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Dear Healthcare Provider:

This Occupational Lung Disease Bulletin was written by two physicians during their rotations at OHSP while studying occupational medicine at the Harvard School of Public Health. They posted a version in the New England College of Occupational and Environmental Medicine newsletter. We appreciate their contributions to OHSP.

To receive your Bulletin by e-mail, please send a message to <u>occupational.asthma@state.ma.us</u>. Sincerely, Elise Pechter MPH, CIH

Evaluating a Patient Exposed to Mold Stasia Muhlner, MD, MPH Yolanta Petrofsky, MD, MPH¹

A 32-year-old female logistic supply administrator presents to an occupational/environmental medicine clinic with a history of asthma symptoms that had progressively worsened over the past few months. She is concerned about mold exposure at the paper warehouse where she started working ten months ago and states "my workplace made me so sick that I had to quit." The building is a 1940s era brick building leased by three different occupants in an open space, with broken original floor tiles, a leaking roof, and water-stained walls. She describes her office as a damp open corner of the warehouse adjacent to the delivery truck loading docks. Her only fresh air source is a window that opens toward the loading dock. The warehouse has many openings to the outdoors and is infested with nesting and breeding pigeons and rodents.

The patient states that she was having shortness of breath at work and at home, and would return home with visible black particulate surrounding her nostrils. She states that co-workers complained of the black-tinged mucous as well. The patient had been working at the warehouse for about five months before developing pneumonia.

Both her primary care provider and pulmonologist concurred that her asthma was exacerbated by her work environment and wrote a note to the company recommending her removal from the warehouse. Her company tried to relocate her to a trailer outside of the building. However, she was still near the warehouse and still had to travel through the warehouse and loading dock frequently, and her symptoms did not improve. July 2011

The company hired a reputable company to do an environmental assessment. The patient brought in the environmental sampling results to her office visit. The report showed elevated levels of *Stachybotrys* inside the warehouse compared to outside of the building. She also showed 20 photos of water-stained walls, nesting pigeons, and forklifts and tractor trailers idling during cargo transfer.

Physical Exam is normal. Allergen profile for regional allergen zone 1 RAST Panel demonstrated moderate elevations in IgE (class II-IV), implying sensitization to dust mites, various grasses, but no elevation in the common mold species. CBC did not show elevation of eosinophils.

This case illustrates the difficulty that physicians face in finding the specific cause for a patient with work-related asthma. Potential exposures for this worker include mold, dust, diesel exhaust, pigeons, and rodents. Although the patient did not have a positive allergy test to mold, the discussion below highlights potential limitations of allergy testing, and also points out other asthmagens that may be found in damp places. Ultimately the patient quit her job, and her symptoms improved when away from her work environment.

Dampness and Mold-Related Asthma

In 2004 the Institute of Medicine reviewed the scientific literature on damp environments and mold. In addition to 200 common species of mold, damp conditions give rise to bacterial growth, mycotoxins, dust mites, cockroaches, chemical leaching from building materials and volatile organic compounds. These may cause allergy-like symptoms, such as eye irritation, rashes, cough and chest tightness. The IOM concluded that there is sufficient scientific evidence linking damp environments and mold to upper respiratory symptoms, cough and wheeze. There is limited or suggestive evidence of an association between dampness and dyspnea. However, there is inadequate evidence to determine whether an association exists between dampness or mold and airflow obstruction in otherwise healthy people. Both dampness and mold can induce symptoms in sensitized people (sufficient evidence). If a patient is allergic to mold, resultant allergic inflammation can cause new onset asthma or exacerbate pre-existing asthma. (See Prevention on page 2.)

Medical Evaluation and Exposure Reduction

Evaluation of a worker with suspected mold- or dampness-related asthma should begin with an

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¹ With review by, and thanks to, Dr. Rose Goldman.

occupational and environmental history. Evaluation proceeds to the diagnosis of asthma. In the presence of airflow obstruction as seen by a reduced FEV₁/FVC ratio, pulmonary function tests (PFT) pre- and postbronchodilator should be performed to assess for reversibility after inhalation of a short-acting bronchodilator.² If there is no evidence of airflow obstruction or reversibility on PFT in a worker who has asthma-like symptoms, a methacholine challenge can be performed shortly after exposure or a symptomatic period to test for bronchial hyperresponsiveness. To determine workplace causation, the physician can evaluate the patient with physical exam and PFT after two days away from work (pre-shift) and again after re-exposure to the work environment at end of day (post-shift). Alternatively, the worker can measure his/her peak expiratory flow rate (PEFR) at least four times/day for at least two weeks while working and at least two weeks while not working. If the patient has late phase asthma, hyper-sensitivity pneumonitis, or chronic rhinitis, a longer removal from work may be required to evaluate symptoms away from work. PEFR should be done in a structured manner with charting on a log, with repeated blows each time to be able to check for consistency.

