A Case of Occupational Asthma Induced by Cleaning Agent

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Background: Cleaning agents have an airway irritant effect. There have been few reports of occupational asthma induced by cleaning agents at workplace or at home. Chlorine, the most common chemical used in cleaning products, has a strong irritative effect on the airways. We experienced a case of occupational asthma caused by chlorine that developed in a health care worker.

Case History: Eleven years later, she developed dyspnea and cough, which was aggravated after exposure to cleaning agent spray that released chlorine at workplace.

Results: The patient was positive to 2 house dust mites on skin prick tests but negative to the cleaning agent. Methacholine bronchoprovocation tests showed a negative result; however, a specific bronchoprovocation test with exposure to the cleaning agent showed a positive response of more than 15% fall of forced expiratory volume in 1 second. There were no significant changes in sputum cell counts and exhaled nitric oxide levels after the specific bronchoprovocation test.

Conclusion: To the best of our knowledge, this is the first case of occupational asthma caused by chlorine, a cleaning agent that developed in a health care worker in Korea, which was confirmed by the specific bronchoprovocation test.

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Key words: Chlorine; Cleaning; Occupational asthma

INTRODUCTION

More than 10% of all cases of adult-onset asthma occurred from exposure to substances in the workplaces. Cleaning agents have been used on a daily basis in nearly all workplaces and homes, and have an irritating effect on mucous membranes and skin, and a sensitizing potential. There have been reports that cleaning agents are associated with occupational allergic diseases, such as asthma, asthma-like symptoms and hand dermatitis. In addition, cleaning and nursing are known as the occupations with the highest risk of developing new-onset asthma. To the best of our knowledge, this is the first case of occupational asthma (OA) caused by a cleaning agent developed in a nurse working at a hospital in Korea.

CASE REPORT

A 33-year-old female presented with dyspnea and cough for 3 months, which was aggravated after exposure to cleaning agent at a hospital. She had allergic rhinitis for 10 years with mild and persistent symptoms. She had worked as a nurse at medical ward of a tertiary hospital for 11 years. During the last 6 months, she was intensively exposed to the cleaning agent during bed making process due to preparation of hospital certification assessment, and then her asthmatic symptoms developed. She had no symptoms before work, however, symptoms developed immediately after cleaning agent spraying at the workplace. The single component of cleaning agent was sodium hypochlorite, a chlorine releasing agent, which was diluted with water and used for a bleach or disinfectant. The cleaning agent was used in a spray form throughout the
hospital, including wards and offices. She had experienced recurrent
dyspnea and cough immediately after this spraying process.

There were no abnormal findings in blood cell count, serum
biochemistry, total immunoglobulin E (IgE) level, eosinophil cationic
protein level or radiologic examinations including chest and
paranasal sinus. Positive result was noted on skin prick test (SPT)
and serum specific IgE to Dermatophagoides farinae, however, negative result was noted on SPT to the cleaning
agent at a concentration from 1:100 dilution in 0.9% NaCl to
undiluted cleaning agent. Baseline pulmonary function parameters
including the forced expiratory volume in 1 second (FEV1) and
forced vital capacity were normal, and methacholine bronchoprov-
ocation test showed a negative result. The cleaning agent used in
the hospital was prepared for bronchoprovocation test. The test was
performed in a challenge chamber using spray press with the cleaning
agent for 3 minutes, after then the patient entered the chamber.
She developed immediate onset of dyspnea and cough and showed
a significant decrease of FEV1 value (18.3% fall from 3.04 to 2.48
L) as shown in Fig. 1. There were no changes in sputum cell count
and exhaled nitric oxide (NO) level after the bronchoprovocation
test. Her asthma symptoms improved after avoiding the work of
bed making.

DISCUSSION

OA was suspected in 5~15% of the asthmatic subjects, and
about 0.2~0.5% of young adults become asthmatics or have their
asthma exacerbated because of their occupation. More than 250
known specific occupational agents have been associated with
asthma, and exposure to cleaning agents has been associated with
respiratory and asthma symptoms both at the workplace and
home. The risk of new-onset asthma was 1.7 times higher in
individuals involving in cleaning or caretaking than those involving
in professional or administrative jobs. In elite swimmers, long-term
and repeated exposure to chlorine compounds increased bronchial
hyperresponsiveness (BHR) and airway inflammation. The nurses
also showed an increased risk on asthma 2 times greater than others
exposed to cleaning products, in which the highest risks were the
use of ammonia, bleach and cleaning products at work. The
nonoccupational use of common household cleaning products in
ones’ own homes were significantly associated with asthma symptoms
or medication and wheeze.

The risk of asthma caused by cleaning agents was higher in cleaning
and caretaking workers compared those with other occupations. An
increased risk for incident asthma was also observed among nurses,
in which ammonia, bleach and/or cleaning products such as chlorine
in spray form at work was associated with asthma. The use
of products in spray form at least once a week significantly increased the
risk of asthma from 30% to 50%, and frequent domestic use of
hypochlorite bleach was associated with the prevalence of lower
respiratory tract symptoms. In the present study, this patient had
been exposed for 11 years. Her asthmatic symptoms developed after
increased exposure for the last 6 months. These findings demonstrated
that the exposure to chlorine in a spray form cleaning agent can induce
OA in exposed health care workers in hospital.

Specific bronchoprovocation test is the gold standard for the
diagnosis of OA. The previous studies suggested that diagnosis of
OA can be accepted if the FEV1 value decreases more than 15%
during the specific bronchoprovocation test. In the present
study, the patient was a nurse, and she had experienced recurrent
asthmatic symptoms developed immediately after the exposure to
sodium hypochlorite containing cleaning agent in a spray form.
Although methacholine bronchoprovocation test showed a negative
result, the specific bronchoprovocation test showed more than 15%
decrease of FEV1 with typical asthmatic symptoms. Based on these
findings, we confirmed her diagnosis of OA caused by cleaning
agent.

Recent studies suggested low-to-moderate exposure to cleaning
agents may induce respiratory irritants, and inflammatory
changes. However, among 13 cleaning employees with work-related
asthma-like symptoms, no clinically significant changes in sputum
cell counts or fractional exhaled nitric oxide (FeNO) were detected
after chlorine inhalation. The active component of household
bleach is sodium hypochlorite, and it can release chlorine in amounts that are equivalent to 3–10% available chlorine. Chlorine, the most common chemical used in cleaning products, has a strong irritant effect on the airways and may increase bronchial BHR. However, only 1 had a significant decrease in the provocative concentration producing a 20% fall in FEV1 methacholine after provocation test with bleach among 13 cleaning employees with or without BHR. No significant pulmonary function effect was noted after chlorine inhalation. There were correlations between increased BHR and the number of sputum eosinophils, as well as the amount of eosinophil markers in some studies, but not in others. In elite swimmers, it has been suggested that a combination of chronic exposure to chlorine and high ventilator rates lead to osmotic degranulation of mast cells and bronchoconstriction. Although many OA patients had BHR, there are some reports of OA without BHR in cases of disocyanate or hydroxyapatite induced OA. In the present study, the patient had a significant fall in FEV1 after the exposure to cleaning agent, but no significant changes were noted in BHR and inflammatory markers such as FeNO and sputum eosinophil counts after the specific bronchoprovocation test. These findings suggest that although the pathogenic mechanism of chlorine-induced asthma was not clarified clearly, cleaning agent can induce OA without development of BHR.

CONCLUSION

We report a case of chlorine-induced OA confirmed by bronchoprovocation test. Further studies are needed to investigate the pathogenic mechanism of cleaning agent-induced OA.

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