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Case Report

Occupants in Moisture-Damaged Buildings May be at Risk for Various Symptoms and Inflammatory Reactions: A Case Series Report and Literature Review

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Abstract

Objective: Moisture damage in buildings is a common problem. Prolonged exposure to dampness microbiota is a health hazard. There is convincing evidence of increased risk of new asthma or asthma exacerbation due to exposure to dampness microbiota and to decaying products in building materials. In addition, irritation symptoms such as rhinitis, cough, eye problems, eczema, headache, and fatigue have been reported. Certain other diseases might also be associated with exposure to dampness microbiota.

Design: Here, we describe four cases of individuals working in water-damaged buildings who in addition to respiratory diseases, suffered from tendinitis, synovitis, arthralgias, muscular pain, cardiac arrhythmias, and urticaria.

Conclusion: We review the contemporary literature and present arguments that prolonged or cumulative exposure to dampness microbiota may cause far more complicated disease than asthma alone.

Keywords: Dampness and mold hypersensitivity syndrome; Water-damaged buildings; Occupational disease; Mold; Asthma; Inflammation; Tendinitis

1. Background

Moisture damage is a common problem [1]. Microbes begin to grow rapidly in a building in which there is condensation, water damage, or both [2]. Microbes can remain viable over a wide range of temperatures; the temperatures of buildings and structures do not limit microbial growth if other growth requirements are met. An adequate air flow can limit microbial growth. Many indoor moisture damage microbial species are known to produce different toxins [2].

Based on international research data, it is known that living or working in a building with moisture damage can cause asthma, asthma exacerbation, allergic alveolitis, and increased susceptibility to infection. Other symptoms have also been described, e.g., eye, ear, and skin irritation [2-16]. According to a recent review by the California Public Health Organization [17], there is a sufficiently strong relationship between moisture and mold damage and the risk for respiratory problems and asthmatic attacks to prove causality. There are also indications of a link between moisture damage or microbial exposure to neurological symptoms [12, 18], chronic fatigue syndrome (CFS), and autoimmune diseases [12, 19, 20].

2. Patients and Methods

In this study, four patients' medical records were analyzed. Patients were asked to provide consent to participate in this study. As this was a non-interventional study, approval by an ethics committee was not required.

2.1 Case 1

A 42-year-old non-smoking, male police officer and active sportsman had been in good health until the year 2015. He had psoriasis. He had been working in a moist building since 2001. His uncle suffered from rheumatoid arthritis. There was no family history of asthma.

He suffered from respiratory tract symptoms while at work. From June 2015, he started to experience arrhythmias. One month later, July 2015, he suffered pain in his muscles, shoulders, and palms and also had numbness in his fingers. He felt warmth and pain in his skin and muscles. His body temperature rose when he was exercising. At that time, he also suffered from urticaria, which was suspected to be dermatomyositis. He later developed muscle weakness, which required a sick leave. He was unable to cope with the demands of his profession.

He was prescribed an antihistamine drug with a four-time daily dose to treat his urticaria. In 2015, he was examined by electroneuromyography (ENMG), with normal findings. The level of creatine kinase was not elevated when he was exercising. Further examinations were performed by a neurologist and in 2016 he was subjected to serum myositis examination, muscle and skin biopsy, and MYOcap-genetic assessment with no diagnostic findings. Biopsy of an urticaria lesion revealed lymphocytes, excessive collagen fiber growth, and granuloma annulare. TNF receptor-associated periodic syndromes (TRAPS) was suspected and a genetic specialist was consulted. No hereditary disease was found. He was examined with a DNA panel for periodic fever syndrome, which showed no

deviation in the 26 genes examined. Plasma levels of C1 esterase inhibitor and its biochemical activity (C1inhBk), immunoglobulins (IgD, IgG, IgM), and complement 3 (C3) were all normal. Myocyte examination (PMDM), platelet additive solution (PAS), nicotinamide adenine dinucleotide (NADH), cyclooxygenase (COX), myokine, fetal and neonatal myokines were normal. A skin biopsy revealed hyperkeratosis.

