Impact of Passive Smoking on Lung Function and Asthma Severity in Children

Impacto del tabaquismo pasivo en la función pulmonar y gravedad del asma en la población pediátrica

To the Editor,

Smoking is a large-scale health problem. According to the World Health Organization, 40% of children are exposed to passive smoking in the home.\(^1\)

The prevalence of childhood asthma in Spain is estimated at 10%.\(^2\) Bronchial hyper-reactivity in these children makes them more vulnerable to environmental pollutants, such as tobacco smoke, which increases airway inflammation, bronchial secretion, and airflow limitation.\(^3\)

A multicenter study conducted by the Working Group on Smoking in Childhood and Adolescence of the Spanish Society of Pediatric Pulmonology found a prevalence of passive smoking of 37%, which was associated with exacerbations among children with asthma.\(^4\)

Few publications in our setting have objectively studied changes in lung function parameters and the severity of acute asthma exacerbations among asthmatic children exposed to passive smoking.\(^5\)–\(^7\) Lung function impairment is known to be more common among infants born to mothers who smoke,\(^8,\)\(^9\) and children exposed to tobacco during their development have altered lung capacities and a greater risk of developing asthma.\(^10,\)\(^11\)

In this study, we analyzed the prevalence of passive smoking in asthmatic children and its effect on the severity of acute episodes and lung function. This was a retrospective analysis of patients aged 4–16 years who required hospitalization for acute asthma in the Hospital General Universitario Gregorio Marañón, Madrid, Spain, from 2011 to 2015. Asthma diagnosis, severity of exacerbations, and changes in lung function, such as FEV1/FVC ratio <80% according to the GEMA 2017 guidelines, were evaluated.\(^12\) Most variables included in the statistical analysis did not show a normal distribution. The statistical tests used were the Mann–Whitney U test and Kruskal–Wallis for continuous variables, and Fisher’s test or chi-squared test for categorical variables. The study was approved by the Clinical Research Ethics Committee.

The analysis included 365 patients. Median age was 5 years (IQR 4–7) and median length of stay was 4 days (IQR 3–5); 63% of the children were boys, and 45% had a previous diagnosis of asthma. Median body mass index (BMI) was 16 kg/m\(^2\). Concomitant atopy was diagnosed in 65%. Forty-one percent (n=151) were passive smokers (6%, mother smoked; 14%, father; 19%, both; 2%, other family members).

Table 1 shows the analysis of some severity markers observed in asthmatic children, according to exposure to passive smoking. Patients exposed to passive smoking had lower oxygen saturation levels on admission, higher scores on the asthma exacerbation severity scale, worse lung function parameters, and more visits to emergency departments in the previous months (P<.05).

Asthma exacerbations were more severe when the father was the smoker (PS 6; IQR 4.2–6.7), compared to the mother or both parents (PS 5; IQR 4.6; P=.05). Moreover, passive smokers had significantly more previous episodes of bronchitis (68% vs 56%, P=.02; OR 1.5) and bronchospasm (80% vs 65%, P=.02).

Spirometry was performed in 55% of the children within 2 months following the acute episode to provide information on their baseline asthma severity,\(^1\) and lung function impairment was detected in 25% (n=51).

In the group of asthmatic children exposed to smoking, the FEV1/FVC ratio was lower if the smoker was the mother (median 86%, IQR 83%–90%) than if the father was the smoker (89%, 85%–105%), both parents were smokers (87%, 77%–92%) or neither smoked (89%, 80%–95%), although these differences were not significant (P=.07). The proportion of cases with impaired lung function (FEV1/FVC<80%) was greater if both parents smoked (33%), than if only the father (7.7%) or the mother (13.3%) smoked (P=.03), irrespective of severity.

A multivariate analysis was performed to evaluate the association between independent variables such as age, sex, and passive smoking, with impaired lung function as the dependent variable. The logistic regression model showed that the absence of passive smoking was an independent protective factor against impaired lung function (P=.029; OR 0.44; 0.204–0.919). A second multiple linear regression model was used to evaluate the association between age, sex, atopy, and passive smoking, with the severity score as the dependent variable. This showed that passive smoking (β 0.2, 0.032–0.98; P=.037) and atopy (β 0.3, 0.2–0.98; P=.002) were associated with higher severity scores.

With respect to tobacco exposure in the home, while the literature has placed greater emphasis on the role of the mother,\(^1\)\(^3\) we found that asthma exacerbations were more severe when the father was the smoker (P=.05). This may be related with differential

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<th>Table 1 Bivariate Analysis of Severity Parameters of the Acute Asthma Episode in Study Patients (n=365) Associated With Passive Smoking.</th>
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* Pulmonary Score (PS): scale to evaluate severity of the asthma exacerbation, used in our study.\(^12\) A score of 0–3 is assigned to each of the sections analyzed (breathing, wheezing, and use of accessory muscles), providing a score of 0–9. Mild exacerbation: 0–3; moderate: 4–6; severe: 7–9.

| *P* values: 0.001; 0.01; 0.05; 0.10; 0.20; 0.50; 1.00. |

\(^{a}\) Data expressed as a percentage of patients who attended at least 1 emergency room visit in the same hospital for any asthma-associated event in the 3 months prior to the exacerbation for which they were admitted. The remaining data in the table are expressed as median and interquartile range.

\(^{b}\) FEV1: forced expiratory volume in 1 second; FVC: forced vital capacity. Obstruction in children is defined as a FEV1/FVC ratio <80%.\(^12\)

\(^{c}\) Statistical significance. A P-value of <.05 was considered statistically significant.
Anisocoria Associated With Spontaneous Pneumomediastinum

Anisocoria como síntoma asociado a neumomediastino espontáneo

To the Editor,

Pneumomediastinum is defined as the presence of free air in the mediastinum. This is a rare manifestation and usually presents spontaneously, as a consequence of injury, rupture of a hollow viscus, or gas-producing infection. It generally occurs in young adults exposed to a sudden change in intrathoracic pressure that results in the rupture of alveolar septa and alveoli, causing air to escape from the pulmonary interstitial tissue to the peribronchial and perivascular tissues of the upper mediastinum and the neck. In clinical practice, it is often the result of precipitating factors such as previous muscle exertion (physical exercise, coughing fit, or asthma attack), which lead to a Valsalva maneuver or an increase in intrathoracic pressure. In many cases, it is difficult to differentiate spontaneous pneumomediastinum from more subtle causes of secondary pneumomediastinum, such as esophageal perforation, small tears in the central tracheobronchial tree, or lung or mediastinal infections.

The most commonly described symptom is central chest pain, which may radiate to both sides of the chest and the neck. Dyspnea and irritative cough may also appear. Dysphagia, hypernasal speech, and tachycardia are less common. The classic triad of spontaneous pneumomediastinum consists of chest pain, dyspnea, and subcutaneous emphysema. However, no symptoms associated with compression of the cervical neurovascular bundle (pupillary changes, loss of visual acuity, headache, etc.) have been described in the literature.

We report the case of a patient with a diagnosis of spontaneous pneumomediastinum associated with anisocoria.

This was a 19-year-old man, with no significant clinical history or known toxic habits, who attended the emergency room due to a 12-h history of cervical neck pain associated with central chest discomfort, and a “crackling” sound on palpation of the neck. He reported watery rhinitis in the previous days, and 2 episodes of

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References