

Occupational asthma due to colophony in a violin player

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Clinical Implication

- This is the first report of occupational asthma caused by colophony in a musician and one of the few due to unheated colophony. The case highlights that occupational asthma caused by this agent does not solely affect industrial workers.

TO THE EDITOR:

Colophony (rosin) is a common cause of occupational asthma and occupational contact dermatitis.¹ It is derived from pine trees and consists of a mixture of resin acids and neutral substances. Occupational asthma usually is caused by inhalation of fumes from heated colophony. This form of colophony is frequently used by solderers in the electronics industry² or as hot melt glue.³ However, occupational asthma caused by unheated colophony dust is very rare.⁴ String instrument players use the unheated colophony on their bows to enhance the sound by increasing friction between the bow and the strings. Allergic contact dermatitis has been described in a violoncellist,⁵ but occupational asthma due to colophony has not yet been reported in musicians. Because no specific IgE for colophony has been identified, the diagnosis of allergic sensitization to colophony has to be made on a clinical basis. The specific bronchial challenge test is a useful tool to confirm the diagnosis in case of suspected occupational asthma.⁶

CASE REPORT

Our patient, a now 37-year-old woman, played the violin since age 7. She was 35 years old and working as a first violin player (concertmaster) in a famous Belgian orchestra when the first respiratory symptoms occurred. She consulted a respiratory physician for rhinitis, chronic dry cough, dyspnea, wheezing, and chest tightness. Spirometry, lung volumes, and diffusion capacity were completely normal, but the histamine bronchoprovocation test showed significant bronchial hyperresponsiveness with a provocative histamine dose, PD₂₀ FEV₁ of 1.22 mg. Skin prick tests confirmed allergy to house dust mite, grass pollen, and tree pollen. The patient was diagnosed with atopic asthma and allergic rhinitis, and was advised on allergen avoidance. Treatment with low-dose inhaled corticosteroid—long acting β -agonist fixed combination and montelukast was initiated. Despite the maintenance treatment, the patient remained symptomatic, especially during violin playing. A wrist fracture forced her to stop playing the violin temporarily, and a prompt improvement in asthma symptoms was seen. This led to the hypothesis of occupational asthma due to allergy to colophony.

TABLE I. Results of the diagnostic workup

	Day 1 ("sham")	Day 2 (colophony)	Day 3 (control)
PEF (L/min)	600	600	620
PEF diurnal variability (%)	6.9	18.2	5.0
FEV ₁ baseline (L)	3.79	3.77	3.63
FEV ₁ baseline (L) (% pred)	110	110	106
FEV ₁ response (L)	−0.01	−1.11	—
FEV ₁ response (L) (% change)	−0.3	−30	—
FEV ₁ diurnal variability (%)	9.4	22.6	7.7
PC ₂₀ FEV ₁ histamine (mg/mL)	2.91	0.436	0.155

PEF, peak expiratory flow.

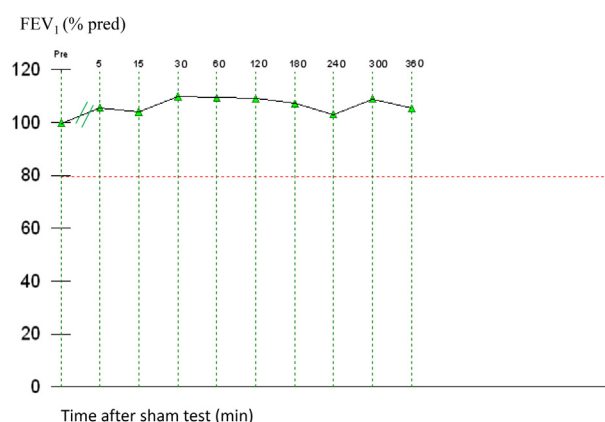


FIGURE 1. FEV₁ (% pred) until 360 min after the "sham" test. "Pre" represents the baseline value. On the x axis, the time line after exposure is shown, starting 5 min after completion of the challenge test.

