used to analyze the data.

Results: The mean salivary flow rate decreased in group I children with ECC (0.15 ± 0.05) when compared to group II children without ECC (0.67 ± 0.14), which was statistically significant. In caries active children, no statistically significant correlation was found between salivary flow rate and the dmfs scores [*r*-value (−0.247)] and *p*-value (0.147). The mean level of salivary pH is decreased in group I children with ECC (4.65 ± 0.4) when compared to group II children without ECC (7.28 ± 0.18). In the caries active group, the levels of salivary pH decrease as the dmfs scores increase, and this correlation is found to be statistically significant (*r*-value of 0.547 and *p*-value of 0.002). The mean level of buffering capacity is decreased for caries-active children (5.45 ± 0.49) when compared to caries-free children (8.94 ± 0.42). In caries active children, as the dmfs scores increase, the salivary buffering capacity decreases, and this correlation is found to be not statistically significant (*r*-value of −0.334 and *p*-value of 0.161). The mean levels of sIgA in group I children with ECC were higher (10.61 ± 0.90) than that in group II children without ECC (6.11 ± 1.22). In the caries-active group, the salivary sIgA levels were comparatively higher than in the caries-free children. As the dmfs scores increase, the level of the sIgA increases in caries-active children, and this correlation is noted to be highly statistically significant (*r*-value of 0.769 and *p*-value 0.008).

Conclusion: Children with ECC showed decreased salivary flow rate, pH, buffering capacity, and increased sIgA levels, while children without ECC showed increased salivary flow rate, pH, buffering capacity, and decreased sIgA levels. The salivary parameters, such as salivary flow rate and buffering capacity, showed no correlation with the dmfs score, while salivary pH and sIgA levels have a positive correlation in caries-active children.

Keywords: Buffering capacity, Early childhood caries, Salivary flow rate, Salivary immunoglobulin A, Salivary pH.

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Introduction

Good oral health is an essential part of overall health. It affects the quality of someone's life, including self-esteem, learning, and employment. Active and unchecked tooth decay causes poor oral and general health in many children. Dental caries is the most widespread and multifactorial infectious disease of mankind, which causes the dissolution of tooth minerals. The effects of an untreated carious tooth include pain, infection, nutritional insufficiencies, and even learning and speech problems. Dental decay remains a serious global health issue that affects people of all ages but is especially problematic for children despite advancements in the science of oral disease. In 2017, Pitts et al. described dental caries as a noncommunicable, dynamic, diet-modulated, and biofilm-mediated disease that causes a net mineral loss of dental hard tissue. It is influenced

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by environmental, behavioral, psychological, and biological factors.[1]

In many developing countries, the most common chronic childhood disease is early childhood caries (ECC), which is associated with local, systemic, psychological, and social consequences. ECC is a virulent form of tooth decay affecting the primary teeth soon after its eruption. ECC is defined by the American Academy of Pediatric Dentistry (AAPD) as the presence of one or more decayed, missing, or filled tooth surfaces in any primary tooth in a child of 71 months of age or younger.[2] ECC is caused by *Streptococcus mutans (S. mutans)* that ferment dietary carbohydrates and produce acids, which will lead to demineralization of tooth enamel. Because children had already developed caries by the time they were 3 years old, the AAPD recommends that the first dental visit occur no later than 6 months after the primary tooth eruption. The cyclic nature of ECC makes the affected children remain endangered throughout childhood, even when preventive measures are accessible. Primary etiological factors of ECC are dental plaque, *S. mutans*, feeding patterns, brushing habits, salivary factors, sugars, and oral clearance of carbohydrates. Secondary etiological factors include immunological factors, tooth maturation defects, race and ethnicity, socioeconomic status, dental knowledge, and stress. Proper oral hygiene techniques promote the oral health of children by modifying or eliminating risk factors for the development of ECC. The expenses associated with treating tooth decay and its complications place a significant financial strain on both individuals and society at large.[2]

