

Case Report



Occupational asthma related to indoor air pollution in a worker at an indoor air gun shooting range: a case report

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OPEN ACCESS

Received: Mar 13, 2023

Revised: May 19, 2023

Accepted: May 20, 2023

Published online: Jun 19, 2023

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ABSTRACT

Background: Indoor air pollution can cause and exacerbate asthma. We report a previously undescribed case of occupational asthma related to indoor air pollution in a worker at an indoor air gun shooting range and highlight the potential risk of developing occupational asthma in this environment.

Case presentation: A 31-year-old man presented with dyspnea, cough, and sputum and was diagnosed with asthma complicated by pneumonia. Objective evidence of asthma was obtained by performing a methacholine bronchial provocation test. It was suspected that the patient had occupational asthma, which began one month after changing jobs to work within the indoor air gun shooting range. The highest peak expiratory flow (PEF) diurnal variability on working days was 15%, but the highest variation was 24%, with 4 days out of 4 weeks having a variation of over 20% related to workplace exposure. Conversely, the diurnal variability on the rest days was 7%, and no day showed a variation exceeding 20%. The difference in the average PEF between working and rest days was 52 L/min. PEF deterioration during working days and improvement on rest days were noted.

Conclusions: The results obtained from the in-depth analysis of the PEF were adequate to diagnose the patient with occupational asthma. Exposure to indoor air pollution and lead and the patient's atopy and allergic rhinitis may have contributed to the development of occupational asthma.

Keywords: Indoor air gun shooting range worker; Occupational asthma; Indoor air pollution; Lead

BACKGROUND

Asthma in the workplace comprises both occupational asthma, which is caused by exposure to specific substances in the workplace, and asthma exacerbated by the workplace. Studies have estimated that the population-attributable risk of occupational asthma ranges from 10%–25% of all new-onset asthma cases in adults.¹ It has been reported that approximately 10% of asthma patients experience an exacerbation in the workplace. However, occupational asthma is often underdiagnosed due to a lack of expertise in the field. Several occupational

Abbreviations

BLL: blood lead level; CHG: change; FEV₁: forced expiratory volume in one second; FVC: forced vital capacity; GHDC: Gimhae-si Urban Development Corporation; IgE: immunoglobulin E; IRB: Institutional Review Board; MAST: Multiple allergens simultaneous test; MVV: maximal voluntary ventilation; PC20: provocative concentration resulting in a decrease in FEV₁ by > 20%; PEF: peak expiratory flow; PEFT: peak expiratory flow time; sIgE: serum-specific IgE; TWA: time weighted average; VOC: volatile organic compound.

Competing interests

The authors declare that they have no competing interests.

Author Contributions

Conceptualization: Kim SY. Data curation: Kim SY, Lee S, Lee SE. Formal analysis: Kim SY, Lee S. Funding acquisition: Kim SY. Investigation: Kim SY, Lee SE, Lee S. Methodology: Kim SY. Project administration: Kim SY. Resources: Kim SY, Lee SE. Software: Kim SY, Lee S. Supervision: Kim SY, Kang D, Kim Y. Validation: Kim SY. Visualization: Lee KM, Kim YJ. Writing - original draft: Kim SY, Lee KM. Writing - review & editing: Kim SY, Lee KM, Kim YJ.

groups, such as bakers, laundry workers, shoemakers and repairers, and railway and station personnel, have been identified as being at high risk for occupational asthma.² However, information on occupational asthma among shooting range workers is limited.

Indoor air pollution is recognized as a risk factor for the development and exacerbation of asthma. Although the specific substances that contribute to asthma have not been identified yet, particulate matter, nitrogen dioxide (NO₂), volatile organic compounds (VOCs), and mold are some of the suspected pollutants that can contribute to asthma.³ The elevated levels of air pollutants in indoor shooting ranges are caused by shooting and chemical use during maintenance. Poor ventilation in indoor shooting ranges can cause the accumulation of airborne pollutants in indoor environments.⁴

The United States (US) has an estimated 16,000 to 18,000 shooting ranges.⁵ Shooting range workers are a distinct group consistently exposed to noise and air pollution. They are also exposed to other occupational hazards, such as physical and psychological harm. Shooting range workers are at risk of developing occupational asthma because of their consistent exposure to air, lead, and other pollutants. The prevalence of occupational asthma among workers within a shooting range remains unclear. However, it can have a significant impact on the quality of life of individuals and their ability to continue working in the industry.

