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Teeth Structure Analysis of Primary Teeth in Children with Congenital Heart Disease (Ventricular Septal Defect) in Erbil City.

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ABSTRACT

Objective: This study is performed to find out any differences in the chemical composition of primary teeth between children with ventricular septal defect (VSD), and those without (VSD) in Erbil city.

Methods: Children enrolled in this study were divided into two groups—group I (no VSD) and Group II (VSD). The collected teeth were (n=22) in each group. The structural and chemical composition of enamel and dentin were examined by scanning electron microscope/energy-dispersive x-ray (SEM/EDEX). An unpaired t-test was used in statistical analysis. P<0.0001 was considered as significant.

Results: EDEX analysis of the enamel layer in group I showed that calcium, phosphorus, silica, oxygen, fluorine, and sodium were significantly higher (P<0.0001) while carbon ions concentration was not. In the dentin layer, only calcium, phosphorus, fluorine, and sodium components were significantly higher in group I (P<0.0001). SEM analysis showed that disruption of the enamel layer was significantly higher in group II (7.60±15.41, and 14.21 ± 46.09) respectively for groups I and II (P<0.0001). Significant differences in dentin layer thickness were found (7.32 ± 33.28 and 3.807±11.94) respectively (P<0.0001). Dentin tubule occlusion was significantly higher in group I (7.59 ±74.18) than in group II (49.51± 45.27), P<0.0001. The number of odontoblast cell layers between groups was significantly higher in group I (7.32±33.28, and 3.807±11.94)respectively (P<0.0001).

Conclusion: VSD can result in significant structural differences in the enamel and dentin layers of primary teeth. It can also cause sub-optimal concentrations of certain minerals like Ca, P, O2, Na, and Silica in primary teeth.

Keywords: Ventricular Septal Defect, chemical structure, primary teeth.

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INTRODUCTION

Globally, eight out of every 1000 newborns are affected by congenital heart disease (CHD), one of the most prevalent heart conditions in the world.[1] Both the oral disease itself and its systemic effects are more prevalent in children with congenital heart disease (CHD).[2]

Ventricular septal defect (VSD), in particular, stands as one of the frequently encountered manifestations of CHD.[3] A significant correlation between oral health and CHD has been established, with the risk of infective endocarditis elevated due to bacterial colonization and oral infections. Consequently, the dental care of CHD patients warrants heightened attention. There is evidence suggesting a bidirectional relationship, where oral pathologies may exacerbate cardiac conditions, while CHD can negatively impact oral health.[4]

Various determinants contribute to the increased susceptibility to dental caries in pediatric CHD patients, such as the pharmacological treatments prescribed for their heart condition, socioeconomic influences, and dietary habits. Despite an absence of marked differences in caries rates or enamel abnormalities compared to their healthy counterparts, children with CHD frequently experience delayed interventions for dental decay, lower overall dental care standards, and a higher incidence of primary tooth extractions. It has been proposed that the dental structure in CHD children is compromised, leading to a greater likelihood of caries., despite no significant differences in dental caries or enamel defects.[5]

It has been proposed that the dental structure in CHD children is compromised, leading to a greater likelihood of caries. Alterations in the enamel's integrity, along with irregular orientation of dentin tubules, contribute to this vulnerability. In addition, diminished concentrations of calcium and phosphorus have been observed in the dental tissues of CHD children.[6]

Research consistently highlights a higher prevalence of dental issues in children with CHD compared to those without.[7] Notably, the occurrence of enamel opacities (8% vs. 2%) and hypocalcification (10.5% vs. 2%) is significantly more common in CHD patients.[8] Previous findings have also demonstrated reduced levels of calcium and phosphate in the dental structures of these children.[9]

Multiple factors influence dental development from infancy through adolescence, including genetic predispositions, systemic illnesses, the composition of saliva, environmental conditions, and behavioral traits. These elements collectively contribute to defects in both enamel and dentin formation.[10] This study is performed to find any differences in the chemical composition of primary teeth between children with ventricular septal defect and those without (VSD) in Erbil City.