A broad and complex relationship between saliva and caries has been recognized, and this subject is one of the most challenging and exciting within the field of cariology. Saliva serves as a body's health mirror with the potential to detect many diseases. Because it alters the environment inside the mouth cavity, it is essential to maintain oral homeostasis. The natural protection mechanism inherent to saliva controls the carious process. Salivary flow rate, dilution, pH, buffering, and remineralization capacitance are important variables that influence and control the caries process's progression and regression. Saliva is easy to collect, has a low-risk profile, is tolerable to the patient (especially the pediatric population), and is less infectious for a healthcare provider. Early screening of caries development will help to identify children who are at risk. Hence, this study considered salivary factors like pH, flow rate, secretory immunoglobulin A (sIgA), and buffering capacity.

The most important function of saliva in caries is the elimination of bacteria and food debris from the mouth. The knowledge of normal salivary flow is essential to provide home-care instructions. The unstimulated salivary flow rate is about 0.3 mL/minute on average. Consequently, the oral half-life of inert substances suspended in the saliva is only a few minutes, which is significantly less than the oral microorganisms' mean generation time. The primary factor affecting the composition of saliva is the flow rate, which may change with the type, intensity, and duration of the stimulus. Saliva's pH and buffering ability are influenced by the amount of bicarbonate present. Tooth demineralization happens when saliva's actual pH stays below the critical pH for an extended period of time. Below the critical pH, which is about 5.5 for enamel, saliva loses its phosphate and calcium saturation, allowing the hydroxyapatite in the dental enamel to disintegrate. Buffering capacity mainly depends on bicarbonate concentration, which is crucial for maintaining the pH of saliva and for tooth remineralization. In the case of unstimulated whole saliva, sIgA has a specific role. The concentration of IgA depends on hormones, emotional moods, physical activity, and salivary flow rate. Salivary sIg includes IgA, IgG, and IgM. Of the total immunoglobulin count in saliva, salivary IgA makes up 60%, which has antibacterial action by blocking the adhesion of bacteria and neutralizing the toxins and enzymes produced by the bacteria. Thus, it reduces the agglutination and hydrophobicity of bacteria.

The knowledge regarding the role of saliva in the pathophysiological process of ECC still remains controversial and unexplored. Only a very few research have looked into the value of saliva and its immunologic components in the diagnosis and evaluation of caries risk. The diagnosis of caries may be aided by the detection of quantitative and/or qualitative changes in the saliva. The assessment of salivary components is the best caries risk assessment tool. Simple salivary tests can be employed to determine children who are susceptible to dental caries and thereby evolve preventive strategies.[3,4] This study was focused on probing the role of sIgA levels, buffering capacity, pH, and salivary flow rate in unstimulated whole saliva of children with ECC and caries-free children. The study objectives were:

- To measure and compare the salivary flow rate, pH, buffering capacity, and sIgA levels in unstimulated whole saliva between children with ECC and those without ECC.
- To identify quantitative alteration in the saliva of children with ECC and without ECC.
- To evaluate the role of salivary flow rate, pH, buffering capacity, and sIgA levels in the prediction of ECC.

Materials and Methods

After attaining informed written consent from guardians and parents, children between the ages of 24 and 71 months who reported to the Department of Pedodontics at the Azeezia College of Dental Sciences and Research, Kollam, were chosen. The Institutional Ethics Committee gave its approval to the study (AEC/REV/2018/835). They provided a thorough medical history and information about their dental and dietary behaviors. Using the Decayed, Missing or Filled Surfaces (Permanent dentition) (DMFS) system, a comprehensive clinical examination was conducted to determine the children's carious status. Samples were grouped into two groups; group I: children with ECC (dmfs score of ≥5) and group II: children without ECC (dmfs score = 0). The sample size was calculated as 32 for each group using the formula:

$$n = \frac{\left(z1-a/2+z1-\beta\right)^2\left(S1\right)2+\left(S2\right)^2}{\left(x1-x2\right)^2}$$

There were 32 samples in each group; that is, 64 was the total sample size.

