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Original Article

Environmental tobacco exposure linked to lower oxygen saturation in infants with community-acquired lower respiratory tract infections

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ABSTRACT

Objective: Tobacco smoke is a pervasive environmental hazard, particularly detrimental to the developing respiratory systems of infants. Exposure to environmental tobacco smoke (ETS) has been consistently implicated in the etiology of a spectrum of lower respiratory tract infections (LRTIs), which are a leading cause of morbidity in children under two years of age. The pathophysiological impact of ETS extends beyond exposure; it actively exacerbates the severity of respiratory conditions, often resulting in increased hospital admissions and prolonged medical care for the youngest and most vulnerable.

This study aimed to explore the relationship between exposure to ETS and the severity of clinical manifestations, laboratory findings, and hospitalization duration in infants with community-acquired LRTIs.

Material and Methods: A cohort of 115 infants aged 1–24 months, hospitalized due to community-acquired LRTIs and without prematurity or chronic diseases, was evaluated. Data on household tobacco use were collected, and infant cotinine levels were measured to assess the impact of ETS on the severity of LRTIs.

Results: Findings revealed that the frequency of urinary cotinine positivity is significantly higher in infants from households with smokers (p=0.001). Among patients with household smoking, the proximity of tobacco consumption to the child did not affect the frequency of cotinine positivity (p=0.501). Notably, the cotinine-positive group had significantly lower oxygen saturation at admission (p=0.038). In the RSV-positive subgroup, this association remained significant (p=0.015), providing stronger evidence that ETS independently exacerbates respiratory distress.

Conclusion: This study demonstrated that tobacco exposure is associated with increased respiratory distress in infants with lower respiratory tract infections. Emphasizing the importance of smoke-free environments during infancy, it also proved the negative effects of not only secondhand smoke but also thirdhand "surface" smoke exposure on infants' respiratory health.

Keywords: Environmental tobacco smoke pollution, Infant health, Oxygen saturation, Respiratory tract infections, Secondhand smoke

INTRODUCTION

Childhood pneumonia remains a critical public health issue, particularly in developing nations, where it accounts for 14% of deaths among children under five years of age (1). Exposure to environmental tobacco smoke (ETS) is a recognized risk factor exacerbating respiratory conditions, particularly in infants under two years (2, 3).

ETS comprises two key components: secondhand smoke (SHS) and thirdhand smoke (THS). SHS refers to the inhalation of airborne tobacco pollutants. Due to their smaller lung capacity and higher respiratory rates, children are more rapidly and significantly affected by these airborne pollutants. In contrast, THS represents residual pollutants left on surfaces and fabrics, such as clothing, furniture, and toys, after tobacco consumption. Children are particularly vulnerable to THS because of behaviors like playing on surfaces, touching objects, and exploring their environment by putting their hands and objects into their mouths (4, 5).

ETS exposure, encompassing both SHS and THS, has been associated with increased hospitalizations for respiratory illnesses, recurrent wheezing, and chronic cough (6-8). The global burden of SHS remains significant, with nearly one-third of the world's population exposed and approximately

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600.000 premature deaths annually attributable to SHS (3, 9). The inclusion of THS in the concept of ETS underscores the pervasive nature of exposure, revealing that the risk extends far beyond direct inhalation and includes contact with contaminated environments (10, 11).

The mechanisms underlying these adverse effects involve complex interactions. SHS exposure in children has been linked to oxidative stress, increased airway inflammation, and disruption of pulmonary defense mechanisms, compounding their susceptibility to respiratory infections (12). Cotinine, a stable nicotine metabolite, serves as a reliable biomarker to quantify ETS exposure. Elevated cotinine levels correlate strongly with respiratory morbidities, including reduced oxygen saturation, heightened airway reactivity, and increased susceptibility to infections (13).

While numerous studies have examined the relationship between ETS exposure and the incidence of acute lower respiratory tract infections (LRTIs), there is a notable gap in understanding how such exposure influences disease severity. This study aimed to address this gap by investigating the impact of ETS exposure on clinical outcomes, including oxygen saturation, hospitalization duration, and laboratory findings, in infants with community-acquired LRTIs.

