

Occupational Asthma



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INSTRUCTIONS

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1. Review the target audience, learning objectives, and author disclosures.
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Overall Purpose/Goal: To provide excellent case-based reviews on key aspects of allergic disease to those who research, treat, or manage allergic disease.

Target Audience: Physicians and researchers within the field of allergic disease.

Accreditation/Provider Statements and Credit Designation: The American Academy of Allergy, Asthma & Immunology (AAAAI) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The AAAAI designates this journal-based CME activity for 1.0 AMA PRA Category 1 Credit™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

List of Design Committee Members: Nabeel Farooqui, MD, and David Peden, MD

Learning objectives:

1. To recognize the causes and clinical presentation of occupational asthma.
2. To describe the approach to diagnosing suspected cases of occupational asthma.
3. To identify management options for asthma in the workplace.

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The Difficult Cases Feature is based on the *Difficult Cases* Course presented at the AAAAI Annual Meeting and coordinated by the AAAAI New Allergist-Immunologist Assembly (NAIA). View the entire presentation and obtain CME or MOC credit by visiting the *JACI: In Practice* homepage www.jaci-inpractice.org.

Key words: Asthma; Workplace; Occupational

A 63-year-old woman presented to the clinic with complaints of nasal congestion, recurrent cough, wheezing, and exertional dyspnea. Her symptoms first started 5 years ago, before which she was healthy, and have progressively worsened since. For the

past 35 years, she has worked at her family auto dealership, with primary duties consisting of back office paperwork. She experiences onset of breathing symptoms daily around noon, which begin to improve once she goes home. By nighttime, she feels much better and denies nocturnal awakenings. She has tried antihistamines with no improvement. The patient was recently on vacation and was able to walk long distances without experiencing much respiratory impairment. Her physical examination revealed mild congestion of nasal mucosa with no wheezes or rhonchi on chest auscultation. Skin prick testing was positive for dust mites, and chest radiography was normal, as were spirometry loops and lung volumes. She was started on nasal mometasone, fluticasone/salmeterol 250/50 mcg, and albuterol. On follow-up visit 6 weeks later, the patient's condition had slightly improved, with symptoms reduced to occurring only 3 days out of the week. However, she was requiring rescue albuterol multiple times on her symptomatic days and had mild expiratory wheezing during her visit. Spirometry had worsened

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from initial presentation, demonstrating FEV₁/FVC 68% and FEV₁ 71%. Inspection of the patient's workplace revealed communicating air ducts between her office and an auto body shop, which was built adjacent to the dealership 10 years earlier. Evaluation of the Material Safety Data Sheets from the supply inventory suggested hexamethylene diisocyanate (HDI) found in polyurethane paint as the suspect allergen causing the patient's respiratory inflammation. The patient was advised to move her office to one furthest from the body shop, and additional outflow ventilation was installed throughout the dealership to minimize air contamination. Over the ensuing months, the patient experienced significant reduction in her cough and dyspnea with corresponding improvement in lung function.

This patient presented with adult-onset asthma after years of exposure to a common sensitizing agent (HDI) encountered at her workplace. Occupational asthma (OA), one subcategory of the broader term, work-related asthma, is the *de novo* development of asthma subsequent to a workplace exposure. The other major phenotype, work-exacerbated asthma, describes patients with pre-existing asthma triggered by a workplace exposure.¹ Substances responsible for inducing OA are high-molecular-weight proteins and polysaccharides (eg, flour, animal proteins, enzymes, latex), and low-molecular-weight chemicals and metals (eg, isocyanates, wood dusts, metal salts, cleaning agents). These agents are capable of stimulating immunologic responses through IgE and non-IgE-mediated mechanisms, respectively.²

Specific inhalation challenge is considered the reference standard test for OA, but is costly and difficult to obtain. One

approach is to first establish the diagnosis of asthma, through clinical evaluation and assessment of bronchial hyper-responsiveness (BHR), followed by linking respiratory symptoms with the work environment.³ The absence of BHR after a person has worked for 2 weeks under his or her usual work conditions virtually excludes OA. Serial peak flow measurements, at and away from work, for a 4-week period are useful in objectively demonstrating workplace symptom exacerbation.

The majority of patients with OA will develop permanent lung impairment, with persistent BHR even when no longer exposed to their sensitizing agent.⁴ Therefore, early recognition and workup of OA is recommended so appropriate management, including evaluation and/or removal from workplace, can be pursued.

Further information on this case can be found in this article's Online Repository at www.jaci-inpractice.org.

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