PATIENTS AND METHOD

Study design: It is a case-control study. This is a comparative in-vitro study to find out the structural difference in the composition of primary teeth between both the control group and children with VSD.

Setting: The children who were diagnosed with VSD were gathered from the pediatric cardiac center, this center is specific for heart diseases, while the children who were enrolled in the control group were taken from Rapareen Pediatric Hospital, who attend there for reasons other than heart diseases. This study was performed from September 1 st 2021 to July 30 th 2022 in Erbil City, Kurdistan region- Iraq.

Inclusion criteria: The age of included children ranged between 4 to 10 years old. Children should have sound, pre-shedding mobile primary tooth that is bothering the child during eating, lingually erupted or retaining primary incisors (shark teeth) that are affecting the alignment of newly erupting permanent incisors.

Selection of cases: The parents of all children diagnosed with VSD who were between the ages of 4 and 10 were asked to permit their children to take part in the study; however, 147 of those parents declined, and only 22 agreed to give permission.

Selection of controls: The control group consisted of children of the same age as the patients, who did not have any history of heart disease, and who lived in the same geographic region. In addition, members of the control group went to a Rapareen pediatric hospital, dental department, who had dental problems. A structured questionnaire including the child's biological sex, age, medical history, including ultrasonography, medical reports, and types of medicine in use. The primary oral examination was performed in Rapareen Hospital for (Group I) and Erbil Cardiac Center for (Group II). The examination was performed while the child was sitting comfortably on an ordinary chair, un-

der good illumination obtained using a pen light and mouth mirrors to determine any mobile primary tooth or shark teeth needed for dental extraction.[11] prophylactic antibiotic for CHD patients was discussed with the cardiologist then a perinatal or oral antibiotic (in case it is needed) was given one to two hours earlier to the extraction procedure.[12] Extraction was done using topical and infiltration anesthesia.

Ethical consideration: The principles outlined in the Helsinki Declaration were adhered to throughout the research. On Sep. 28th, 2021, the College of Dentistry at Hawler Medical University issued its ethical approval with the reference number (HMU-D-30). Everyone who participated provided their informed oral consent, which was collected. The criteria established by STROCSS 2021 have been accounted for in this study.[13]

Sample preparation: Samples were fixed in 1% formaldehyde and 1% osmium tetroxide for 15 min each and treated with 40% phosphoric acid for 10 sec and sodium hypochlorite for 15 sec. Finally, samples were sputter-coated with osmium plasma[14] and images were obtained using a scanning electron microscope(TESCAN MIRA II). The composition of the tooth surface was analyzed using an energy-dispersive x-ray spectroscopy instrument (TESCAN MIRA II, SAMX Detector) attached to the SEM. The structure of the tooth tissue was analyzed for Ca, P, Si, O, Na, C, and F. Unpaired t-test statistical significance was set at ($P<0.05$).

Results of EDEX analysis:

In the Enamel layer, the concentrations of Ca, P, Si, O_2, F, and Na were significantly different between both groups (P value <0.0001). While carbon ion concentration was not significantly different between groups. (Table 1).

In the dentin layer, only calcium phosphorus, fluorine, and sodium components were significantly higher in group I than in group II($P<0.0001$), Table 2.

Table 1: chemical concentration of different components in the enamel of both groups(I and II).

	Ca	P	Si	O2	C	Na	F
Group 1	685.3 ± 275.8	396.1 ±147.5	38.21 ±15.85	210±66.33	224.2±111.1	23.25±11.23	9.515±3.955
Group II	80.45 ± 56.38	74.8-47.66	17.58±8.1	93.1±35.38	199.8±84.79	13.58±7.40	5.368±2.562
P value <	0.0001	0.0001	0.0001	0.0001	0.417	0.0001	0.002

Table 2: chemical concentration of different components in the dentin of both groups, I and II.