MATERIALS and METHODS

Study population and design

This prospective observational study included patients aged 1–24 months admitted with community-acquired lower respiratory tract infections to Dr. Sami Ulus Pediatric Training and Research Hospital between December 2012 and February 2013. Exclusion criteria comprised a history of prematurity and chronic diseases such as congenital heart disease, chronic renal or pulmonary conditions, neurologic developmental delay, significant malnutrition, obesity, or immunodeficiency.

Data collection

Informed consent was obtained from the caregivers upon admission, followed by a structured interview using a standardized questionnaire. This questionnaire captured demographic data (age, gender), environmental factors (smoking exposure within the home, relationship of the smoker to the patient, and the number of cigarettes smoked daily in the home), and clinical parameters. A urine specimen was collected on admission day for cotinine analysis, which were stored at -20°C until analysis.

Laboratory analysis

Thawed urine samples were centrifuged at 3000 rpm for 5 minutes. Cotinine levels were quantitatively measured using the solid-phase competitive chemiluminescence immunoassay on an Immulite 2000 Analyzer Nicotine Metabolite device by Siemens, USA, as detailed by Florescu et al. (4). The cotinine threshold to differentiate between exposure and non-exposure was set at ≥10 ng/ml, based on manufacturer recommendations and corroborated by previous literature (5).

Statistical analysis

Statistical procedures were conducted using IBM SPSS Statistics for Windows, version 22 (IBM Corp., Armonk, N.Y., USA). Shapiro-Wilk test assessed the normality of data distribution. The Mann-Whitney U test analyzed non-normally distributed variables, and Pearson's chi-square test assessed categorical variables. Descriptive statistics are presented as mean, standard deviation, minimum and maximum for continuous variables and as frequencies and percentages for categorical variables. A p-value < 0.050 denoted statistical significance.

RESULTS

Among the initial 141 cases, 115 completed the questionnaire and provided urine samples. The average age of the 115 patients participating in the study was 6.13±5.75 months (1–23 months). Of these, 80 were male (69.6%). The questionnaire responses identified 86 patients (74.8%) as having exposure to environmental tobacco.

Urinary cotinine was positive in 40% of cases (n=46), and undetectable in 60% (n=69). When considering the daily number of cigarettes to which subjects were exposed, the frequency of urinary cotinine positivity increased with the number of cigarettes smoked daily. In particular, 53.4% of infants from households that smoked more than 20 cigarettes a day had positive cotinine levels. On the other hand, 33.3% of infants in households with 1-10 smokers per day and 10.3% of infants

Table I: The relationship between presence of continine in urine and the number of cigarettes consumed around infants

Cigarette consumption per day	Cotinine* (-)	Cotinine* (+)	p†
Total	69 (60)	46 (40)	
No consumption	26 (89.7)	3 (10.3)	
1-10	6 (66.7)	3 (33.3)	0.001
11-20	10 (52.6)	9 (47.4)	
>20	27 (46.6)	31 (53.4)	

**: n(%), †: Pearson Chi-Square test*

Table II: The relationship between smoking area and the presence of cotinine in urine in cases with cigarette exposure in the study questionnaire

Consumption area	Cotinine* (-)	Cotinine* (+)	p†
Total	43 (50)	43 (50)	
Next to	4 (40)	6 (60)	0.501
Other room / Balcony / Outside	39 (51.3)	37 (48.7)	

**: n(%), †: Pearson Chi-Square test*

Table III: The relationship between clinical parameters and presence of continine in urine

	Cotinine* (-)	Cotinine* (+)	p†
Oxygen saturation	94 (80-100)	93 (65-98)	0.038
Respiratory rate	60 (20-80)	60 (40-84)	0.719
CRP (mg/L)	9.7 (1-144)	15 (1-172)	0.214
Leucocyte count/mm^3	12100 (4200-25400)	10800 (10000-23800)	0.219
Hospitalization, days	6 (3-14)	6 (4-15)	0.819