This case report presents a previously undescribed instance of occupational asthma in a worker within an indoor air gun shooting range. This will bring attention to the potential risk of indoor air pollution in developing occupational asthma in this environment and inform healthcare professionals, occupational health and safety specialists, and indoor air gun shooting range workers about this risk. This study is expected to aid in establishing suitable prevention and management strategies.

CASE PRESENTATION

Patient information

The patient was a 31-year-old man.

Chief complaints

Dyspnea.

Present illness

He visited a local hospital in November 2020 with symptoms of dyspnea, cough, and sputum, which began a week before the presentation. Following an evaluation at the hospital, he was diagnosed with asthma complicated by pneumonia and was admitted for treatment. In December 2020, he was transferred to the Pulmonary Medicine Center of Pusan National University Yangsan Hospital for additional assessment and management (Fig. 1).

Imaging (December 2, 2020)

The posteroanterior chest radiograph showed no abnormal findings (Fig. 2).

Pulmonary function testing (December 2, 2020)

A spirometry test was conducted, and the results indicated that the forced expiratory volume in one second (FEV₁) was 3.69 L (85% predicted), the forced vital capacity (FVC) was

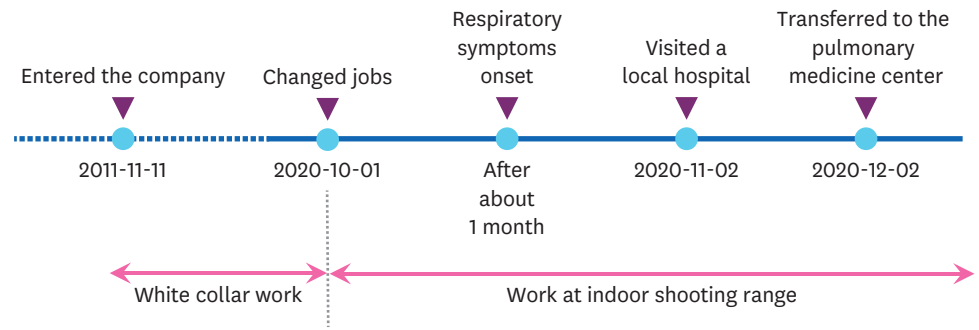


Fig. 1. Flowchart of events.

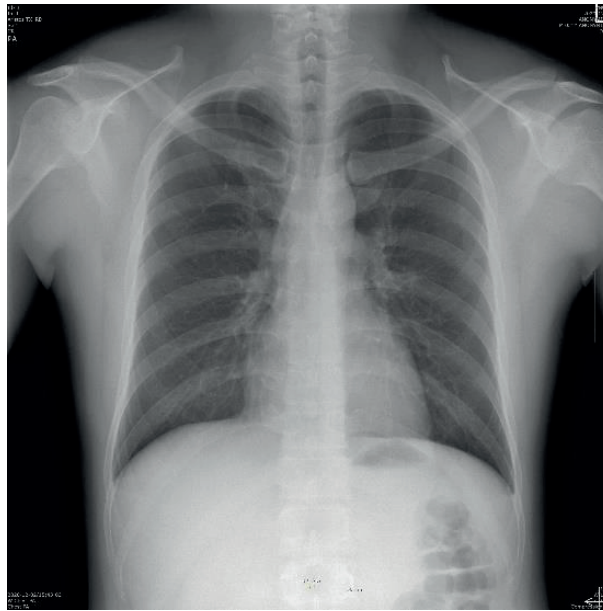


Fig. 2. Posteroanterior chest radiograph obtained on December 2, 2020.

4.36 L (85% predicted), and the FEV_1/FVC ratio was 85% (Fig. 3). According to spirometry results, no airflow limitations were evident. A methacholine bronchial provocation test was performed to confirm the diagnosis of asthma.

