

Evaluation of the relationship between dental caries and urinary tract infections

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ABSTRACT

Objective: The most common infections in childhood and adolescence are the upper respiratory tract and urinary tract infections (UTI). Tooth decay is a multifactorial and infectious disease resulting from bacteria multiplying within the mouth and settling on the teeth and gums. The purpose of this study was to investigate whether any relationship exists between tooth decay and UTIs.

Material and Methods: This study was performed between January and June 2017 at the Adıyaman University Faculty of Medicine Department of Pediatrics. One hundred and forty-one cases were included. Patients' age, sex, tooth brushing habits, hematological, biochemical and serological tests, complete urine examination, urine culture, blood culture, urinary system ultrasonography parameters, and numbers of decayed teeth were separately recorded.

Results: Comparison of the cystitis and pyelonephritis groups revealed significantly greater tooth decay in the pyelonephritis group. *Escherichia coli* was the most common agent in all groups. Positive acute phase reactants such as white blood cell (WBC), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESH) were high in the group with tooth decay and pyelonephritis, while red blood cells (RBC), Hct, albumin, amylase, calcium, chloride, iron, and phosphorus levels were low.

Conclusion: Our study shows a positive association between pyelonephritis and tooth decay. We think that examination in terms of tooth decay among children under going pyelonephritis attacks will be beneficial to diagnosis and treatment. Further studies are now needed on this subject.

Keywords: Child, cystitis, pyelonephritis, tooth decay, urinary tract infection.

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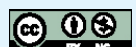
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INTRODUCTION

The most common infections in childhood and adolescence are the upper respiratory tract and genitourinary system infections. The most common genitourinary system infections are in the urinary tract.^[1,2]

The most frequent mode of urinary tract infection (UTI) is through the ascending pathway.^[3] Hematogenous spread is a less common cause of UTI that generally occurs through bacteria in another system entering the urinary system through the blood. Agents giving rise to UTIs through the hematogenous route include *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Candida* spp.^[4]

Tooth decay is a multifactorial and infectious disease that emerges due to bacteria multiplying inside the mouth and settling in the teeth and gums, and to the gradual effect of host factors. Infections of the teeth and gums are frequently caused by bacteria, particularly anaerobic bacteria.^[5–8]

Tooth decay, a source of bacteremia through hematogenous spread, can play a role in the development of several infections in the body. Studies have reported that bacteria originating from tooth decay produce infection in different tissues through the hematogenous route.^[9,10] The kidneys receive 20–25% of cardiac outflow, and any micro-organism entering the circulation can easily cause infection by reaching the urinary system.^[4]

The subject of examining the relationship between dental caries and UTI is the first research known in the literature. In our clinic, we observed that UTI is more frequent in dental decay. We decided to do this work to see if our observation is correct. The purpose of this study is to examine whether any association exists between tooth decay and UTI.

MATERIAL AND METHODS

Patient Selection and Data Collection

One hundred and forty-one children aged 5–17 years undergoing tests and treatment due to UTI at the Adıyaman University Teaching and Research Hospital Pediatrics Clinics, Türkiye, between January and June 2017 were included in the prospective study.

Exclusion Criteria

Patients with UTI but also with chronic disease (diabetes, hypertension, etc.), with urogenital system anomaly (horseshoe kidney, VUR, hypospadias, etc.), with UTI detected under the age of 5 years, diagnosed with monosymptomatic primary enuresis, with congenital or acquired neurogenic bladder, with neutropenia, with diagnoses of primary or secondary immune failure, receiving immunosuppressive therapy, or with other system infections accompanying UTI were excluded from the study. Cases of acute UTI recruited into the study were assigned into four groups: Group I, tooth decay+cystitis; Group II, tooth decay+pyelonephritis; Group III, cystitis only, and Group IV, pyelonephritis only.

Cases' age, sex, and tooth brushing habits were recorded. In addition, cases' hemostatic parameters (leukocyte count WBC, platelet, hemoglobin, hematocrit, men cell volume, mean platelet volume, RBC, neutrophil count, lymphocyte count, eosinophil count, and basophil count), biochemical parameters (glucose, urea, creatinine, uric

acid, albumin, total bilirubin, direct bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma glutamyl transferase (GGT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), amylase, sodium, potassium, chloride, calcium, and iron), CRP, ESH, and complete urine test parameters (density, nitrite, pH, leukocyte esterase, leukocyte count, and urine culture), blood culture, and imaging results were recorded.

Blood Samples

Complete blood count, biochemistry tests, and CRP were studied with machines in the hospital.

