

OCCUPATIONAL ALLERGIC RHINITIS IN A LABORATORY WORKER DUE TO MOULD CONTAMINATION IN A WATER-DAMAGED HOSPITAL BUILDING

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ABSTRACT

Mould exposure from water-damaged buildings and indoor dampness has been associated with allergies, infections and irritation, however, the immune mechanisms are complex. Occupational rhinitis is referred to as an inflammatory disease of the nose characterised by intermittent or persistent symptoms (nasal congestion, sneezing, rhinorrhoea, itching) and/or variable airflow limitation and/or hypersecretion attributable to a particular work environment and not to stimuli encountered outside the workplace. The purpose of this case report is to highlight the challenges in evaluating the relationship between mould exposure in water-damaged buildings and suspected allergic rhinitis.

The case report is one of probable occupational rhinitis caused by sensitisation to *Alternaria alternata* and *Aspergillus sp.* The 52-year-old patient experienced recurring nasal polyps and sinus infection and underwent several operations. Patient serum tests to the mould mixture and specific moulds using the UniCAP system (Pharmacia, Sweden) revealed moderate to elevated IgE levels for various mould allergens. Paint scrapings and swab samples collected from her work environment revealed the presence of *Aspergillus sp.* and *Alternaria* amongst other moulds, which she was sensitised to. Her condition improved after the building was repaired.

INTRODUCTION

Allergic rhinitis is the most common chronic disease caused by a nasal reaction to allergens, (e.g. pollen and fungal spores) through an IgE-mediated inflammation.^{1,2,3} The prevalence of allergic rhinitis is increasing globally⁴ affecting approximately 10-40% of the population¹ and is a common and persistent disease in South Africa.^{4,5}

Work related rhinitis (Figure 1)⁶ can be occupationally caused as a result of exposure to specific agents in the work environment or work-exacerbated when pre-existing rhinitis is worsened by non-specific workplace exposures. These could be irritant agents, (e.g. chemicals, dusts, fumes), physical factors (e.g. temperature changes), emotions, second hand smoke and strong smells (e.g.

perfumes).⁶ Symptoms can be intermittent or persistent and include nasal obstruction (congestion or blockage), sneezing, rhinorrhoea, nasal itching and post nasal drip.^{1-2,5-8} Nasal congestion is the most common and troublesome symptom which occurs in up to 90% of patients.^{3,7} Patients can also suffer from insomnia, fatigue, irritability, memory and/or learning deficit.³

There is increasing evidence that the work environment contributes significantly to the burden of rhinitis and asthma worldwide, negatively affecting the quality of life, work ability and socioeconomic status.⁹

Various studies have reported an association between exposure to fungal (mould) spores and allergic rhinitis.¹⁰

Excessive moisture in buildings promotes mould growth and is associated with an increased prevalence of allergy, infection and irritation.¹¹ The most common airborne moulds implicated include species of *Alternaria*, *Aspergillus*, *Cladosporium* and *Penicillium*. Atopic individuals exposed to aerosolised moulds develop an IgE-mediated response due to spores infiltrating the sinuses.^{12,13} A study conducted by Seedat et al. at Universitas Academic Hospital in Bloemfontein reported a high prevalence of sensitisation to moulds (26%) in patients with allergic rhinitis.⁷ Studies in the United States and elsewhere, report that sensitisation to *Alternaria* is common¹⁴ and is strongly associated with allergic rhinitis.¹⁰ Evaluations in clinical practice are challenging since damp building related illnesses have similar symptoms to other clinical conditions such as rhinitis, asthma, hypersensitivity pneumonitis, allergic bronchopulmonary aspergillosis and allergic fungal sinusitis (AFS).^{15,16} In general, the diagnosis of allergic rhinitis is based on a positive history, clinical signs, assessment of atopy, initial *in vitro* or *in vivo* screening (measurements of specific IgE antibodies and/or by skin prick tests (SPT) with allergens. For example, fungal extract mixtures using the most common species (*Penicillium notatum*, *Cladosporium herbarum*, *Aspergillus fumigatus*, *Alternaria alternata* and *Candida albicans*) known to induce allergic reactions. This should be followed by confirmatory tests or nasal provocation challenges, e.g. with single specific mould extracts to define the specific cause of sensitisation.^{5-7,9} In an individual with symptoms of rhinitis and a suggestive occupational history, accompanied

by positive immunological tests to the specific allergen known to be present in the workplace, a diagnosis of probable occupational rhinitis should be considered.⁹ The purpose of this case report is to present a case of probable occupational allergic rhinitis due to mould exposure in a laboratory known to have a leaking roof predisposing it to damp and mould contamination.

