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Authors: Tamara Pawlaczyk-Kamieńska, Maria Borysewicz-Lewicka, Renata Śniatała, Halina Batura-Gabryel

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Clinical evaluation of the dental hard tissues in an adult population with cystic fibrosis

Tamara Pawlaczyk-Kamieńska¹, Maria Borysewicz-Lewicka¹, Renata Śniatała¹, Halina Batura-Gabryel²

1.Department of Pediatric Dentistry, Poznan University of Medical Sciences, Poznan, Poland
2.Department of Pulmonology, Allergology and Respiratory Oncology, Poznan University of Medical Sciences, Poznan, Poland

SHORT TITLE: Evaluation of dental tissues in CF patients

Corresponding author : Tamara Pawlaczyk-Kamieńska, DDS, Ph.D.
Department of Paediatric Dentistry, Poznan University of Medical Sciences
70 Bukowska Street, 60-812 Poznan Poland
e-mail: tamara_pawlaczyk@wp.pl

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Introduction

In recent decades, mainly due to multidisciplinary health care the life expectancy of patients with cystic fibrosis (CF) has increased [1] and nowadays new aspects of medical and dental care must be considered. Dental issues do not seem to be receiving sufficient attention from multidisciplinary CF teams, whereas research shows that CF patients, compared with healthy controls, show an equal or higher prevalence of (and more severe) developmental enamel defects in the permanent dentition [2]. This is not only an aesthetic problem, but it may also increase caries susceptibility.

On the one hand, dental enamel is a unique body tissue because it is the hardest and most mineralized tissue, and it is exposed to external influences such as food and air. On the other hand, molecular identification of enzyme proteins associated with transepithelial ion transport in enamel formation (amelogenesis) is similar to that described for other organs. The enamel biology is a part of an integrated systems biology and can be affected by the same anomalies [3-5]. Enamel-forming cells (ameloblasts) are highly sensitive, even to relatively minor changes in their microenvironment such as temperature increases, hypocalcemia, and acidic pH [4, 6]. Factors that may interfere can be either local (trauma or infection of a primary predecessor of the permanent) or systemic (systemic pathologies, toxic factors, nutritional disorders; that may directly affect the process or indirectly through the complications of the underlying disease or their pharmacotherapy) [3, 5]. The researchers suggested that in CF patients the disruption of cystic fibrosis transmembrane conductance regulator (CFTR) may also have a negative impact on enamel phenotype [3, 5, 7-10].

The aim of this study was to determine the dental status in adult CF patients: the prevalence and severity of enamel defects, as well as caries incidence.
Patients and methods

The ethical principle expressed in the world Medical Association Declaration of Helsinki were followed in this study. The study approval was requested and obtained by the Ethical Committee of the Poznan University of Medical Sciences, Poland (No 427/16).

The study involved 22 patients ≥18 years of age with confirmed diagnosis of cystic fibrosis, treated in the Department of Pulmonology, Allergology and Respiratory Oncology of the Poznan University of Medical Sciences, Poland. The control group consisted of 22 generally healthy people who were sex and age compatible with CF patients. The age of patients in each group ranged from 20 to 43. Both study groups were living in the same environment. All subjects participating in the study, after explaining to them the aim of the study and its procedure, gave informed consent to participate in this study.

Dental clinical examinations were carried out by two professionals, in the same room and the same lighting conditions, using a dental mirror and dental probe, according to the recommendation for oral epidemiological surveys by the WHO [11]. The examiners were calibrated (k=0.85, enamel defects; k=0.82, dental caries). To assess the presence of enamel defects teeth surfaces were examined and scored using modified Developmental Defects of Enamel index (DDE) [12]. When two different defects were found in the same tooth, the more severe was registered. To determine dental caries experience the decayed (D-T), missing (M-T) and filled (F-T) permanent teeth index (DMF-T) was used, were D-T means the number of teeth with carious lesions, M-T - the presence of dental fillings and M-T - missing teeth caused by complications of dental caries.

Statistical analysis was carried out using the Statistica v. 12 program (Program StatSoft, Inc. (2014). STATISTICA www.statsoft.com.). The difference between the test and control groups in relation to the parameters of the DMF-T index and its components (D-T,
MT, F-T) was tested using Mann-Whitney tests and the prevalence of enamel defects was by
the difference test between the two structure indices. Spearman’s Correlation Coefficient was
used to evaluate the relationship between dental status and FEV1% predicted and between
dental status and CFTR mutation class. P-value <0.05 indicated statistically significant for all
analyses.

Results

In CF patients compared to control group the prevalence of enamel defects was a
significantly higher (p=0.03) (tab.), the defects were more severe, and more teeth were
affected (respectively in CF patients 7.41%-100%, and healthy individuals 7.14%-36%). In
CF patients defects were noted in all groups of teeth, and in the control group only on the
incisors and the first molars.

Caries experience (DMF-T) in CF patients was significantly higher comparing to the
control subjects. Moreover, in CF patients significantly higher decayed (D-T) and missing
(M-T) indices were reported. In the control group, 66.7% of all teeth extracted were the first
molars, while in CF patients none of the tooth groups dominated in this index.

In CF adults there was not statistically significant correlation between dental status
and FEV1% predicted neither between dental status and CFTR mutation class (p≥0.05).