For blood culture, 1–2 ml venous blood collected under sterile conditions was added to BACTEC cultures and incubated for 7 days in the microbiology laboratory on an automated BACTEC FX device (BD, USA). Blood specimens giving positive results were added to 5% sheep's blood agar and eosin-methylene blue (EMB) agar. Following incubation for 24 or 48 h, Gram staining was performed on colonies exhibiting growth. Identification of growing bacterial agents at the subspecies level and antibiogram procedures were performed using catalase, oxidase, and coagulase experiments and the BD Phoenix 100 fully automated culture-antibiogram sensitivity test (BDA, USA).

Urine Samples

For complete urine tests and microscopy, urine specimens collected using the midstream method in children capable of bladder control were studied using the flow cell technology (flow digital imaging technology) on a fully automatic urine analysis device (FUS 100/H800, DIRUI Industrial Co. Ltd. China). For urine culture, 5% sheep's blood agar and EMB were added to midstream urine specimens collected after cleaning in line with general hygiene rules, and evaluation was performed after incubation for 18–24 h at 37°C.

Definitions

Diagnosis of UTI relies on a well taken history, complete physical examination, and laboratory tests (complete urine test, microscopic urine examination, enzymatic tests, urine culture, and imaging techniques).^[5,6] Definite diagnosis is made with urine culture. Significant bacteriuria may vary depending on the manner of collection of the urine specimen and the patient's clinical condition. If the number of micro-organisms growing in a specimen taken from midstream urine exceeds 100,000 this is regarded as UTI.^[1–3] Positive urine analysis, the patient being symptomatic and a single organism growing in culture with a colony number >100,000 shows the presence of UTI.^[5,6]

Statistical Analysis

Statistical analysis was performed on licensed SPSS (Statistical Package for the Social Sciences) 22.0 software (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean±standard deviation. The Chi-square (χ^2) test was used to compare categorical variables. The independent Student t-test was applied to normally distributed continuous data from two groups, while non-normally distributed data were analyzed using the Mann–Whitney U test. ANOVA, one-way analysis of variance and *post hoc* tests were used to

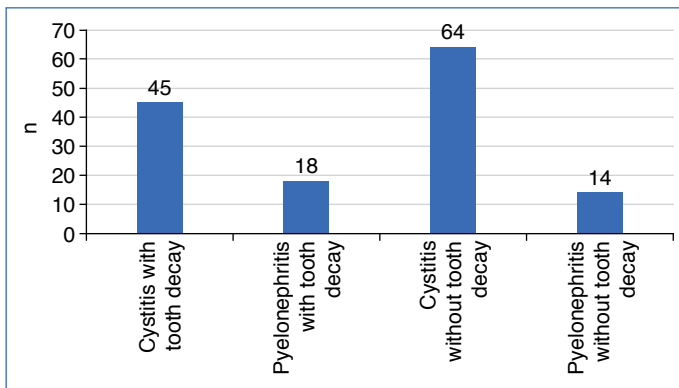


Figure 1: Cases of urinary tract infections with and without dental caries.

assess parametric data between more than two groups. If the difference between groups was found to be significant, Türkiye's test was applied for normally distributed groups and the Tamhane test for non-normally distributed tests. $P < 0.05$ was regarded as statistically significant. This prospectively designed study was conducted in accordance with the principles of the Declaration of Helsinki and with the approval of the Adiyaman University Faculty of Medicine Clinical Research Ethical Committee (no. 2016/8-11 dated 20.12.2016).

RESULTS

Our patients were divided into four groups. Group 1 consisted of 38 girls (84.5%) and seven boys (15.5%), Group 2 of 14 girls (77%) and four boys (23%), Group 3 of 59 (92%) girls and five boys (8%), and Group 4 of 12 girls (85%) and two (15%) boys. Cystitis was present in 109 (77%) cases and pyelonephritis in 32 (23%). 97 (89%) of the cystitis and 26 (81%) of the pyelonephritis cases were girls.

While the number of cases with UTI and dental caries was 63 (52 of them girls), the number of cases with UTI without dental caries was 78 (71 of them were girls) (Fig. 1).

Mean ages were 8.78 ± 3.6 years in Group 1, 7.61 ± 2.99 years in Group 2, 8.83 ± 3.71 years in Group 3, and 9.07 ± 3.77 years in Group 4. There was no statistically significant difference among the groups in terms of age ($p = 0.596$). In terms of bacteria growing in urine cultures, the most commonly identified bacteria in both the tooth decay and non-tooth decay groups were, in descending order, *Escherichia coli* > *Proteus mirabilis* > *Klebsiella* spp.