CASE REPORT

PATIENT HISTORY

The patient is a 52-year-old female with atopic asthma. She is a non-smoker and has been employed for 5 years as a manager in a medical laboratory which is situated inside a public hospital building. Her main job task is managing and working in the haematology laboratory. In November 2012, she presented to the National Institute for Occupational Health (NIOH) Clinic complaining of intermittent blocked nose and headaches. She was using a corticosteroid nasal spray (27.5 mcg fluticasone furoate). She reported suffering from allergic symptoms for the past 10 years but her symptoms flared up in the preceding 3 years of her current presentation. The symptoms usually lasted for a number of hours while at work and decreased substantially when at home. She had consulted an otolaryngologist since 2009 and was diagnosed as having chronic allergic fungal sinusitis (AFS). On further enquiry there appeared to be insufficient clinical evidence from the clinician to support this diagnosis (information of findings from CT scan, nasal polyp histology, etc. were not reported).^{17,18} She experienced recurring nasal polyps and sinus

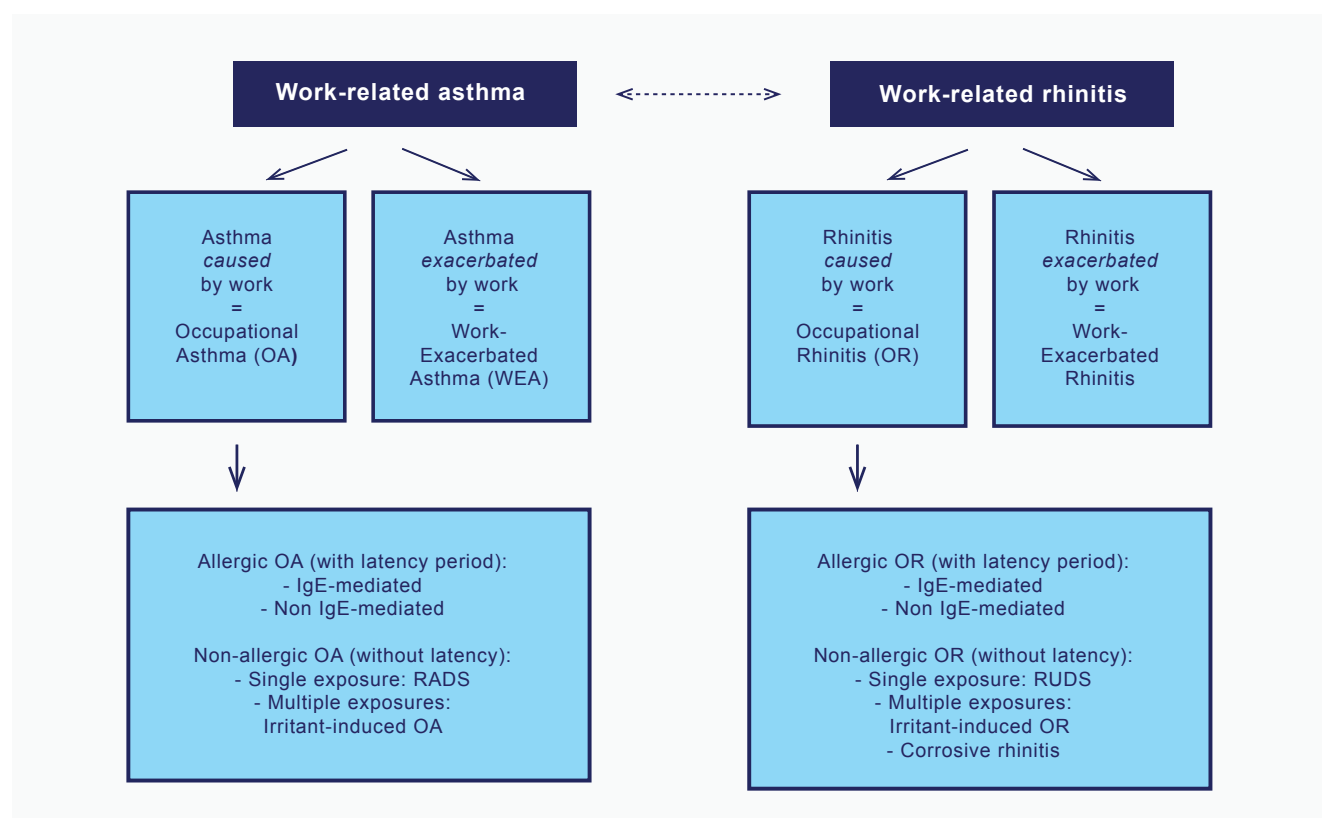


Figure 1: Classification of occupational rhinitis and asthma⁶

TABLE I: PATIENT SERUM IgE PROFILE FOR SENSITIVITY TO MOULD MIXTURE INHALANT AND SPECIFIC MOULD ALLERGENS BEFORE (2012) AND AFTER (2015) WATER DAMAGE REPAIR WORK