The levels of IgE-antibodies to home dust and a food panel and gliadin, latex-RAST, complement concentration and antinuclear antibody (ANA), were all within reference values. Omalizumab was considered as a treatment for his refractory urticaria. After a long sick leave, the symptoms gradually declined and medication was no longer needed. In September 2016, after several months of sick leave, he returned to his workplace and developed pneumonia after 3 weeks. He was then transferred to a different task and no longer worked inside the building. In November 2016, he worked within the building for only one day, but on that same day, he developed a serious cough and a new case of pneumonia. Subsequently, he minimized the time he spent in the building. Asthma was diagnosed in May 2017 and he was prescribed asthma medication until spring 2018. In spring 2018, he experienced another pneumonia-like infection, which recurred in October 2018.

In 2019, he has been working in another building and his neurological symptoms and muscle weakness have not returned. He still has asthma and urticaria when he comes into contact with material from the contaminated building where he worked or when he is newly exposed to dampness microbiota.

2.2 Case 2

A 51- year-old policewoman was still active in sports as an adult. She did not suffer from recurrent infections in childhood. She required medication for high blood pressure and hypothyroidism. She had never smoked. Her mother suffered from severe asthma and psoriasis. She had worked since 1988 in a moisture-damaged building. In 2017 building inspections reported microbial findings with *Streptomyces* and *Aspergillus versicolor* in the air conditioning pipes and the outer walls of the building.

From 1993, she had suffered from respiratory symptoms and had several episodes of bronchitis. She was examined by a lung specialist. Values of spirometry and diffusion capacity were normal. She did not have eosinophilia, nor was there any elevation in total IgE. Levels of alpha-1-antitrypsin were elevated, 2.1 g/l, (0.96 - 1.78 g/l reference value). The concentration of alpha-1-antitrypsin was examined later in 2018 and had declined to 1.01 g/l. She also suffered from joint pain and inflammation and experienced pneumonia when working in the building.

From 1995 to 1996, she was on maternity leave from work and suffered no infections or respiratory symptoms. In October 1996, she returned to her former workplace and she became feverish and had recurrent respiratory infections and cough. From 1996 to 1997, she required seven antibiotic treatments for bronchitis. In 1997, she underwent a tonsillectomy due to chronic tonsillitis. A 32% decrease in Forced expiratory volume, 1 min (FEV1) was observed in the histamine provocation test. She had normal bronchoscopy and BAL findings. She exhibited IgG antibodies to *Aspergillus fumigatus* and *Sporobolomyces salmonicolo* i.e. species that had been recovered from the

building where she worked. A specific inhalation challenge with *Aspergillus fumigates* antigen was conducted in the Finnish Occupational Institute of Health (FIOH), but no deterioration was detected in the peak expiratory flow (PEF) test. No medication was given at this time. Asthma was diagnosed in 1999 and therapy was initiated.

From 2000 to 2008, she was absent from work due to maternity leave and personal reasons. She had regular asthma medication and did not experience any infections or asthma symptoms and her physical condition was good. In 2009, she started working in another moisture damaged building and there was a recurrence of her respiratory tract symptoms. In 2012, she suffered from myocarditis and was examined by thoracic computerized tomography (CT). She was diagnosed with an atrial septum defect (ASD) and a leaky bicuspid valve. She experienced arrhythmias and a pulmonary embolus. She had no genetic aberration which would make her susceptible to embolic diseases. She also underwent an operation to treat a rare malformation of the veins in her lower limb.

In 2016, she was examined by a lung specialist due to severe asthma. She had four to five oral corticosteroid treatments on an annual basis. Her PEF values were constantly 400 to 500 l/min while working and 600 l/min when away from the working environment. When absent from work, she did not cough, but when she was in her workplace, she was constantly coughing. She also gained weight (current BMI 35 kg/m^2, previous BMI was 22.4 kg/m^2 in 1997). CT of chest revealed atelectasis and 6-mm nodulus inactive lymphatic node from 2012. Bronchoscopy revealed mucus plugs; bronchomalacia was absent and cytology was normal. No reflux was detected in gastroscopy or in the measurement of pH-impedance.