When all four groups were compared, the highest mean CRP value, the highest mean WBC count, the lowest mean RBC count, and the lowest mean Hct values were all determined in the group with tooth decay and pyelonephritis ($p = 0.001$ for all).

The lowest mean albumin level among all the groups was determined in the tooth decay plus pyelonephritis group ($p = 0.005$). No statistically significant differences were determined among the groups in terms of AST, ALT, ALP, LDH, and GGT levels ($p = 0.128$, $p = 0.059$, $p = 0.278$, $p = 0.741$, and $p = 0.281$, respectively). The lowest mean amylase, chloride, calcium, and phosphorus levels were observed in the tooth decay plus pyelonephritis group ($p = 0.037$, $p = 0.005$, $p = 0.002$, and $p < 0.001$, respectively). A comparison of the cases with pyelonephritis with those with cystitis revealed greater mean tooth decay in the cases with pyelonephritis ($p = 0.032$) (Table 1).

Table 1: A comparison of the cases with pyelonephritis and cystitis in terms of age, sex, and number of decayed teeth

Groups parameters	Cystitis (n=109) Mean±SD (Min–Max)	Pyelonephritis (n=32) Mean±SD (Min–Max)	p
Age (years)	8.81 ± 3.65 (5–16)	8.25 ± 3.65 (5–16)	0.442 ⁺
Sex (F/M)	(89%)/(11%)	(81%)/(19%)	0.194 [#]
Number of decayed teeth (range)	0.89 ± 1.22 (0–4)	1.47 ± 1.62 (0–6)	0.032 [*]

SD: Standard deviation; Min: Minimum; Max: Maximum; +: Student's t test; #: Chi-square (χ^2) test; *: Mann-Whitney U test.

DISCUSSION

We noticed that the patients presenting with UTI had dental caries. It is known that dental caries is a risk factor for infective endocarditis. We thought that dental caries may also be a risk factor for UTI, and if tooth decay is treated, there will be less UTIs.

UTI is defined as infection of any region of the urinary system by organisms such as bacteria, viruses, fungi, or protozoa.^[11,12] Although the pathogens that cause UTIs are often Gram-negative bacteria, sometimes Gram-positive bacteria (*Staphylococcus aureus*, *Staphylococcus epidermidis*, *Enterococcus* spp., and Group B Streptococci) can cause it.^[13]

Tooth decay, the principal source of bacteremia associated with hematogenous spread, can be involved in the development of numerous infections in the body. Studies have shown that bacteria deriving from tooth decay cause infection in various tissues through the hematogenous pathway.^[9,10]

In a research *Streptococcus mutans*, a major pathogen of dental caries, is considered to be one of the causative agents of infective endocarditis.^[14] Chronic oral infections and the manipulation of teeth and supporting structures can lead to the hematogenous spread of infection including the infection of artificial joints and endocardial implants.^[15] Data suggests that methods used to prevent cases of Infective endocarditis that originate from oral bacteria should focus on improving oral hygiene and reducing or eliminating gingivitis, which should reduce the incidence of bacteremia after tooth-brushing and the need to extract teeth owing to periodontal disease and caries.^[16]

Infectious dental foci and oral dental care constitute one of the leading causes of arthroplasty infection after infections involving the skin and the urinary tract. In one of the articles, the relationship between dental infections and arthroplasty infection was discussed. Successive episodes of spontaneous bacteremia arising from an oral-dental foci are probably the main cause of arthroplasty infections, more so than bacteremia triggered by dental care. Antibiotic therapy is not indicated for routine dental care in the majority of patients but is recommended whenever there is a high risk of arthroplasty contamination.^[17]

The mean reported the prevalence of UTI in children is approximately 1% in boys and 1–3% in girls.^[18] Çoban et al.,^[19] İpek et al.,^[20] and Senel et al.^[21] all reported that infection in children with UTI is more common in girls. Consistent with the literature, the proportion of girls was higher in all our study groups. Çoban et al.,^[19] İpek et al.,^[20] Senel et al.,^[21] Konca et al.,^[22] Kozlova et al.,^[23] and Tekin et al.^[24] reported that UTI is more common between 1 and 8.6 years of age. The mean age in all our study groups ranged between 4.6 and 12.8 years. Our mean ages were higher because our study was not concerned with the prevalence of UTI and since patients aged 5 years and over were included.