	Ca	P	Si	O2	C	Na	F
Group1	595.8 ± 299.9	193.8 ±135.5	16.71 ±14.84	147.2±95.85	276.5.2±157	8.808±6.17	7.375±6.23
Group2	116.2± 74.30	55.35±35.33	16.71±14.84	146.1±87.22	190.8±135.4	16.56±13.50	4.674±2.45
P value <	0.0001	0.0001	0.092	0.967	0.06	0.0007	0.0001

Results of scanning electron microscope (SEM) analysis :

Fingers (1 & 2) show that, in the control group(I), the cohesion of dental pulp tissue and connective tissue of dental pulp was higher than in group II. In addition, the pulp mesenchymal stem cell population and the fibroblast cells that make up the dental pulp micro-environment were significantly higher than in group I($P < 0.0001$). The blood vessels inside the dental pulp were visible in the form of capillaries, venules, and atriols as regular and distinct vessels, while in group (II) some of the arteries ruptured, and blood cells were seen in the interstitial space.

In the control group (I), the odontoblast cell layer along with the odontoblast cells were easily visible. Odontoblast cells were not well visible in group(II), however, a clear boundary between dentin and tooth pulp was visible.

The thickness of the dentin layer in group (I) was more than in group (II). In addition, dentin tubule occlusion was visible in group (I) compared to group (II). At the same time, the number of the orifice of dental tubules and the integrity of these tubules in group (II) has decreased.