The results showed that the provocative concentration resulting in a decrease in FEV_1 by $\geq 20\%$ (PC_{20}) was 2.60 mg/mL (reference range: ≥ 16 mg/mL) and positive reversibility with a short-acting β_2 agonist (Fig. 4). The results of the bronchoprovocation challenge provided objective evidence of asthma. The fraction of exhaled nitric oxide was estimated as an advanced diagnostic test, and a level of 139 ppb was obtained (reference range: low (< 25 ppb), intermediate (25 ppb to ≤ 50 ppb), high (> 50 ppb)). A level of > 50 ppb is indicative of eosinophilic airway inflammation and provides further evidence supporting airway inflammation as a characteristic of asthma.

Immunologic testing (December 2, 2020)

A quantitative test for total immunoglobulin E (IgE) revealed levels exceeding the upper limit of 2,000 IU/mL (normal range: ≤ 87 IU/mL). Multiple allergens simultaneous test (MAST) for 92 allergens was also performed for serum-specific IgE (ssIgE), and the results were

Occupational asthma related to indoor air pollution

Spirometry	(BTPS)	PRED	PRE-RX		POST-RX		% CHG
			BEST	%PRED	BEST	%PRED	
FVC	Liters	5.16	4.36	85			
FEV1	Liters	4.36	3.69	85			
FEV1/FVC	%	86	85				
FEF25-75%	L/sec	4.36	3.89	89			
FET100%	Sec		6.81				
PEF	L/sec	9.37	12.50	133			
PEFT	Sec		0.06				
FIVC	Liters	5.18	3.89	75			
Vol Extrap	Liters		0				
MVV	L/min	173	150	87			

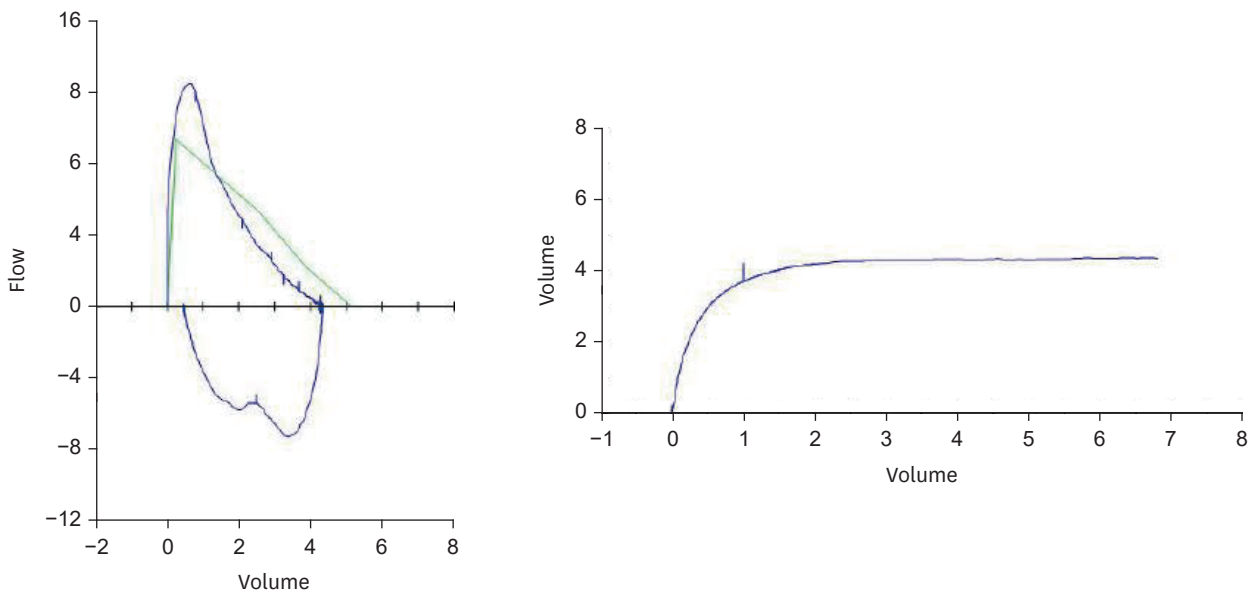


Fig. 3. Findings of the spirometry examination obtained on December 2, 2020.