**: median (min-max), †: Mann-Whitney U test*

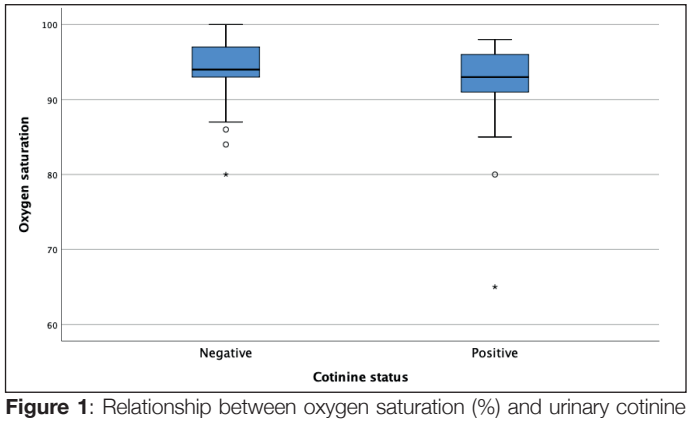

Figure 1: Relationship between oxygen saturation (%) and urinary cotinine in infants hospitalized due to community acquired lower respiratory tract infection (n = 115, p = 0.038, "○" signs for outliers, "*" signs for extreme values)

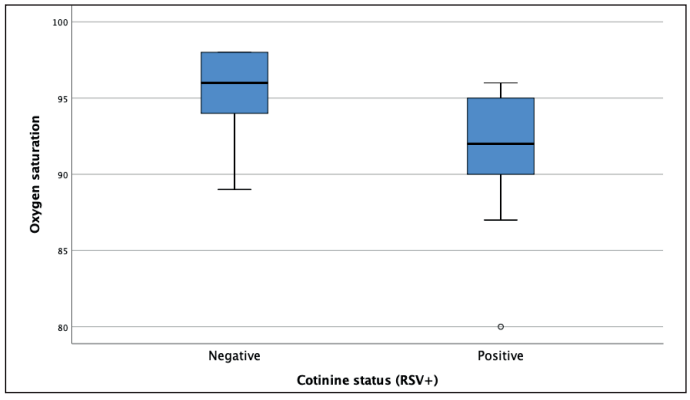

Figure 2: Relationship between oxygen saturation (%) and urinary cotinine in infants hospitalized due to RSV positive community acquired lower respiratory tract infection (n = 24, p = 0.015, "○" signs for an outlier)

in non-smoking households were positive for cotinine in urine (n=115, p=0.001; Table I).

Smoking sites were reported as follows: 11.6% (n=10) in the same room as the baby, 60.4% (n=52) on the balcony, or outside, and 28% (n=24) in another room. The frequency of cotinine positivity did not vary significantly based on the site of cigarette consumption. For example, 60% of infants from households where smoking occurred in the same room tested positive for cotinine, compared to 48.7% in households where smoking occurred outside, on the balcony or in another room (n=86, p=0.501; Table II).

Among all participants, infants with positive urinary cotinine levels had a lower oxygen saturation at admission (p=0.038; Table III, Figure 1). In contrast, no significant differences were found in respiratory rate (p=0.719), C-reactive protein (CRP) level (p=0.214), leukocyte count (p=0.219), or length of hospital stay (p=0.819) between cotinine-positive and cotinine-negative groups (Table III).

Forty-seven participants underwent viral PCR analysis of nasal swab samples, with 24 testing positive for Respiratory Syncytial Virus (RSV). In this RSV-positive subgroup, oxygen saturation levels at admission were significantly lower in the cotinine-positive group compared to the cotinine-negative group (p=0.015; Figure 2). No significant differences were observed in respiratory rate, CRP level, leukocyte count, or length of hospital stay between these groups.

DISCUSSION

Our findings reveal ETS exposure is associated with higher frequency of urinary cotinine positivity. Infants with positive cotinine levels had significantly lower oxygen saturation upon admission for community-acquired LRTIs. This association persisted even in the RSV-positive subgroup, where cotinine-positive group again exhibited lower oxygen saturation at admission. The confirmation of this result in the RSV-positive subgroup underscores the impact of tobacco smoke exposure on respiratory outcomes, independent of the infecting agent. These findings further strengthen the evidence that ETS exposure exacerbates respiratory distress in vulnerable populations.