IgE testing via skin prick, intradermal, or immunoassay blood test (RAST or ELISA) can indicate sensitization and suggest an immunologic (allergic) mechanism for moldinduced asthma. Unfortunately, mold antigen reagents have not been standardized and are not well-identified nor purified; therefore, skin testing with a higher concentration of antigen or contamination from other irritants or histamine releasers can create false positives. Immunoassay testing has increased false negatives as well if paticular antigens are excluded. Mold species produce several different allergens, depending on the strain cultured, the growth conditions, and the life cycle stage, and there is substantial cross-reactivity among mold allergens. In addition, it is important to remember that individuals can develop antibodies to specific allergens (sensitization) without developing the manifestation of clinical allergic disease, and so a positive test is not diagnostic of disease. Patients with a strong history of extensive systemic reactions may have negative skin test results; skin prick testing can also cause anaphylaxis in very sensitive patients. In addition, some individuals can have allergic illness mediated through non-IgE mechanisms which may not be detected by the above tests.

Treatment of work-related asthma begins with reduction of exposure, and in this case would include temporarily removing the worker from the work environment while the area is remediated to eliminate mold, dust and dampness. This should include removing the water source and porous materials that have remained wet for > 48 hours, improving ventilation, repairing areas of water intrusion, and removing mold and infestations. Individuals who have

become sensitized should not be involved in the abatement. Remediators should use, at a minimum, a fitted respirator with N95 protection. If exposure reduction does not improve symptoms, the worker should be relocated to another area or, as a last resort, removed from work entirely. Shorter durations of exposure correlate with more complete resolution of symptoms.

Prevention

Prevention of asthma is key. Once a worker has developed (or exacerbated) asthma, they may still have asthma symptoms due to hyperreactive airways, even if they are no longer exposed to the original trigger. Finding and removing any water source or water damage to walls, carpets and furniture, and keeping the building below 50% relative humidity will prevent mold growth. If mold growth or water damage is not clear, environmental sampling including air sampling, surface sampling, bulk sampling of building materials and water sampling from condensate drain pans or cooling towers may be helpful. However, the EPA and other experts discourage air sampling to initially address concerns about indoor mold.^{3,4} Failure to find mold may reflect limited test methods and may not address all the hazards associated with dampness. Therefore, negative results may be falsely reassuring.

If air sampling is done and shows increased mold indoors compared to outdoors or a proliferation of one specific indoor mold, there may be an indoor source of mold growth. However, sampling must be done by experienced professionals with proper protocols to demonstrate mold growth. Post-remediation sampling may be helpful to determine effectiveness of control measures.

Maintaining HVAC systems is also important because they can harbor mold or bacterial growth and because they can disperse them throughout the indoor environment. It has been shown that maintaining HVAC systems has improved upper respiratory symptoms of building occupants.

In summary, the patient with respiratory symptoms and exposure to dampness and mold on the job presents a challenge to the healthcare provider. A diagnosis of asthma may be straight forward. However, connecting the diagnosis to mold or dampness in the work environment may be challenging—because of limitations of both the biological testing and environmental sampling. Better understanding of these challenges can help providers make accurate diagnoses and appropriately manage care. A clinician's letter removing a patient from a hazardous environment and requesting that an employer abate the problem can be very influential.

References available by request.

² Determined either by an increase in FEV₁ of \geq 12 percent from baseline or by an increase \geq 10 percent of predicted FEV₁.

³www.epa.gov/mold/moldcourse/chapter3/lesson3.html www.epa.gov/mold/mold_remediation.html

⁴ Storey E et al. Guidance for clinicians on the recognition and management of health effects related to mold exposure and moisture indoors. 2004 <u>http://oehc.uchc.edu/clinser/mold%20guide.pdf</u>