She underwent a wrist operation because of synovitis in 2012. In 2015, she had severe joint problems, especially in her wrists, hips, and knees. Treatment with oxychlorine was initiated, without response. She was examined by a rheumatologist in 2013 and 2016. Her level of rheumatic factor (RF) was very high (191-524 and 179 IU/ml in 2015, and in November 2018, respectively (reference value <14). She also had permanently elevated fibrin D-dimers (FIDD). Human leucocyte antigen 27 (HLA B27) was negative; no anti-nuclear and extractable nuclear antigen antibodies, antibodies (ENA, ANA) were detected. She will have a consultation with a rheumatologist again in spring 2019.

She has also been examined by infectious disease and genetic specialists. She had low IgG2 levels and signs of a deficient innate immune system in the mannose binding lectin pathway, but a good response to vaccination. She was absent from work for 7 months due to personal reasons in 2017 and during that time, she experienced no asthma symptoms. After returning to work in another building, asthma symptoms returned. Mold-contaminated materials, which had been transferred from her first workplace, seemed to be the trigger for her symptoms.

2.3 Case 3

This patient is a 56-year-old female social worker with obstructive sleep apnea. She is a former smoker. The patient's work-space was housed in an old three-story building in which melting water would drip inside the building in the spring and the ground floor structures had suffered significant microbial damage.

In 2010, the patient experienced an exacerbation of chronic dry cough. Voice hoarseness also developed. These symptoms were evident when she was in the workplace, but they were relieved during her free time. Mechanical air conditioning was installed in the workplace prior to the onset of symptoms. According to the municipal building engineering experts, the replacement air came from the ground floor of the building. Consequently, most workers in her small working community experienced respiratory symptoms. The space between the basement and the workspaces was later separated and some of the symptoms were relieved.

The patient's cough was evaluated in occupational health care and by a lung disease specialist. The cough was later examined in the FIOH. The patient was diagnosed with asthma on the basis of PEF measurements, spirometry, pulmonary status, and disease history. The patient was not able to work in her former office due to symptoms despite workstation repair and asthma treatment. Workplace PEF measurements were performed in a new workstation in a different building. PEF values averaged 377 l /min on working days and 390 l /min on holidays. The daily variation on working days was on average 17%, whereas on holidays, it was only slightly lower - 14%. On the basis of these data, the patient was not diagnosed with an occupational disease.

Currently, the patient is able to work in her new work-space. Occasionally, the employee has to make a short visit to her former work-space and soon after, she tends to suffer a recurrence of her respiratory symptoms. She has also experienced symptoms when staying for even a short time in any building infested with dampness microbiota.

2.4 Case 4

This patient is 50-year-old female teacher who has suffered with asthma since childhood. The patient's respiratory symptoms began when she moved to a new job at the end of 2007. The symptoms were reduced at the beginning of weekends and worsened on working days. Later, the symptoms were reduced only during the long summer holidays as well as when she was on prolonged sick leave or study leave. The patient used asthma medication properly. It is noteworthy that the patient had worked in the same school building prior to beginning a more permanent employment relationship and during that time, she had experienced asthma attacks even though she had been asymptomatic for years.

The patient was examined thoroughly for co-morbidities. In the early stages of the symptoms, the patient was found to have thickened Achilles tendons, inflammation of the plantar fascia, and fluid in the peroneal tendon. Running was considered as an etiological factor even though the patient has not been running for about 10 years because of her symptoms. Despite abstaining from running, the co-morbidities worsened. Imaging studies revealed inflammation of the right biceps tendon. A local subpleural mass of 1.7 × 1 × 1.3 cm was observed in the lower end of the right segment of the right lung.

Rheumatological investigations ruled out rheumatoid arthritis and fibromyalgia. The disease was defined as polyarthralgia, insertion, and tenosynovitis of unknown etiology. The patient was treated with oxychlorine and prednisolone. Oxychlorine was not effective and prednisolone was poorly tolerated. A private specialist concluded

that her symptoms were associated with the exposure to dampness microbiota, and that symptoms could be most significantly relieved when the patient avoided exposure to damaged buildings. The patient is currently on study leave; the symptoms of irritation have resolved and her joint symptoms are greatly reduced. The walls of the patient's workplace were infested with dampness microbiota. The building is currently being renovated.