FVC: forced vital capacity; FEV1: forced expiratory volume in one second; FEF25-75%: forced expiratory flow at 25 and 75% of the pulmonary volume; FET100%: forced expiratory time; PEF: peak expiratory flow; PEFT: peak expiratory flow time; FIVC: forced inspiratory vital capacity; MVV: maximal voluntary ventilation; CHG: change.

qualitatively categorized into seven classes (class 0: 0.00 to 0.34 IU/mL, class 1+: 0.35 to 0.69 IU/mL, class 2+: 0.70 to 3.49 IU/mL, class 3+: 3.50 to 17.49 IU/mL, class 4+: 17.50 to 49.99 IU/mL, class 5+: 50.00 to 99.99 IU/mL, and class 6+: ≥ 100 IU/mL). The allergens that showed a class of +2 or higher on the MAST included *Dermatophagoides pteronyssinus*, *D. farinae*, cat, dog, shrimp, apple, peach, house dust, *Aspergillus fumigatus*, *Alternaria alternata*, birch, pork, peef, pupa, and silk cocoons (Table 1).

Social history, family history, and medical history

The patient had owned three indoor cats since 2017. He experienced chronic allergic rhinitis, which tended to worsen during spring. No other notable medical history was noted.

Evaluation of the relationship between work and illness

The pulmonary medicine center suspected that the patient had occupational asthma due to the onset of asthma symptoms one month after changing jobs and referred him to the Department of Work Environment Medicine to assess the relationship between work and illness. He was prescribed two doses of Symbicort Rapihaler (budesonide/formoterol

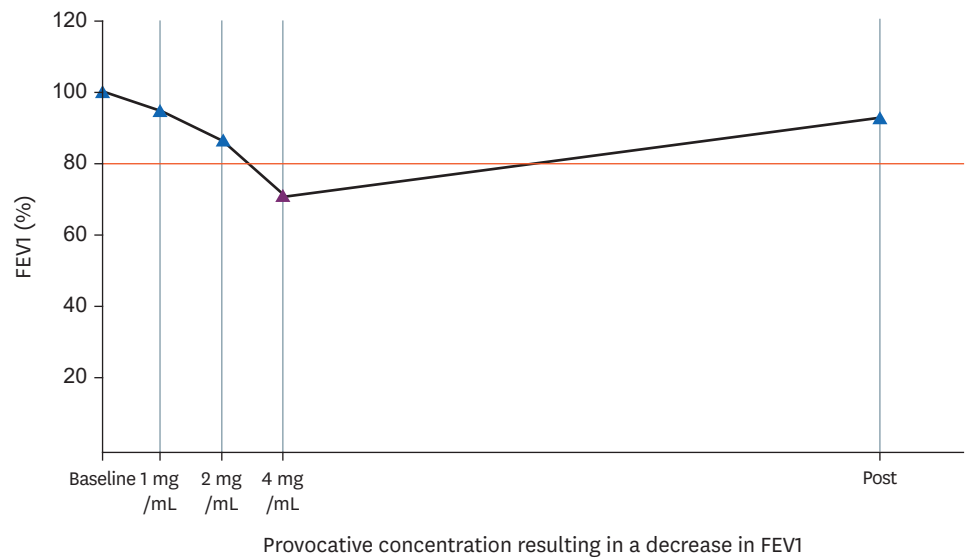


Fig. 4. Findings of the Methacholine bronchial provocation test performed on December 2, 2020. FEV1: forced expiratory volume in one second.

fumarate hydrate) at 160/4.5 μg daily, and one capsule of Montelukast sodium/levocetirizine hydrochloride) at 10/5 mg as needed. The patient started working in a clerical position at a sewage treatment plant operated by the Gimhae-si Urban Development Corporation (GHDC) in South Korea in 2011. In October 2020, he transitioned to a facility manager role at the Gimhae-si Shooting Range, also managed by the GHDC. The shooting range is an indoor air gun facility with 60 shooting stations, and the patient's job duties included testing air guns for inspection, assisting citizens in experiencing the shooting range, performing administrative work, and collecting lead pellets and targets for 3 hours once a week. The monthly consumption of lead pellets was 30 kg. He wore a dust mask while performing these duties, but his nose turned black after the work. The patient worked five days a week from 9:00 am to 6:00 pm, with a once-every-two-weeks Saturday shift.