The sample size includes children aged between 24 and 71 months with food lodgment complaints, discolored teeth, and pain. Children not having any positive medical history, those who have not had any antibiotic and analgesic in the past 2 weeks have been involved in the present research, while children with systemic diseases, mentally and physically compromised children, patients who have been on medication, children on supplementation of fluoride and participants not giving consent have been excepted.

Dental Examination

Examination of dental caries was carried out using the dental explorer and mouth mirror by the researcher. The guidelines and procedures followed by the World Health Organization were

followed. On the data collection form, the decayed, missing, and filled teeth scores have been noted. The selected children were grouped into two. Children aged between 24 and 71 months fulfilling the criteria have been included under group I with ECC (dmfs of ≥5), and group II includes children without ECC (dmfs = 0).

Saliva Sample Collection

Unstimulated mid-morning whole samples of saliva were collected from the participants by the draining method after examination. Samples were taken between 10 and 11:30 am to control for circadian variation. Kids were told not to eat or drink anything for at least 1 hour prior to the sample collection. Before being collected, participants were instructed to thoroughly rinse their mouths with water for 10 minutes. After that, they were forced to sit upright in the dental chair and given some time to decompress. There were no salivating agents used. The unstimulated saliva was allowed to accumulate for a minimum of five minutes and noted for salivary flow rate estimation. A total of 2 mL of samples were drained directly from the floor of mouth utilizing a disposable syringe and transferred to an appropriately labeled preweighed graduated sterile vial. After collection, the saliva was placed into ice-chilled boxes and sent to the immunochemistry laboratory. Following that, the samples were kept at –70°C in a pharmaceutical refrigerator until analysis. The saliva that was collected was used to estimate the levels of sIgA, pH, salivary flow rate, and buffering capacity.

Salivary Flow Rate

After collecting samples by draining method, the estimation of unstimulated salivary flow rate was done by using the formula stated below,

$$\frac{\text{Post collection weight} - \text{pre collection weight}}{\text{Collection period}}\ \text{gm/min}$$

Preweighed vials containing saliva were weighed after five minutes of sample collection and recorded in data form. Later, the estimation of the salivary flow rate was done using the above formula. The values thus obtained were converted from gm/minute to mL/minute to express the unstimulated whole salivary flow rate. Accordingly, each sample from both study groups was subjected to an estimation of the salivary flow rate, and the values were recorded.

Salivary pH

A handheld digital pH meter was used to measure the salivary pH directly (Nexqua Dew Digital LCD Display Portable Total Dissolved Solids Meter). The pH meter had a resolution of 0.1 and a measuring range of 0–14. First, a pH 7 buffer was used for calibration, then a pH 4 buffer (if an acidic sample is anticipated) or a pH 9 buffer (if the sample is anticipated to be basic). After calibrating the pH electrode, samples of saliva are simply submerged in it in a container. A few seconds are then allowed for the digital reading to stabilize before the salivary pH value is determined. Before immersing the sample again, the electrode's submerged section is cleaned with filter paper. Similarly, an estimation of salivary pH was done for each sample of both groups, and the values were recorded.

Buffering Capacity

Using the Ericsson method, salivary buffering capacity was calculated. The effectiveness of a buffer in withstanding pH variations is gauged by its buffering capacity. Typically, the buffer capacity is stated as the gram-equivalents of strong acid and base, which need to be added to one liter of the solution in order to change the pH by 1 unit. We measured and recorded the saliva's initial pH. Next, 1.5 mL of 5 mmol/L HCl and 0.5 mL of saliva were combined in a centrifuge tube. After giving it a good shake to thoroughly combine the saliva and HCl, the mixture is centrifuged for 1 minute, allowed to stand for 10 minutes, and the pH is finally measured. The amount of acid added to the pH change that is formed is the quantitative expression of buffering capacity. The buffer capacity was computed *via* the subsequent formula:

$$\text{Buffering capacity}(\beta) = \frac{\Delta B}{\Delta pH}$$

ΔB = 1 L of buffer solution's pH can be changed with 1 gm of strong acid.