3. Discussion

Our patients from water-damaged occupational environments presented with new asthma or an exacerbation of asthma symptoms, which is consistent with previous reports describing the risks associated with exposure to moisture damage [7, 13]. It has been claimed that symptoms are usually aggravated in asthmatics sensitized to moisture-damage microbes when the outdoor air humidity is high, such as in late summer and autumn [7]. Symptoms are exacerbated by rainy periods and on windy days after rain [7, 21]. Asthma and chronic rhinitis associated with exposure to dampness microbiota tend to be persistent and difficult to treat [22]. In addition to upper airway respiratory symptoms, the patients experienced other symptoms, such as recurrent pneumonia, tendinitis, synovitis, arthralgias, muscular pain, arrhythmia, and urticaria. Findings in imaging in the lungs or tendons were not specific.

Recent research and our clinical experience suggest that the adverse health effects caused by moisture-damage microbes may be broader than simply an increased risk for asthma. Exposure to dampness microbiota typically results in mild irritation symptoms that are reversible. Irritation symptoms include dry cough, dry eyes, and nasal congestion. Avoiding exposure at an early stage resolves the symptoms. For example, if exposure occurs at work, the symptoms will be relieved on weekends. As exposure continues, the symptoms worsen and a short period of time, such as the weekend, is too short to allow recovery [23]. Multiple chemical sensitivity, musculoskeletal problems, and neurological symptoms are typical for employees with prolonged exposure to DM (Hyvönen et al., unpublished data). This disease is called Dampness and Mold Hypersensitivity Syndrome (DMHS) [23]. The symptoms of DMHS may be highly variable, even unexpected. There is no single test to diagnose DMHS. Accordingly, it is important for physicians to listen to the patient's narrative.

The most important cure for DMHS patients is avoidance of exposure to moisture damage microbiota [2, 23]. Each illness and symptom should nevertheless be treated according to good medical practice; these should include a nutritional intervention and a consultation about physical exercise. Various methods of detoxification, such as perspiration e.g., low temperature sauna, may be helpful [24]. Repairing a damaged workplace building can reduce the risk of the development of illness in new workers, but often does not improve the condition of those who have been already affected [22]. It is noteworthy, that the materials might become contaminated in a moldy environment and should they be transferred to a new, clean workplace, they may trigger symptoms. While knowledge on the pathophysiology associated with the moisture-damage microbiota is still far from being explicit, there is already a wealth of data on the effects of dampness microbiota at the cellular level. Mycotoxins, endotoxins, exoenzymes, nanoparticles, and volatile organic compounds play key roles in pathogenesis of DMHS. Exposure can cause

inflammatory reactions through allergic immunological pathways, toxicological mechanisms, and secondary infections [2, 3]. Experiments conducted in cell culture and animal models have shown that mycotoxins and bacterial toxins are cytotoxic and possess immune-modifying properties. The adverse health effects that they evoke depend on the level of the toxin exposure and on their composition, and also on the preceding health status of the occupant [2].

For example, it is known that components of dampness microbiota activate multiple pattern recognition receptors (PRR) on the surface of epithelial, macrophage, and dendritic cells in the respiratory tract. The most important PRRs are Toll-like receptors 2, 4, and 9 [3, 25]. Activation of these receptors initiates the inflammatory process, where the cells produce cytokines (including tumor-necrosis factor alpha, interleukin-1 beta, chemokines and adhesion molecules) [14]. The continual overproduction of these molecules damages the surrounding tissues and maintains a state of chronic inflammation.

Significant changes in B-cell profiles [26] have also been observed in exposed and symptomatic patients. Mononuclear cells in patients with upper respiratory disease are known to produce interleukins, growth factors, cytokines and chemokines, such as IL-17, IL-10, TGFβ, and MIP-1β [27]. β-glucans exacerbate allergic asthma and promote a Th2/Th17 immunological response, leading to an inadequate response to steroid treatment [28]. Mycotoxins can also modulate an immunologic response such as reducing macrophage activity and increasing an individual's susceptibility to respiratory tract infections [3, 29]. The increased susceptibility to infections may be explained as follows: many toxins are cytotoxic and may inhibit opsonization. Xenobiotics may not fully coated by immunoglobulins that may lead to impaired phagocytosis. Dendritic cells may not be able to present antigens to T cells effectively. For example, *Aspergillus fumigatus* inhibits the function of dendritic cells [25].