TEST	PATIENT IgE LEVELS (IU/ML)				CUT OFF RANGE (IU/ML)
	2012	INTERPRETATION	2015	INTERPRETATION	
TOTAL IgE	282	high	ND	ND	0.0-22*
MOULD MIXTURE (<i>P.notatum</i> , <i>C.herbarum</i> , <i>A.fumigatus</i> and <i>A.alternata</i>)	12.4	high	ND	ND	<0.35
ASPERGILLUS#	1.63	moderate	0.26	Very low	<0.35
ALTERNARIA ALTERNATA	12.5	high	5.74	Moderate	<0.35

* Age of 10 years and older; ND: not done; # *Aspergillus fumigatus*

infection according to the clinician's report. The report also stated that she had undergone repeated surgery to her sinuses and had been on corticosteroids (nasal spray) for extended periods to alleviate her symptoms. In 2012, the patient underwent two operations and had her blood tests referred to a laboratory for further analysis.

IMMUNOLOGICAL ASSESSMENT: SERUM IgE

As part of the clinical evaluation, IgE to inhalant allergens (mould mixture) and specific serum IgE tests were performed to determine the patient's sensitisation to various mould species. The inhalant screening laboratory results using the UniCAP system (Pharmacia, Sweden) revealed moderate to elevated IgE levels for various allergens tested (Table I). The mould mixture used for testing consisted of *Penicillium notatum*, *Cladosporium herbarum*, *Aspergillus fumigatus* and *Alternaria alternata* mould species.

EXPOSURE ASSESSMENT OF THE PATIENT'S WORKING ENVIRONMENT

a. Paint scrapings

The building walkthrough inspection conducted by the employer's safety health and environment (SHE) team and management revealed evidence of structural damage from water leakage on the roof (Figure 2). The laboratory is situated in a public hospital building. Paint scrapings collected in July 2012 from different areas in the laboratory were cultured on Malt Extract Agar (MEA) plates and incubated at 25°C ±2 for 5-7 days for mould presence. *Penicillium* and *Cladosporium* species were isolated in the tearoom; *Ulocladium* species from the TB room; *Aspergillus nidulans* in the receiving office; *Cladosporium* and *Syncephalastrum* species from the air conditioner in the administration area; *Alternaria* and *Moniliella* species from the passage and *Aspergillus terreus* in the patient's office.

b. Air sampling

Air samples were collected in July 2013 from selected rooms within the laboratory complex including the microbiology

and flow cytometry laboratories, storeroom, the patient's office and tearoom. Duplicate samples were collected using the MAS 100 air sampler (Merck Pty; Germany) by impaction method on Malt Extract Agar (MEA) at a flow rate of 100 L/min. The plates were incubated at 25-30°C for 7 days and moulds were identified to genus level using lactophenol staining and microscopy. All indoor fungal counts were less than outdoors, except the tearoom. *Cladosporium* species was isolated from both indoor (all laboratory areas sampled) and outdoor samples, with the tearoom levels being higher than other rooms. Other moulds that were identified were *Penicillium* from the other rooms and *Curvularia* species from the patient's office.

A clinical diagnosis of probable occupational allergic rhinitis was made at the time and the leaking roof was repaired. In 2014, she presented to the otolaryngologist for a follow up and reported a significant improvement in her symptoms and decline in her specific IgE results in 2015. She was negative to *Aspergillus* and the IgE levels for *Alternaria alternata* reduced from 12.5 to 5.74 IU/ml. She is still using a corticosteroid (fluticasone) nasal spray when necessary, but currently has no other respiratory symptoms. The nasal provocation test was not performed for this patient, hence a diagnosis of probable allergic rhinitis was concluded. The current condition is compensable according to COID Act circular instruction No 27216 of 2005 with 15% disablement awarded if sensitisation persists.¹⁹

DISCUSSION

Allergic sensitisation to moulds is not a new condition, although work-related cases are not widely reported in South Africa, when compared to those reported in the general population. In the current case of probable occupational allergic rhinitis to mould, the patient had highly elevated IgE to *A.alternata* species and moderate IgE to *Aspergillus* species. These findings concur with a study at the Universitas Academic Hospital, which demonstrated a higher prevalence of sensitisation to one or more mould allergens (34%), with *A.alternata* being the most prevalent (28%) followed by *A.fumigatus* (8%).²⁰



Figure 2: Water-damaged walls and ceilings observed during an inspection in some of the work areas of the patient: (a) tearoom, (b) patient's office, and (c) laboratory.

Aspergillus, *Alternaria* and *Cladosporium* species are three highly allergenic moulds^{10,21-22} and were all identified in different areas of the patient's work environment.