A 3-day diagnostic workup was scheduled after stopping all asthma maintenance drugs. The results are shown in Table I. At the time the tests took place, the patient had not played her violin for more than a year. On the first day, results of routine lung function testing proved normal, and mild nonspecific histamine bronchial hyperresponsiveness was measured (provocative histamine concentration, PC₂₀ FEV₁ of 2.91 mg/mL). For the "sham" bronchial challenge test, the patient handled lactose powder to make it airborne. Exposure times were increased progressively, and the control test results remained negative (−0.3% change in FEV₁ after 20 minutes). Diurnal variabilities of peak expiratory flow (PEF) and FEV₁ also were normal (6.9% and 9.4%, respectively). There was no late asthmatic response (FEV₁ and PEF were monitored up to 6 and 24 hours, respectively) (Figure 1). On the second day, a realistic specific bronchial challenge test with colophony was performed. The patient rosined the bow and played the violin for several consecutive periods of increasing duration, each followed by a spirometry. This time a dramatic decrease in FEV₁ (−30%) was seen after a cumulative exposure time of 61 minutes (Figure 2). Also, nonspecific bronchial hyperresponsiveness to histamine at the

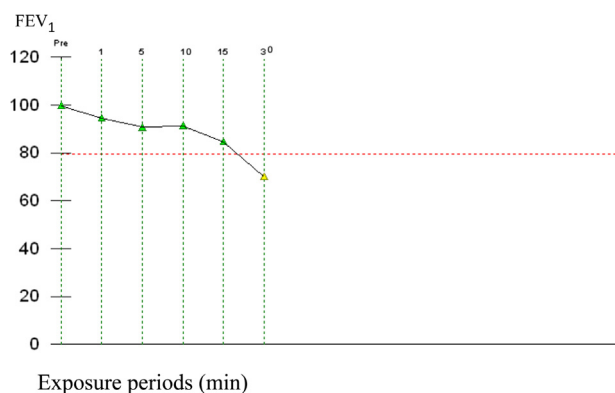


FIGURE 2. FEV₁(% pred) vs consecutive periods of exposure (min). The cumulative exposure time was 61 min.

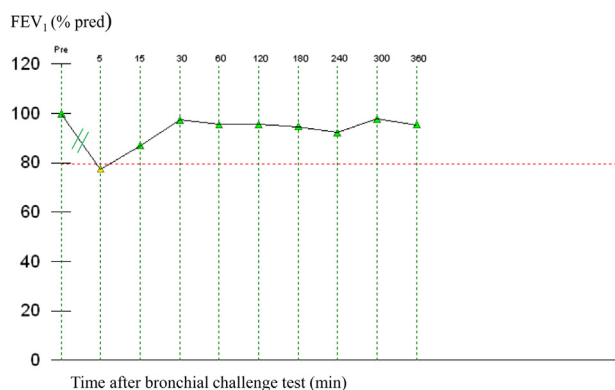


FIGURE 3. FEV₁(% pred) until 360 min after the specific bronchial challenge test with colophony. Same representation as in Figure 1.

end of day 2 (PC₂₀ FEV₁, 0.436 mg/mL) as well as PEF and FEV₁ variability (18.2% and 22.6%, respectively) were increased. No delayed bronchoconstriction was observed (Figure 3). On day 3, the situation returned to normal, except for the persistence of nonspecific bronchial hyperresponsiveness to histamine (PC₂₀, 0.155 mg/mL). This extensive testing procedure confirmed the diagnosis of occupational asthma caused by

colophony. The patient quit violin playing and started a college education in psychology, without the asthma recurring.

DISCUSSION

Although occupational asthma due to colophony is common in various industries and professions, this is the first report of a string instrument player and one of the few reports with unheated colophony. The realistic specific bronchial challenge test confirmed the diagnosis, which in this case forced the patient to a career change. The case also demonstrated a long-time lapse for sensitization because the interval between the first exposure and the onset of symptoms exceeded 25 years, which is longer than observed in electronics factory workers.⁷ Bronchial hyperresponsiveness already increased shortly after the allergen challenge and persisted on the day after, which suggests increased airway inflammation due to re-exposure to the allergen during the specific bronchial challenge test.

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