ΔpH = pH change caused by the strong acid addition.

All the samples from both groups were subjected to estimation of salivary buffering capacity, and the values were recorded.

Salivary Secretory Immunoglobulin A

Saliva samples were centrifuged for 20 minutes at 1000 rpm at 2–8°C to eliminate cellular debris and to decrease the saliva turbidity that can negatively affect the accuracy of the analysis. The supernatant thus obtained is used for immunological assay. A total of 1000 µL of all salivary samples were transferred to appropriately labeled 1.5 mL sterile Eppendorf tubes using a micropipette. Salivary sIgA was quantified by the enzyme-linked immunosorbent assay (ELISA) method using Human IgA (Immunoglobulin A) ELISA kit Catalog No.: EH0415.

Principle of the Assay

Sandwich "enzyme-linked immune-sorbent assay technology was used in the development of this kit. On 96-well plates, the capture antibody was precoated. Biotin-conjugated antibodies were also employed as detection antibodies. After adding the standards, test samples, and biotin-conjugated detection antibodies, the wells were washed using a wash buffer. After adding horseradish peroxidase (HRP)–streptavidin, unbound conjugates were eliminated using a wash buffer. The HRP enzymatic reaction was seen using 3,3′,5,5′-tetramethylbenzidine (TMB) substrates. TMB was catalyzed by HRP to yield a blue product; the addition of an acidic stop solution caused the product to turn yellow. The yellow density and the desired amount of sample captured in the plate are directly related. To find the desired concentration, use a microplate reader to read the optical density (OD) absorbance" at 450 nm.

Measurement of Secretory Immunoglobulin A

The Salivary sIgA was quantified by using a Human IgA ELISA Kit. Catalog No.: EH0415 according to manufacturer's instruction. All reagents, working standards, and all salivary samples from both groups were prepared. Excess strips from the microplate frame were removed and returned to the foil pouch involving the desiccant pack and stored at 4°C. Wash the microplate two times before adding the standard, samples, and seven control wells. A total of 50 µL of standards and all samples were added, sealed, and incubated at 37°C for 90 minutes. The covers were removed, and the contents were. Plates were washed two times with wash buffer and blotted against clean paper towels to remove excess liquid. Without coming into contact with the sidewall, 50 µL of biotin-labeled antibody working solution was added to the bottom of the well. It was then sealed, allowed to incubate at 37°C for 60 minutes,

and then rinsed three times. A total of 50 µL of HRP-streptavidin conjugate (SABC) was added and incubated for 30 minutes. Before adding 90 µL of TMB substrate, plates were washed five times and incubated in the dark for 10–20 minutes. Depending on the actual color variation, the time of reaction may be shortened or prolonged. A total of 50 µL of stop solution was added into each well, which turned the blue color immediately to yellow. OD absorbance has been registered at 450 nm in the Microplate Reader immediately. Curve Expert 1.3 software was used to estimate sIgA levels. All the samples were subjected to estimation of sIgA levels, and the values were recorded.

Statistical Analysis

For statistical analysis, all of the data were gathered, entered, and used in a Microsoft Excel datasheet. The data analysis was done using IBM Statistical Package for the Social Sciences (SPSS) statistics 20.0 (IBM Corporation, Armonk, New York, United States of America), a statistical software. This study used both descriptive and inferential statistical analyses. Outcomes on continuous measurements have been presented on mean ± standard deviation (SD). An independent *t*-test was employed to determine the significance of the study parameters between the groups. A value of 0.05 or less was regarded as statistically significant, with the threshold of significance set at $p < 0.05$. Statistical tools such as Pearson's correlation coefficient and analysis of variance (ANOVA) test were employed to ascertain the significance of the relationship and variations in the variables used for the study.