Chronic inflammation is thought to play an important role in DMHS in the development of other symptoms in addition to respiratory tract problems [2, 3]. It is known that chronic systemic inflammation can damage the blood-brain barrier which might increase the access of immune cells to the brain thereby leading to autoimmune processes [2, 3]. Microbial toxins evoke oxidative stress. Some toxins are vasoactive, some have cardiovascular effects, and many affect the immunological and (neuro) endocrinological systems [2, 3]. Earlier studies have demonstrated that individuals who have been exposed to dampness microbiota may suffer joint and tendon symptoms [30, 31].

A psychological burden has been proposed as a trigger of DMHS. The symptoms of the patients examined here were exacerbated when they were working in a damaged building and improved when they were in a clean environment and furthermore, the symptoms returned when they re-entered the damaged building. This is referred to as the so-called Sick Building Syndrome. In these patients, there were tangible features of a physical disease demonstrated by imaging, which argues against the psychological nature of DMHS. Cell biology studies have confirmed the findings observed by clinicians [2, 3, 32]. Further research is needed to clarify the pathophysiology of DMHS.

4. Conclusions and Implications

- Exposure to dampness and mold is a risk factor for health.
- The symptoms may be pulmonary and extrapulmonary.
- Chronic inflammation plays an important role causing the symptoms.

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Disclosure Statement

The authors report no conflict of interest.

Notes of Contributors

Saija Hyvönen is a specialist in occupational medicine and a PhD student working in Pihlajalinna. Tamara Tuuminen is a specialist of clinical microbiology and adjunct professor working in Medical Center Kruunuhaka. Jouni Lohi is a specialist in general practice, adjunct professor and resident in pathology working in Lapland Central Hospital.

References

1. Becher R, Høie AH, Bakke JV, et al. Dampness and moisture problems in Norwegian homes. Int. J. Environ. Res. Public Health 14 (2017): 1241.
2. Tuuminen T, Lohi J. Immunological and toxicological effects of bad indoor air to cause dampness and Mold Hypersensitivity Syndrome. AIMS Allergy Immunol 2 (2018): 190-204.
3. Global Indoor Health Network (GIHN). Diagnosis and Treatment of Illness Caused by Contaminants in Water-Damaged Buildings. Working Together for Healthy Indoor Environments. PO Box 777308 Henderson, NV 89077-7308. Luettavissa: https://www.globalindoorhealthnetwork.com.
4. Kuhn DM, Ghannoum MA. Indoor mold, toxigenic fungi, and stachybotrys chartum: Infectious disease perspective. Clin Microbiol rev 16 (2003): 144-172.
5. Fisk WJ, Lei-Gomez Q, Mendell MJU. Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. Indoor Air 17 (2007): 284-296.
6. Fisk WJ, Elisevaara A, Mendel MJ. Association of residential dampness and mold with respiratory tract infections and bronchitis: a meta-analysis. Environ Health 15 (2010): 1-11.
7. Hurraβ J, Heinzow B, Aurbach U, et al. Medical diagnostics for indoor mold exposure. Int Hyg Envir Health 220 (2017): 305-328.
8. Lanthier-Veilleux M, Baron G, Genereux M. Respiratory diseases in university students associated with exposure to residential dampness or mold. Environ res publ health 13 (2016): 1154.