Discussion

Available publications on the dental status of CF patients have been concerned mainly
with children and adolescents [2]. To the best of our knowledge, this paper is the first report
on the dental condition of CF adults. It was difficult for us to compare our results with other
authors’ reports because the subjects were fundamentally different in many respects, such as
age, number of erupted permanent teeth, disease duration, and pharmacotherapy. The above-
mentioned publications showed equal or higher prevalence of enamel defects of the
permanent dentition in CF patients compared with that of healthy individuals [2]. Although
our patients’ ages differed from those in the cited publications, our results confirmed the
significantly higher prevalence of and more severe enamel defects in CF patients compared
with those in a healthy population.

The published reports are inconsistent regarding dental caries in CF adults. They show
an equal or lower caries risk in CF patients compared with that of the control group [2].
Results of the present study indicate not only poor dental status (DMF-T) in CF adults but,
most of all, medical negligence. With definitely higher caries experience (D-T, M-T), there
was no difference in the treatment levels (F-T) between the CF patients and healthy controls.

The presence of enamel defects only on the incisors and first molars of the control
group suggests that these anomalies meet the Molar Incisor Hypomineralization (MIH)
criteria, in which the etiology is multifactorial and related to general factors, such as
premature birth, general health problems, and systemic conditions in the first 3 years of life.
In CF patients, theses anomalies were not related to any specific dental group and were
equally distributed in all of them, suggesting a continuing insult of the causative factor.

For a long time, the enamel defects etiology in CF patients was attributed mainly to
the side effects of antibiotic therapy and complications of the underlying disease [2]. But
laboratory studies carried out on CF animals [4, 7, 8, 10] suggest CFTR disruption of
amelogenesis, during which a large number of H⁺ protons is formed. Buffering these protons
to neutrality is essential for mineral deposition and further enamel formation [4, 5, 7–9].
CFTR function in amelogenesis is poorly understood, but at the present state of knowledge, it
is likely to be for controlling the activity of the bicarbonate-chlorine (Cl⁻/HCO₃⁻) exchanger
channel in the cell membranes; this regulator thus neutralizes the H+ protons and provides the
optimal pH necessary for the transport of Ca²⁺ ions [3, 4, 7–10]. The CFTR protein is
localized in the apical plasma membrane of epithelial cells, also in the ameloblasts, where it is
located on the apical side in proximity to the growing enamel crystals [3, 8]. Elemental analysis and investigation of enamel pH carried out by Sui et al. (2003) [4] and Bronckers et al. (2015) [8] showed significantly lower pH, fewer Cl\(^-\) ions, and diminished HCO\(_3^-\) concentration in the enamel of CF mice than found in healthy controls. Moreover, Arquitt et al. (2002) [7] and Bronckers et al. (2015) [8] confirmed the thesis of the unfavorable effect of low pH on Ca\(^{2+}\) transport through the cell membrane. The authors showed a lower calcium level, similar phosphate level, and lower calcium-to-phosphate ratio in the mature enamel of unerupted teeth in CF mice than present in healthy individuals.

Tooth enamel is a protective layer for other tooth tissues, and abnormalities of its structure and chemical composition arising during development are irreversible. These irregularities significantly affect the physical and chemical properties of the tissue, making it less resistant to damaging factors. Reduced enamel hardness and increased porosity promote penetration of cariogenic bacteria into the tissues and the development of carious lesions. Therefore, it is necessary to implement early dental care and to develop an individual long-term prophylaxis and treatment program to prevent complications such as pulpitis and periapical inflammation, conditions that could be a source of autoinfection for CF patients.

Conclusions

1. Due to the high prevalence of enamel defects in CF patients, it is necessary to implement early systemic dental prevention in children. The pediatric dentist should be a member of the CF multispecialist team.

2. Poor dental status in CF adults indicates a lack of proper dental care as well as not meeting patients’ oral preventive needs.

3. It is necessary to develop and implement targeted caries prophylaxis and treatment programs for CF patients.
Acknowledgments

We are grateful to the individuals with cystic fibrosis and from control group for contributing to this study.

References


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Table 1 Prevalence of enamel defects and dental caries among cystic fibrosis adults and control group patients

<table>
<thead>
<tr>
<th>Enamel defects</th>
<th>CF patients</th>
<th>Healthy group</th>
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<td>White/cream coloured opacity</td>
<td>13.64%</td>
<td>13.64%</td>
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<tr>
<td>Yellow/brown coloured opacity</td>
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<td>9.09%</td>
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<tr>
<td>Diffused opacities</td>
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<tr>
<td>Hypoplasia</td>
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<table>
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<tr>
<th>Dental caries</th>
<th>Me</th>
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<th>Q3</th>
<th>Me</th>
<th>Q1</th>
<th>Q3</th>
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<td>D-T</td>
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<tr>
<td>M-T</td>
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<td>3</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>F-T</td>
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<td>2</td>
<td>11</td>
<td>4</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>DMF-T</td>
<td>14</td>
<td>9</td>
<td>18</td>
<td>5</td>
<td>4</td>
<td>10</td>
</tr>
</tbody>
</table>

Abbreviations: CF, cystic fibrosis; D-T, decayed teeth; DMF-T, decayed missing filled teeth (index for dental caries); F-T, filled teeth due to caries; M-T, missing teeth due to caries; ME, median; PT, number of present teeth; Q1, first quartile; Q3, third quartile

Caries with: 100% 86.36%
Caries free: 0% 13.64%