Results

The current study goal has been to compare and evaluate salivary parameters, including sIgA levels, buffering capacity, pH, and salivary flow rate. The study comprised 64 children ranging in age from 24 to 71 months. The children were divided into two groups: group I consisted of 32 children with ECC (dmfs of >5), and group II consisted of 32 children without ECC (dmfs = 0). Table 1 displays the subjects' baseline attributes. The significance of the salivary parameters between the two groups was determined using an independent *t*-test. An attempt was also made to use Pearson's correlation coefficient to determine the relationship between dmfs scores and different salivary parameters in children who were actively developing caries. ANOVA test was applied to ascertain the significance of variations among the group.

Salivary Flow Rate

In the current research, the salivary flow rate estimated among group I children with ECC ranged from 0.30 to 0.84 mL/minute, and for group II children without ECC was 0.10–0.34 mL/minute (Table 1). The mean salivary flow rate was reduced in group I children with ECC (0.15 ± 0.05) when compared to group II children without ECC (0.67 ± 0.14) with a *p*-value of <0.001 (Table 2). It was found that the difference was statistically significant. Salivary flow rate and dmfs scores did not show a statistically significant correlation in children with caries (*r*-value of −0.247 and *p*-value of 0.147) (Table 2).

Salivary pH

The salivary pH among group I children with ECC ranges from 3.70 to 5.90 mmol/L, and for group II children without ECC, 6.81–7.53 mmol/L (Table 1). Compared to group II children without ECC (7.28 ± 0.18), the mean salivary pH level is lower in group I children with ECC (4.65 ± 0.4), with a *p*-value of <0.001 (Table 2). The difference in salivary pH is observed to be statistically significant. In the caries active group, the levels of salivary pH decrease as the dmfs scores increase, and this correlation is found to be statistically significant (*r*-value of 0.547 and *p*-value of 0.002) (Table 3).

Salivary Buffering Capacity

The estimated range of salivary buffering capacity among group I children with ECC and group II children without ECC ranges from 4.40 to 6.40 Meq/pH/mL and 7.60–9.40 mEq/pH/mL, respectively (Table 1). The mean level of buffering capacity is reduced for caries-active children (5.45 ± 0.49) while compared with caries-free children (8.94 ± 0.42), a *p*-value of <0.001 as depicted in Table 2. In caries active children, as the dmfs scores increase, the salivary buffering capacity decreases, and this correlation is found to be not statistically significant (*r*-value of −0.334 and *p*-value of 0.161) (Table 3).

Table 1: Baseline characteristics of children in groups I and II

Baseline characteristics	*Age-group, gender, and dmfs scores*	*Group I: number of children with ECC (n)*	*Group II: number of children without ECC (n)*
Age	24–35 months	5	9
	36–47 months	12	11
	48–59 months	9	6
	60–71 months	6	6
Gender	Male	18	16
	Female	14	16
dmfs	5–6	9	0
	7–8	14	0
	9–10	9	0

Table 2: Comparison of mean levels of dmfs score and salivary flow rate, pH, buffering capacity, and IgA levels in group I—children with ECC and group II—children without ECC

Variables	*Group I (children with ECC)*	*Group II (children without ECC)*	*t-value*	*p-value*
dmfs score; mean ± SD	7.59 ± 0.90	0		
Flowrate (mL/minute); mean ± SD	0.15 ± 0.05	0.67 ± 0.14	−20.43	<0.001
pH (mmol/L); mean ± SD	4.65 ± 0.42	7.28 ± 0.18	−32.43	<0.001
Buffering capacity (mEq/pH/mL); mean ± SD	5.45 ± 0.49	8.94 ± 0.42	−31.14	<0.001
SIgA (mg/dL); mean ± SD	10.61 ± 0.90	6.11 ± 1.22	17.37	<0.001