9. Mendell MJ, Mirer AG, Cheung K, et al. Respiratory and allergic health effects of dampness, mold, and dampness-related agents: A review of the epidemiologic evidence. Environ Health Persp 119 (2011): 748-756.
10. Olaniyan T, Dalvie MA, Röösli M, et al. Asthma-related outcomes associated with indoor air pollutants among schoolchildren from four informal settlements in two municipalities in the Western Cape Province of South Africa. Indoor Air 29 (2019): 89-100.
11. Park JH, Cox-Ganser JM. Mold exposure and respiratory health in damp indoor environments. Front. Biosci. (Elite Ed) 3 (2011): 757-771.
12. Tuuminen T, Lohi J. Revising the criteria for occupational mold-related disease: Arguments, misconceptions and facts. EMJ Allergy Immunol 1 (2018): 128-135.
13. WHO guidelines for indoor air quality: dampness and mould. World Health Organization. Copenhagen, DenmarkI. SBN 978 92 890 4168 3 (2009).
14. Wong J, Magun BE, Wood LJ. Lung inflammation caused by inhaled toxicants: a review. Int J Chron Obsrtuct Pulmon Dis 11 (2016): 1391-1401.
15. Zhang X, Norbäck D, Fan Q, et al. Dampness and mold in homes across China: Associations with rhinitis, ocular, throat and dermal symptoms, headache and fatigue among adults. Indoor Air 29 (2019): 30-42.
16. Zhug Y, Qian H, Zheng X, et al. Residential risk factors for childhood pneumonia: A cross-sectional study in eight cities of China. Environ Int 116 (2018): 83-91.
17. Kanchongkittiphon W, Mendell MJ, Gaffin JM, et al. Indoor environmental exposures and exacerbation of asthma: An update to the 2000 review by the institute of medicine. Environ Health persp 123 (2015): 6-20.
18. Empting LD. Neurologic and neuropsychiatric syndrome features of mold and mycotoxin exposure. Toxicol Int Health 25 (2009): 577-581.
19. Tuuminen T, Jääskeläinen T, Vaali K, et al. Dampness and mold hypersensitivity syndrome and vaccination as risk factors for chronic fatigue syndrome. Autoimmun Rev 18 (2018): 107-108.
20. Tuuminen T, Rinne KS. Severe Sequelae to Mold-Related Illness as Demonstrated in Two Finnish Cohorts. Front Immunol 8 (2017): 382.
21. Aggarwald AN, Chakrabarti A. Does climate mould influence of mold on asthma? Lung India 30 (2013): 273-276.
22. Park J-H, Cho SJ, White SK, et al. Changes in respiratory and nonrespiratory symptoms in occupants of a large office building over a period of moisture damage remediation attempts. PLoS One 13 (2018): e0191165.
23. Valtonen V. Clinical diagnosis of the dampness and mold hypersensitivity syndrome: Review of the literature and suggested diagnostic criteria. Front immunol 8 (2017): 951.
24. Genuis SJ, Sears ME, Schwalfenberg G, et al. Clinical Detoxification: Elimination of Persistent Toxicants from the Human Body. Editorial (2013): 1-3.
25. van de Veerdonk FL, Gresnigt MS, Romani L. Aspergillus fumigatus morphology and dynamic host interactions. Nat Rev Microbiol 15 (2017): 661-674.

26. Gray MR, Thracher JT, Grago R. Mixed mold mycotoxicosis: Immunological changes in humans following exposure in water-damaged buildings. Arch Environ Health 58 (2003): 410-420.
27. Rosenblum Lichtenstein JH, Hsu Y-I, Gavin IM. Environmental mold and mycotoxin exposures elicit specific cytokine and chemocine responses. PLoS One 10 (2015): e0126926.
28. Zhang Z, Biagini Myers JM, Brandt EB. β-Glucan exacerbates allergic asthma independent of fungal sensitization and promotes steroid-resistant TH2/TH17 responses. J Allergy Clin Immunol 139 (2017): 54-65.
29. Ammann HM. Inhalation Exposure and Toxic Effects of Mycotoxins. Biol Microfungi (2016): 495-523.
30. Luosujärvi RA, Husman TM, Seuri M, et al. Joint symptoms and diseases associated with moisture damage in a health center. Clin Rheumatol 22 (2003): 381-385.
31. Myllykangas-Luosujärvi R, Seuri M, Husman T, et al. A cluster of inflammatory rheumatic diseases in a moisture-damaged office. Clin Exp Rheumatol 20 (2002): 833-836.
32. Tuuminen, Lohi. Dampness and Mold Hypersensitivity Syndrome is a Biotoxicosis that should be Diagnosed Promptly. Adv in Clin Toxicol 4 (2019): 000144.

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