Studies from Türkiye and elsewhere in the world have identified *E. coli* as the most frequent agent in UTIs. *E. coli* is seen in 75–90% of girls with UTIs and in the majority of boys, followed by *Klebsiella* spp. and *P. mirabilis*.^[19] Kozlova et al.^[23] described *E. coli* as the most common agent in UTIs, followed by *P. mirabilis* and by *Klebsiella* spp. in third place. In our study from our own region, Konca et al.^[22] reported *E. coli* (60%) in first place, followed by *Klebsiella* spp. (16.5%). Tekin et al.^[24] also identified *E. coli* as the most frequent agent (64.9%), followed by *Klebsiella* spp. in second place (14.4%), and *P. mirabilis* in third (10.8%). In second place (11.3%) and *Enterobacter* spp. In third (2.4%) *E. coli* has always emerged as the most common agent in different studies, while the agents in second and third place vary. *E. coli* was against most frequently identified among the cases without tooth decay in the present study (71%), followed by *P. mirabilis* (14%), and by *Klebsiella* spp. in third place (5%). Similarly in cases with tooth decay, *E. coli* was the most common (81%), followed by *P. mirabilis* (6%) and *Klebsiella* spp. (5%). Analysis of all the groups identified *E. coli* as the most frequently growing bacterium. This is consistent with the available information in the literature.

CRP, ESH levels and WBC count are simple, non-invasive tests used in the diagnosis of invasive bacterial infections and in determining the severity of UTIs. From those previous studies we were able to access, Jaksic et al.,^[25] Tekin et al.,^[26] Konca,^[22] and Ayazi et al.^[27] all reported higher mean CRP, ESH levels and WBC counts in cases with pyelonephritis than in cases of cystitis. Similarly, in the present study, CRP, ESH levels, and WBC counts were higher in cases of tooth decay and accompanying pyelonephritis. We think that this is an expected finding related to pyelonephritis being a systemic infectious disease.

One of the findings associated with pyelonephritis is vomiting.^[7] In a previous study reported hypochloremia and metabolic alkalosis developing as a result of vomiting.^[28] Chloride levels were low among cases of tooth decay plus pyelonephritis in the present study, and we attributed this to vomiting being more common in cases with pyelonephritis. Low mean calcium values have been reported in cases of febrile convulsion with UTI.^[29] Calcium levels were also lower in cases of tooth decay plus pyelonephritis in the present study. This may be due to kidney functions being affected in cases of pyelonephritis and may also derive from impairment of the balance between mineralization and demineralization in decayed teeth.

To the best of our knowledge, no previous studies have investigated the relationship between UTI and tooth decay. The mean number of decayed teeth was higher in cases with pyelonephritis than in those with cystitis. This suggests that an increase number of decayed teeth may be a risk factor for pyelonephritis. Our study is also important in terms of being the first to reveal this relationship.

CONCLUSIONS

The prevention of tooth decay and early treatment can also prevent the development of several systemic infections, including UTI. Further studies are now required on this subject.

Statement

Ethics Committee Approval: The Adıyaman University Faculty of Medicine Biomedical Research Ethics Committee granted approval for this study (date: 20.12.2016, number: 2016/ 8-11).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – MT, HA; Design – FEK, HA; Supervision – İHB, FEK; Resource – MT, FEK; Materials – FEK; Data Collection and/or Processing – FEK; Analysis and/or Interpretation – İHB, HA; Literature Search – FEK; Writing – FEK; Critical Reviews – MT, İHB.

Conflict of Interest: The authors have no conflict of interest to declare.

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REFERENCES

- Balighian E, Burke M. Urinary tract infections in children. *Pediatr Rev* 2018;39:3–12.
- Moreno MA. Urinary tract infections in children and adolescents. *JAMA Pediatr* 2016;170:916.
- Morello W, La Scola C, Alberici I, Montini G. Acute pyelonephritis in children. *Pediatr Nephrol* 2016;31:1253–65.
- Bakkaloğlu SA, Schaefer F. Diseases of the kidney and urinary tract in children. In: Brenner BM, editor. *The Kidney*. 9th ed. Philadelphia: Saunders Elsevier; 2012. p.2622–80.
- Tinanoff N. Development and developmental anomalies of the teeth. In: Behrman RE, Kliegman RM, Jenson HB, editors. *Nelson textbook of pediatrics*. 20th ed. Philadelphia: Elsevier; 2015. p.1774–5.
- Tullus K. Defining urinary tract infection by bacterial colony counts: A case for less than 100,000 colonies/mL as the threshold. *Pediatr Nephrol* 2019;34:1651–3.
- Çakır FY, Gürkan S, Attar N. Microbiology of dental caries. *Clin Dent Res* 2010;34:78–91.
- Koçanalı B, Ak AT, Çoğulu D. Evaluation of the dental caries risk factors in children. *J Pediatr Res* 2014;1:76–9.
- Stang F, Stollwerck P, von Wild T, Mailänder P, Siemers F. Severe infantile wrist empyema due to dental bacteremia. *Ger Med Sci* 2012;10:Doc09.
- Holmberg P, Hellmich T, Homme J. Pediatric sepsis secondary to an occult dental abscess: A case report. *J Emerg Med* 2017;52:744–8.
- Hansson S, Jodal U. Urinary tract infection. In: Avner ED, Harmon WE, Niaudet P, editors. *Pediatric Nephrology*. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2004. p.1007–25.
- Hacımustafaoğlu M. Urinary tract infections in children. *Türkiye Klinikleri J Pediatr Sci* 2011;7:68–75.
- Vachvanichsanong P. Urinary tract infection: One lingering effect of childhood kidney diseases--review of the literature. *J Nephrol* 2007;20:21–8.
- Nomura R, Naka S, Nemoto H, Inagaki S, Taniguchi K, Ooshima T, et al. Potential involvement of collagen-binding proteins of *Streptococcus mutans* in infective endocarditis. *Oral Dis* 2013;19:387–93.