The high prevalence of ETS exposure (74.8%) in our cohort mirrors findings from other studies in Turkey, highlighting a cultural pattern of tobacco use that significantly exceeds reported exposure rates in Europe and North America (9, 14). Such widespread exposure underscores the urgent need for tailored public health interventions to reduce tobacco use and mitigate its impacts on children. Moreover, the observed relationship between household tobacco consumption and positive cotinine levels reinforces the utility of cotinine as a biomarker for ETS exposure and its role in assessing associated health risks (13, 15). The discrepancy between self-reported exposure rates and cotinine levels may be explained by the interval of hospitalization during which the subjects spent time in a relatively smoke-free environment.

SHS exposure has been consistently associated with respiratory morbidities, including increased emergency department visits, recurrent wheezing, and chronic cough in children (7, 16). In our study, the frequency of cotinine positivity did not significantly differ based on the proximity of smoking to the child, indicating that even indirect exposure can lead to measurable biological effects. This finding highlights the role of a relatively new

concept, 'thirdhand smoke exposure,' which refers to the persistence of tobacco smoke residues on surfaces and fabrics. Children's natural behaviors, such as frequent hand-to-mouth activities and close contact with contaminated surfaces, make them particularly vulnerable to THS (4, 5). These findings align with emerging evidence that residual tobacco pollutants pose a significant risk not only through inhalation but also through contact with contaminated surfaces, even when smoking occurs away from children (4, 17). Additionally, non-airborne pollutants can persist long after active smoking has ceased, making it nearly impossible to avoid exposure even with ventilation or air conditioning (10). Public health campaigns should expand their focus to include education about THS and advocate for stricter smoke-free policies to protect children from both SHS and THS.

The finding that the cotinine-positive group had lower oxygen saturation at hospital admission suggests that ETS exposure exacerbates respiratory dysfunction in infants. Components of tobacco smoke are known to impair mucociliary clearance, increase oxidative stress, and trigger inflammatory responses in the respiratory tract, all of which can compound the severity of infections like RSV (12, 18, 19).

Our study contributes to the growing body of evidence linking ETS exposure to worsened clinical outcomes in infants with LRTIs. By utilizing cotinine as a biomarker, we were able to objectively measure ETS exposure, reducing reliance on potentially biased self-reported data. However, the discrepancy between self-reported exposure rates and cotinine levels highlights challenges in accurately quantifying ETS. Future research should adopt standardized questionnaires and explore additional environmental and genetic factors that may mediate the impact of ETS (20).

CONCLUSION

This study highlights the profound impact of ETS exposure on infant respiratory health, emphasizing the critical need for smoke-free environments. The link between positive cotinine levels and reduced oxygen saturation highlights the need for public health efforts to prevent tobacco exposure, especially in homes with infants. By addressing the risks of both SHS and THS, comprehensive strategies can help safeguard children from the short- and long-term health consequences of tobacco smoke exposure.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study protocol was approved by the Ethics Committee of T.C. Ministry of Health Zekai Tahir Burak Hospital Gynecology and Obstetrics (11.12.2012/84).

Contribution of the authors

Altuntaş C: Organizing, supervising the course of progress and taking the responsibility of the research/study, Taking responsibility in patient follow-up, collection of relevant biological materials, data management and reporting, execution of the experiments, Taking responsibility in logical interpretation and conclusion of the results, Taking responsibility in necessary literature review for the study, Taking responsibility in the writing of the whole or important parts of the study, Reviewing the article before submission scientifically besides spelling and grammar. ***Şenel S:*** Constructing the hypothesis or idea of research and/or article, Planning methodology to reach the conclusions, Organizing, supervising the course of progress and taking the responsibility of the research/study, Taking responsibility in logical interpretation and conclusion of the results, Reviewing the article before submission scientifically besides spelling and grammar ***Zorlu P:*** Constructing the hypothesis or idea of research and/or article, Planning methodology to reach the conclusions, Organizing, supervising the course of progress and taking the responsibility of the research/study, Taking responsibility in logical interpretation and conclusion of the results, Reviewing the article before submission scientifically besides spelling and grammar.

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Conflict of interest

The authors declare that there is no conflict of interest.

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