*p-value of <0.05 is statistically significant; **<0.001 is statistically highly significant; #independent *t*-test

Secretory Immunoglobulin A

In the present study, salivary sIgA among group I children with ECC ranges from 9.40 to 12.50 mg/dL, and for group II children without ECC, 5.40–7.30 mg/dL (Table 1). The mean levels of sIgA in group I children with ECC was higher (10.61 ± 0.90) than that in group II children without ECC (6.11 ± 1.22), with a *p*-value of <0.001 as shown in Table 2. In the caries-active group, the levels of salivary sIgA were comparatively higher than in the caries-free children. As the dmfs scores increase, the level of the sIgA increases in caries-active children, and this correlation is noted to be highly statistically significant (*r*-value of 0.769 and *p*-value of 0.008 as shown in Tables 3 and 4 and Fig. 1).

Discussion

The most prevalent chronic oral disease in humans is dental caries. Dental caries remains a global health concern that affects people of all ages, particularly children, despite advances in the science of oral disease. As ECC is so common everywhere in the world, it is a serious public health concern. It has been proposed that the disease is multifactorial, impacted by both host and dietary factors. Furthermore, it is commonly known that saliva functions as a barrier against dental caries. The balance among cariogenic and noncariogenic microbial populations in saliva, along with the interaction of pathologic and protective factors, determine the course of dental caries. The most common salivary factors associated with dental caries are the aciduric/acidogenic bacteria and the acid production rate when glucose is present. Other endogenous factors include salivary characteristics, like the amount of saliva secreted in a provided time (flow rate), salivary pH, acid-neutralizing ability (buffering capacity), sIgA levels, etc. Saliva may hold the key to understanding why some kids experience ECC while others do not. As the studies used different patient selection criteria, different laboratory tests, and different sampling techniques, it was difficult to draw conclusions about the relationship between salivary components and dental caries.

Saliva that isn't stimulated is crucial for the oral cavity's overall health and well-being because it provides a powerful barrier against dental caries. Higher flow rates generally result in quicker clearance times and larger buffer capacities, which reduce microbial attacks. Hormonal, emotional, and other factors, as well as changes in salivary flow rate, can all affect the amount of salivary components. Although salivary flow rate is another significant salivary feature that may influence the dental caries process, not much research has been done on it due to its measurement being challenging in young children. The salivary flow rate is an independent factor that will affect other salivary defense factors. Salivary pH and buffering capacity are dependent variables on salivary Flow rate.

In the current research, the salivary flow rate estimated among group I children with ECC ranged from 0.30 to 0.84 mL/minute, and for group II children without ECC, it was 0.10–0.34 mL/minute. The mean salivary flow rate was reduced in group I children with ECC (0.15 ± 0.05) when compared to group II children without ECC (0.67 ± 0.14). The variation has been observed to be statistically important. There was no statistically significant correlation (*r*-value of −0.247 and *p*-value of 0.147) between salivary flow rate and the DMFS scores in children with active caries. The results of this research are as per the findings of studies performed by Preethi et al.[5] and Prabhakar et al.[3] Children with caries were found to have a slightly lower salivary flow rate than children without caries. Animireddy et al.,[6] in their research study, stated that the mean

Table 3: Correlation between dmfs score and salivary flow rate, pH, buffering capacity, and sIgA levels in group 1—children with ECC

	dmfs	
Salivary parameters	*r-value*	*p-value*
Salivary flow rate	−0.247	0.147
Salivary pH	0.507	0.002
Buffering capacity	−0.334	0.161
sIgA	0.769	0.008