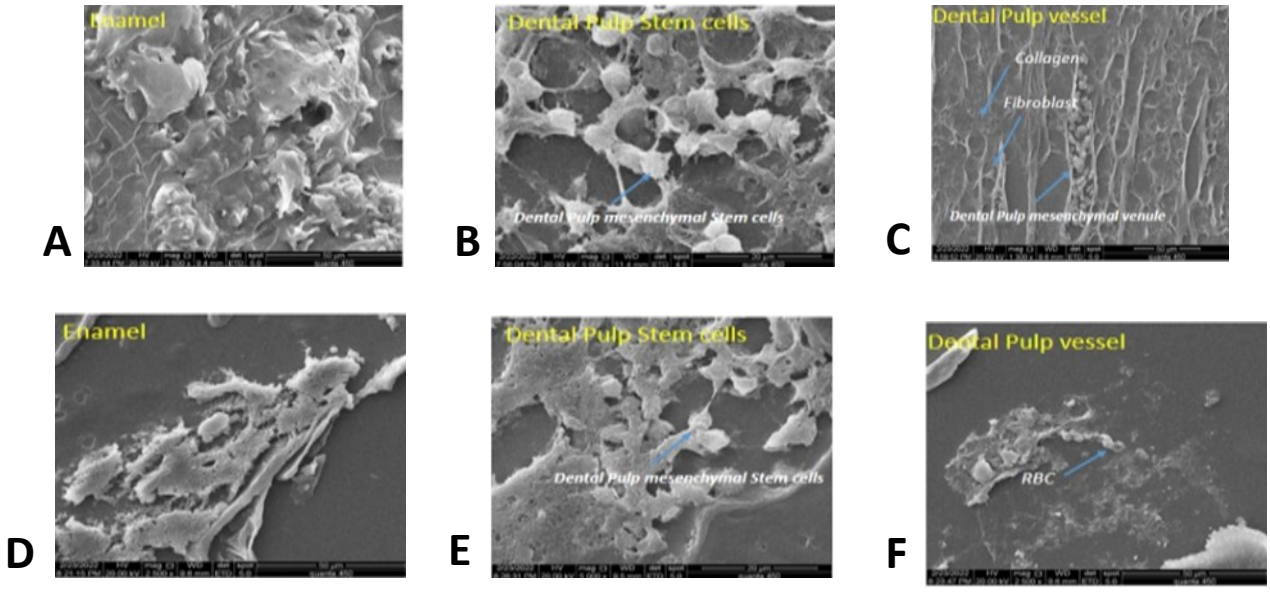

Figure 1: A: SEM image of the primary tooth enamel layer in group I. B: increased disruption of primary tooth enamel layer in group II. C: high cohesion of dental pulp and connective tissue of the pulp of primary teeth in group I.D: less cohesion and mesenchymal cells population in the pulp of the primary teeth of group II.E visible blood vessels in the pulp of group I.F: ruptured vessels and blood cells of the pulp tissue

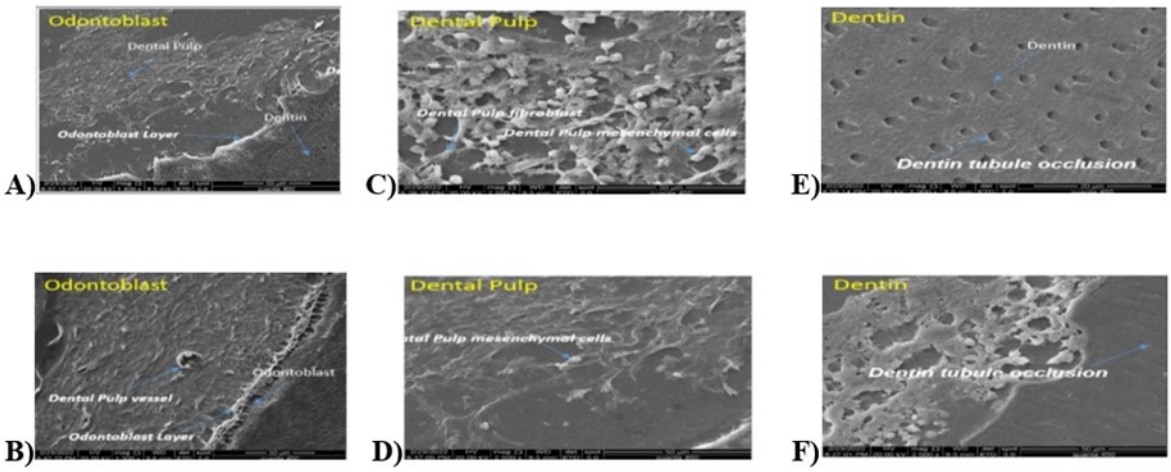

Figure 2: A: SEM image of group I primary tooth showing visible regular odontoblast layer. B: SEM image of group II showing not well visible irregular odontoblast layer.C SEM of dentin pulp stem cells in group I showing significantly higher number of stem cells/100 µm .D: SEM of group II dentin pulp stem cells showing significantly less number of cells/ 100 µm. E: Clear dentinal tubules occlusion in group I.F: Not clear orifices and integrity of dentinal tubules occlusion in group II.

In terms of the number of odontoblast cells/100 µm, the means±SD of both groups(I and II) were (33.28±7.32, and 11.94 ±3.807) respectively. Unpaired t-test analysis resulted in a significantly higher number of odontoblast cells found in group I than in group II (P< 0.001). Figure (3).
Occlusion of dentinal tubules in group I was significantly higher (74.18±7.59 and 45.27±9.51respectively. (P< 0.001). Figure (4).
In terms of dental pulp stem cells per 100 µm, the number of cells was noticeably higher in group I than in group II(P< 0.001).Means±SD in both groups were(89.55±9.96), and(39.36±10.23) respectively. Figure (5).