15. Coll PP, Lindsay A, Meng J, Gopalakrishna A, Raghavendra S, Bysani P, et al. The prevention of infections in older adults: Oral health. *J Am Geriatr Soc* 2020;68:411–6.
16. Carinci F, Martinelli M, Contaldo M, Santoro R, Pezzetti F, Lauritano D, et al. Focus on periodontal disease and development of endocarditis. *J Biol Regul Homeost Agents* 2018;32(Suppl 1):143–7.
17. Bauer T, Maman L, Matha C, Mamoudy P. Dental care and joint prostheses. *Rev Chir Orthop Reparatrice Appar Mot [Article in French]* 2007;93:607–18.
18. Elder JS. Urinary Tract Infections. In: Behrman RE, Kliegman RM, Jenson HB, editors. *Nelson Textbook of Pediatrics*. 20th ed. Philadelphia, Saunders Elsevier; 2015. p.2556–62.
19. Çoban B, Ülkü N, Kaplan H, Topal B, Erdoğan H, Baskın E. Five-year assessment of causative agents and antibiotic resistances in urinary tract infections. *Turk Pediatri Ars* 2014;49:124–9.
20. İpek IO, Bozaykut A, Arman DC, Sezer RG. Antimicrobial resistance patterns of uropathogens among children in Istanbul, Türkiye. *Southeast Asian J Trop Med Public Health* 2011;42:355–62.
21. Senel S, Karacan C, Erkek N, Gol N. A single-center experience of antimicrobial resistance patterns in pediatric urinary tract infection. *Med Princ Pract* 2010;19:359–63.
22. Konca C, Tekin M, Uckardes F, Akgun S, Almis H, Bucak IH, et al. Antibacterial resistance patterns of pediatric community-acquired urinary infection: Overview. *Pediatr Int* 2017;59:309–15.
23. Kozlova EA, Kholodok GN, Alekseeva IN, Kozlov VK. Etiology of acute and chronic pyelonephritis in children in Khabarovsk region. *Zh Mikrobiol Epidemiol Immunobiol [Article in Russian]* 2008;87–9.
24. Tekin M, Konca C, Almis H, Bucak İH, Genc Y, Gunduz A, et al. To evaluate the diagnostic efficacy of urinalysis for the diagnosis of childhood urinary tract infection. *J Dr Behçet Uz Child Hosp* 2015;5: 88–94.
25. Jaksic E, Bogdanovic R, Artiko V, Saranovic DS, Petrasinovic Z, Petrovic M, et al. Diagnostic role of initial renal cortical scintigraphy in children with the first episode of acute pyelonephritis. *Ann Nucl Med* 2011;25:37–43.
26. Tekin M, Konca C, Gulyuz A, Uckardes F, Turgut M. Is the mean platelet volume a predictive marker for the diagnosis of acute pyelonephritis in children? *Clin Exp Nephrol* 2015;19:688–93.
27. Ayazi P, Mahyar A, Daneshi MM, Jahani Hashemi H, Pirouzi M, Esmailzadehha N. Diagnostic accuracy of the quantitative c-reactive protein, erythrocyte sedimentation rate and white blood cell count in urinary tract infections among infants and children. *Malays J Med Sci* 2013;20:40–6.
28. Khanna A, Kurtzman NA. Metabolic alkalosis. *J Nephrol* 2006;19(Suppl 9):86–96.
29. Afsharkhas L, Tavasoli A. Renal function in children with febrile convulsions. *Iran J Child Neurol* 2014;8:57–61.