r-value, correlation coefficient; *p*-value <0.001 is statistically significant

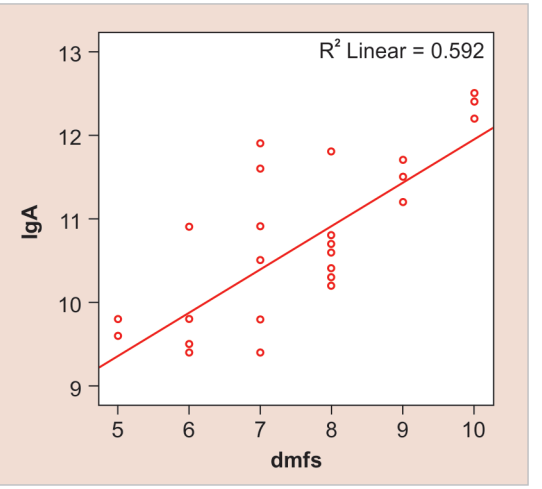

Fig. 1: Correlation between sIgA and dmfs score in group I children with ECC

Table 4: Association of salivary parameters with dmfs score in group I children with ECC

	dmfs score				
Parameters	*5–6*	*7–8*	*9–10*	*F-value*	*p-value*
Salivary flow rate (mL/minute)	0.15 ± 0.04	0.16 ± 0.06	0.11 ± 0.02	2.88	0.08
Salivary pH (mmol/L)	4.73 ± 0.37	4.76 ± 0.37	4.24 ± 0.40	5.14	0.01
Salivary buffering capacity (mEq/pH/mL)	5.58 ± 0.39	5.60 ± 0.43	4.87 ± 0.36	8.55	0.001
sIgA	9.82 ± 0.40	10.57 ± 0.70	11.81 ± 0.55	22.27	<0.001

*p-value of <0.05 is statistically significant; **<0.001 is statistically highly significant; #one-way ANOVA

salivary flow rate in children with ECC (0.19 ± 0.02) is lower than in caries-free children (0.43 ± 0.09). On the other hand, a study by Thaweboon et al.[7] found that children with caries-free and rampant cavities had similar mean salivary flow rates.

The mean level of salivary pH is reduced in group I children with ECC (4.65 ± 0.4) when compared to group II children without ECC (7.28 ± 0.18). The difference in salivary pH is observed to be statistically significant. In the caries active group, the levels of salivary pH decrease as the dmfs scores increase, and this correlation is found to be statistically significant (*r*-value of 0.547 and *p*-value of 0.002). Salivary pH rise activity was examined in both caries-free and caries-active naval recruits by Lamberts et al.[8] They discovered no significant correlation between salivary pH rise activity and caries experience, but they did find a significant positive correlation among the samples' bicarbonate content and minimum pH values.

The estimated range of salivary buffering capacity among group I children with ECC and group II children without ECC ranges from 4.40 to 6.40 mEq/pH/mL and 7.60–9.40 mEq/pH/mL, respectively. The Buffering capacity mean level is reduced for caries-active children (5.45 ± 0.49) while compared to caries-free children (8.94 ± 0.42). In caries active children, as the DMFS scores rise, the salivary buffering capacity decreases, and this correlation is found to be not statistically significant (*r*-value of −0.334 and *p*-value of 0.161). When compared to children who did not have dental cavities, the salivary buffering capacity of caries-active children was only marginally lower. The results of the current research corroborate with the observations of a study carried out by Gopinath and Arzreanne.[9] They reported that salivary viscosity, flow rate, pH, and buffering capacity have been decreased in subjects with higher dental caries. Research performed by Malekipour et al.[10] also reported the same findings, although the variations have been not statistically important. The study by Zhou et al.,[11] on the other hand, discovered that ECC children had a statistically greater salivary buffering capacity than children who were caries-free.