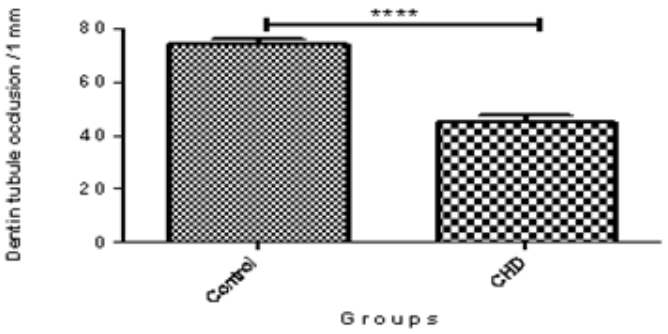

Figure 4: Graph showing the differences in dentinal tubules occlusion in both groups, I and II.

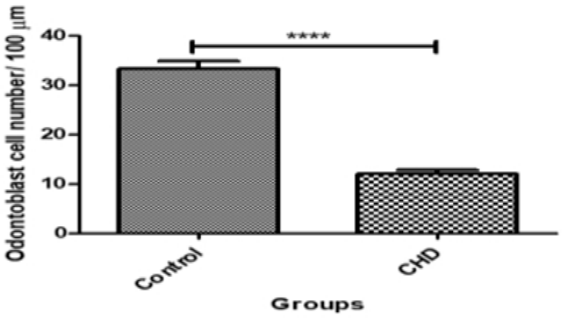

Figure 3: Graph showing the differences in the number of odontoblast cells in both groups, I and II.

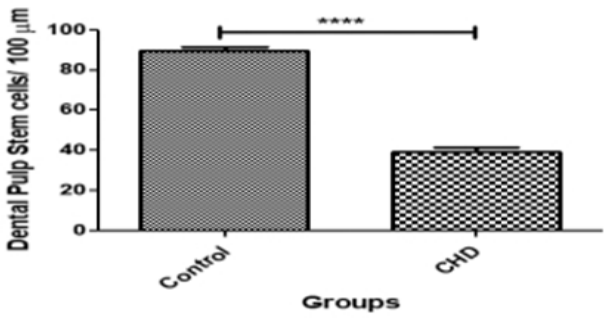

Figure 5: Graph showing the differences in numbers of dental pulp stem cells in both groups, I and II.

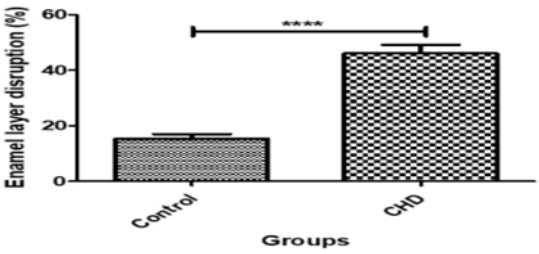

Figure 6: Graph showing the disruption in the enamel layer between both groups (I and II).