These results suggest that *Alternaria* and *Aspergillus* species were primarily responsible for the patient's allergic rhinitis as these moulds were detected in the environment (paint scrapings) and in her serum samples. *Cladosporium* identified in air samples was also present in the previous paint scrapings. Air sampling is the preferred measure of exposure to surface sampling and bears relevance to mode of transmission since inhalation of airborne mould spores leads to fungal hypersensitivity. It is plausible that *Penicillium* and *Cladosporium* species detected in the air samples could have contributed to the patient's sensitisation as these species are known to cross-react with *Aspergillus* sp., a complex phenomenon complicating the diagnosis of fungal sensitisation. There is widespread consensus that most mould sensitised patients are monosensitised (>75%), with *Alternaria* being the most common due to the major allergen Alt a1. It is very rare (<1%) in other species (*Aspergillus*, *Cladosporium*, *Penicillium* and *Saccharomyces*) due to high cross-reactivity of these species. The study by Mari et al. 2003 showed a higher prevalence of reactivity to two or more fungal allergens with *Aspergillus* and *Cladosporium* being the most common (>80%) followed by *Penicillium* (~50%) illustrating the high potential of cross reactivity among these fungal species.²³

It can be deduced from the current case report that the patient also demonstrated polysensitisation suggesting possible cross-reactivity as has been reported in the literature. However, this was not specifically investigated by ELISA inhibition experiments.

Allergic Fungal Sinusitis (AFS) is the most common form of non-invasive fungal sinusitis with approximately 5-10% of all chronic rhinosinusitis cases undergoing surgery.^{12,18} Atopic hosts exposed to aerosolised moulds develop an allergic reaction (Type 1, IgE-mediated) from spores which infiltrates the sinuses, resulting in AFS.¹² The diagnostic

criteria for AFS vary in the literature as there is currently no universally accepted criterion, however, Bent and Kuhn is regarded standard criteria and includes:

1. Type 1 hypersensitivity to moulds and elevated total serum IgE;
2. Formation of nasal polyps;
3. Characteristic computed tomography (CT) scan findings;
4. Eosinophilic mucin within the nasal cavity and sinuses without invasion; and
5. A positive fungal stain of sinus content removed during surgery and the exclusion of fungal rhinosinusitis.^{12,17,18}

Although allergic sensitisation is the fundamental requirement for diagnoses of AFS and assessment of atopy by *in vitro* measurements of specific IgE were done for this case,^{18,21} the Bent and Kuhn criteria was not met for this case, thus the previous AFS diagnosis was not supported. Occupational Rhinitis (OR) has been getting little attention, although there is increasing acknowledgement that the burden of this condition is underestimated.⁶ This case will bring awareness to employers, employees and clinicians on probable OR from potential mould exposure, which is commonly not reported, especially in SA.

After a six year period of disabling symptoms, the patient's health improved following the repair of the leaking hospital roof. This highlights the importance of undertaking an immediate response to repairing water-damaged building problems, thereby alleviating ill-health complaints. The patient's follow-up visit to the otolaryngologist in August 2014 showed no growth or fungal infection from her sinuses. The patient also reported a relief of upper airway symptoms and an improved quality of life. These findings are consistent with the recent meta-analysis, which showed that dampness and visible mould presence are important determinants of allergic rhinitis and emphasise that prevention and remediation of indoor dampness and mould problems are likely to reduce allergic rhinitis.²⁴ However, although moisture problems are common, not all are associated with increased health risk. Should there be no visible growth but health complaints are received

from workers, then full investigations must be conducted by a multidisciplinary team to define the extent of the water damage, the exposure assessed and clinical outcomes ascertained. Minimising exposure to the sensitisers are important to prevent persisting symptoms and the risk of poor work ability outcomes.²⁵ Effective preventive measures can be achieved through several remediation steps. These may include cleaning the visible mould growth by experienced specialists, repairing water leaks and damage, applying an antifungal paint to walls and ceilings, improving the ventilation, preferably fresh air supply with extraction in occupied areas to reduce airborne mould spores and lastly, maintaining indoor relative humidity to below 60% to minimise mould growth.

CONCLUSION

This case report has demonstrated that water-damaged buildings can be a source of exposure to mould spores which can cause allergic rhinitis in exposed workers.

Therefore, buildings should be properly maintained and remediated if there is any visible or musty odour from mould growth and/or water damage to prevent excessive exposure of workers. Clinicians should also recognise the potential of workplace exposures to cause allergic rhinitis and have a high index of suspicion, which will more likely lead to appropriate diagnosis and effective management. There is a need for improved awareness of employers, workers and clinicians on occupational allergic disorders specifically caused by moulds, to develop effective preventive strategies.

DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest.

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