In the current research, the levels of salivary sIgA were comparatively greater in the caries-active children in comparison to the caries-free children. As the dmfs scores increase, the level of sIgA increases in caries-active children, and this correlation is noted to be highly statistically significant (*r*-value of 0.769 and *p*-value of 0.008). These results are consistent with the research conducted by Ranadheer et al.[12] According to their findings, children with a Decayed, Missing and Filled Teeth (Permanent dentition) (DMFT) score of ≥3 had significantly higher mean levels of sIgA compared to those with a score of 0. Due to the higher concentration of *S. mutans* in their whole saliva, children with dental cavities may have elevated levels of sIgA as a defense mechanism. It should be mentioned that Gornowicz et al.[13] discovered a rise in total salivary IgA in children with ECC.

Results of the previous studies conducted by de Farias and Bezerra[14] and Amoudi et al.[15] reported that IgA levels of mothers and children have a positive correlation. The relationship between total salivary sIgA and mutans antigen-specific SIgA was established by Pal et al.[16] They found that the high caries group had higher levels of *mutans*-specific sIgA but lower levels of total sIgA. Numerous comparable studies with a range of outcomes have been carried out in the past. Research by Cogulu et al.[17] revealed that patients with Down syndrome had significantly greater levels of salivary sIgA and a significantly lower prevalence of caries, supporting the theory that salivary sIgA may act as a barrier against dental caries. Whole salivary IgA levels were significantly higher in children who were free of dental caries than in children who were actively developing dental caries, according to research by Doifode and Damle.[18] This finding suggests that naturally occurring salivary IgA antibodies may be important in the immunological control of dental caries. According to Bagherian and Asadikaram,[19] there is a negative correlation between a decrease in caries activity and an increase in sIgA levels. This suggests that the induction of IgA immunoglobulin has a protective function. On the other hand, research by Shifa et al.[20] did not discover any connection between sIgA levels and dental caries. The mean IgA value of the caries-resistant group increased, but the findings have not been statistically important.

The cause of this controversial discrepancy in research could be traced to changes in sampling size, various environmental factors influencing the time, oral hygiene, diet, and saliva sample collection. Additionally, variations in salivary immunoglobulin concentration in various studies are influenced by hormonal factors, physical activity, emotional state, and salivary flow rate.

Conclusion

The current research was focused on probing the role of salivary flow rate, pH, buffering capacity, and sIgA levels among 64 children aged between 24 and 71 months. The children were divided into two groups: group I consisted of 32 children with ECC (dmfs of >5), and group II consisted of 32 children without ECC (dmfs = 0). The following findings were reached using this comparative observational study's framework:

- Compared to children without caries, there has been a decline in the mean levels of salivary flow rate, pH, and buffering capacity in children diagnosed with ECC.
- In children with ECC, the mean levels of sIgA were observed to be increased when compared with the caries-free children.
- Among children with ECC, as the DMFS score rises, the sIgA level was found to be increased, and a positive correlation existed.
- In caries-active children, no statistically important correlation has been observed among salivary flow rate, buffering capacity, and the dmfs scores, whereas a statistically significant correlation has been observed among salivary pH and the dmfs scores.

The results of this study indicate that there were notable changes in the salivary characteristics of children who were actively developing dental cavities. This suggests that the physiochemical properties of saliva can serve as markers of a child's dental health. More research with a bigger sample size is needed in order to extrapolate these results.

Clinical Significance

Early screening of caries development will help to identify children who are at risk. The diagnosis of caries may be aided by the detection of quantitative and/or qualitative changes in saliva. The awareness of the normal rate of salivary flow is essential to provide home-care instructions. The pH and salivary buffering capacity depend upon bicarbonate concentration in the saliva. sIgA in whole saliva has a specific role in the defense mechanisms of the oral cavity. The concentration of sIgA depends on hormones, physical activity, emotional state, and salivary flow rate.

The best method for assessing caries risk is to evaluate salivary flow rate, pH, buffering capacity, and sIgA levels. The purpose of this study is to estimate the sIgA levels, buffering capacity, pH, and salivary flow rate in children with ECC and caries-free children and to evaluate their role in caries risk assessment. The current study

assumes special relevance in the context of its sensitivity as well as the limitations associated with the conduct of such an investigation.

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