DISCUSSION

The current investigation utilized scanning electron microscopy (SEM) to elucidate the abundance of mesenchymal stem cells (MSCs), odontoblasts, fibroblasts, and dental pulp mesenchymal cells per 100 µm, which were significantly heightened in Group I. Fibroblasts, recognized as the predominant cellular elements within the pulp, play a pivotal role in the reparative mechanisms of pulp tissue by releasing crucial factors that stimulate stem cell activity.[15] This observation may shed light on the heightened incidence of dental caries in pediatric patients diagnosed with congenital heart disease (CHD)[16] Prior research has established that children with CHD frequently undergo endodontic procedures at a greater rate compared to their healthy peers. The preservation of the vitality and sensory function of dental pulp is dependent on an intricate network of blood vessels and nerve fibers that extend through the apical foramina. The regenerative capacity of pulp is fundamentally tied to the angiogenic and neurogenic properties of MSCs[18] This study identified diverse vascular structures, including capillaries, venules, and arterioles, which were prominently observed in Group I. Conversely, Group II exhibited signs of compromised vascular integrity, as evidenced by ruptured blood vessels and extravasated red blood cells within the surrounding tissues. Additionally, there was a statistically significant increase in dental pulp stem cell counts in Group I ($P < 0.001$). These findings could explain the increased occurrence of necrotic teeth and pulp de-vitalization among children suffering from congenital cardiac anomalies. The SEM assessments further illustrated significant occlusion of dentinal tubules in Group I compared to the diminished number and compromised structural integrity of tubules in Group II. These observations are consistent with previous findings in the literature.[19] In Group I, the odontoblast layer was well-defined, accompanied by a notable increase in the thickness of dentin. A reduced population of odontoblasts may adversely impact the production of reparative dentin when faced with external stimuli, potentially leading to widespread inflammation within the pulp and a subsequent loss of tooth vitality. Previous studies corroborate the assertion that children with congenital heart defects show a greater prevalence of carious teeth.[16] The conclusions drawn from this study suggest that the elevated risk of caries among children with congenital heart disease may arise from diminished regenerative capabilities in the dental pulp, characterized by lower counts of dental pulp stem cells, mesenchymal cells, and fibroblasts. Furthermore, the decrease in the number of odontoblasts in these children negatively affects secondary dentin formation and delays the restoration of dental structures. This is in agreement with earlier research indicating that dmft/DMFT indices were significantly lower in healthy children than in those with congenital heart conditions.[16] Moreover, earlier investigations have reported that enamel affected by acyanotic and cyanotic heart diseases shows increased dissolution and abnormal orientation of enamel prisms. [19] Another study indicated that 29% of primary teeth in children with CHD displayed enamel defects. [20] In this study, there was a greater degree of enamel disruption and discontinuity observed in Group II. Enamel hypoplasia is widely acknowledged as a significant risk factor for early caries.[21] Research conducted by AL-Etbi N and Al-Alousi identified a heightened incidence of enamel defects in children with ventricular septal defects.[22] The outcomes of this study align with previous literature, emphasizing the critical need for preventive dental care for children with congenital heart disease.

In contrast, some studies have reported no significant differences in the incidence of dental caries or enamel defects between healthy children and those diagnosed with congenital heart disease. [23]

Furthermore, SEM analysis demonstrated that the concentrations of calcium and phosphorus within both the enamel and dentin layers were significantly lowered in Group II ($P < 0.0001$). The observed reduction in mineral content among chil-

dren with congenital heart disease may be linked to underlying hemodynamic and metabolic alterations.[24] The results go in the same direction with another study.[19]

Additionally, the fluorine concentrations were significantly lowered in Group II ($P < 0.002$), supporting prior assertions that insufficient fluoride intake correlates with an increased risk of developing dental caries.[25]

The analysis also indicated that oxygen ion concentrations were significantly elevated in the enamel layer of Group I ($P < 0.0001$). A decrease in oxygen levels, leading to hypoxic conditions, can impair cellular proliferation, potentially resulting in cellular damage and apoptosis.[26]

Furthermore, silica levels were markedly elevated in the enamel layer of Group I ($P < 0.0001$). Silica is critical for bone regeneration, facilitating the proliferation and differentiation of various stem cell types, including those derived from bone marrow, dental pulp, periodontal ligaments, and adipose tissue.[27] Notably, sodium concentrations were also significantly higher in Group I ($P < 0.001$).

The observed decreases in silica and sodium levels among children with congenital heart disease may be attributed to the hemodynamic and metabolic shifts associated with their condition.[24] These findings may elucidate the increased susceptibility to dental caries within this population.

Additionally, SEM results indicated no statistically significant differences in carbon concentrations between the two groups ($P < 0.417$).

This study faced several limitations, notably the absence of a dental tissue repository in the research area, which severely restricted the availability of dental samples. Many caregivers prioritized cardiac health over dental issues, often lacking adequate understanding of the potential consequences of oral health on overall cardiac function, particularly the risk of infective endocarditis. Additionally, the negative effects of ventricular septal defects on the structural integrity of primary teeth were not fully acknowledged. Furthermore, the research was constrained by insufficient funding, which limited the sample size and overall scope of the study.

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