The Gimhae-si Shooting Range is a workplace subject to work environment monitoring in accordance with South Korea's Occupational Safety and Health Act. Based on the 2018 q, the hazardous factors to be evaluated were lead and its inorganic compounds, and the time weighted average (TWA) was 0.0063 mg/m^3 , which is lower than South Korea's threshold limit for TWA (TLV-TWA) of 0.05 mg/m^3 .

Indoor air pollution within the shooting range was monitored in accordance with South Korea's Indoor Air Quality Control Act. The data from 2019 showed the following: particulate matter less than 10 microns, 37.0 $\mu\text{g}/\text{m}^3$ (reference range: $\leq 75 \mu\text{g}/\text{m}^3$); carbon dioxide, 553 ppm (reference range: $\leq 1,000$ ppm); formaldehyde, 18.9 $\mu\text{g}/\text{m}^3$ (reference range: $\leq 80 \mu\text{g}/\text{m}^3$); carbon monoxide, 1.1 ppm (reference range: ≤ 10 ppm); NO_2 , 0.013 ppm (reference range: ≤ 0.05 ppm); radon, 50.9 Bq/m^3 (reference range: $\leq 148 \text{Bq}/\text{m}^3$); and VOC, 155.6 $\mu\text{g}/\text{m}^3$ (reference range: $\leq 400 \mu\text{g}/\text{m}^3$). Compared to the reference range set by South Korea's Indoor Air Quality Control Act, the exposure levels of indoor air pollutants in the shooting range were low.

A blood test to assess the level of lead exposure based on the biological exposure index was conducted as lead is a hazardous factor previously measured in working environment monitoring. The blood test results revealed lead levels of 1.8 $\mu\text{g}/\text{dL}$.

Table 1. Multiple allergen tests performed simultaneously on December 02, 2020

Allergen	Class	Allergen	Class
Dermatophagoides pteronyssinus	3+	Reed	0
Dermatophagoides farinae	3+	Redtop, bent grass	0
Storage mite	0	Honey bee	0
Cat	5+	Yellow jacket	0
Dog	3+	Latex	0
Egg white	0	Penicillium notatum	0
Milk	0	Sycamore mix	0
Maize	0	Sallow willow	0
Sesame	0	Poplar mix	0
Soybean	0	Ash Mix	0
Crab	0	Pine	0
Shrimp	2+	Japanese cedar	0
Potato	0	Acacia	0
Apple	2+	Hinoki cypress	0
Cacao	0	Oxeye daisy	0
Peach	3+	Dandelion	0
Mackerel	0	English plantain	0
Carbohydrate cross-reactive determinant	0	Russian thistle	0
Rye pollens	0	Goldenrod	0
House dust	3+	Pigweed	0
Cockroach	0	Pork	4+
Cladosporium herbarum	0	Beef	2+
Aspergillus fumigatus	5+	Cheddar cheese	0
Alternaria alternata	3+	Chicken	0
Alder	0	Pupa, silk cocoon	2+
Birch	4+	Tomato	0
Oak white	0	Kiwi	0
Ragweed,short	0	Mango	0
Mugwort	0	Banana	0
Japanese hop	0	Citrus mix	0
Acarus siro	1+	Peanut	0
Horse	1+	Walnut	0
Guinea pig	3+	Chestnut	0
Sheep	0	Wheat flour	0
Rabbit	4+	Barley	0
Hamster	2+	Rice	0
Hazel	0	Buchk-wheat	0
Sweet vernal grass	0	Garlic	0
Bermudas Grass	0	Onion	0
Orchard grass	0	Celery	0
Timothy grass	0	Cucumber	0

To investigate and evaluate the potential connection between airflow obstruction and work, serial peak expiratory flow (PEF) was monitored four times daily for four weeks (**Fig. 5, Supplementary Table 1**). Diurnal variability was calculated as $\frac{\text{Maximum PEF of a Day} - \text{Minimum PEF of a Day}}{\text{Mean PEF of a Day}}$. The mean PEF diurnal variability was 15% on working days and 8% on non-working days. The value of the PEF variation exceeded 20% on four of the 15 workdays, whereas on days not related to the workplace, the variation did not exceed 20%. The maximum value of the PEF diurnal variation on a working day was 24%.

Ethics statement

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board (IRB) Committee of the Pusan National University Yangsan Hospital (IRB No. 05-2023-033 and date of approval was February 13, 2023).

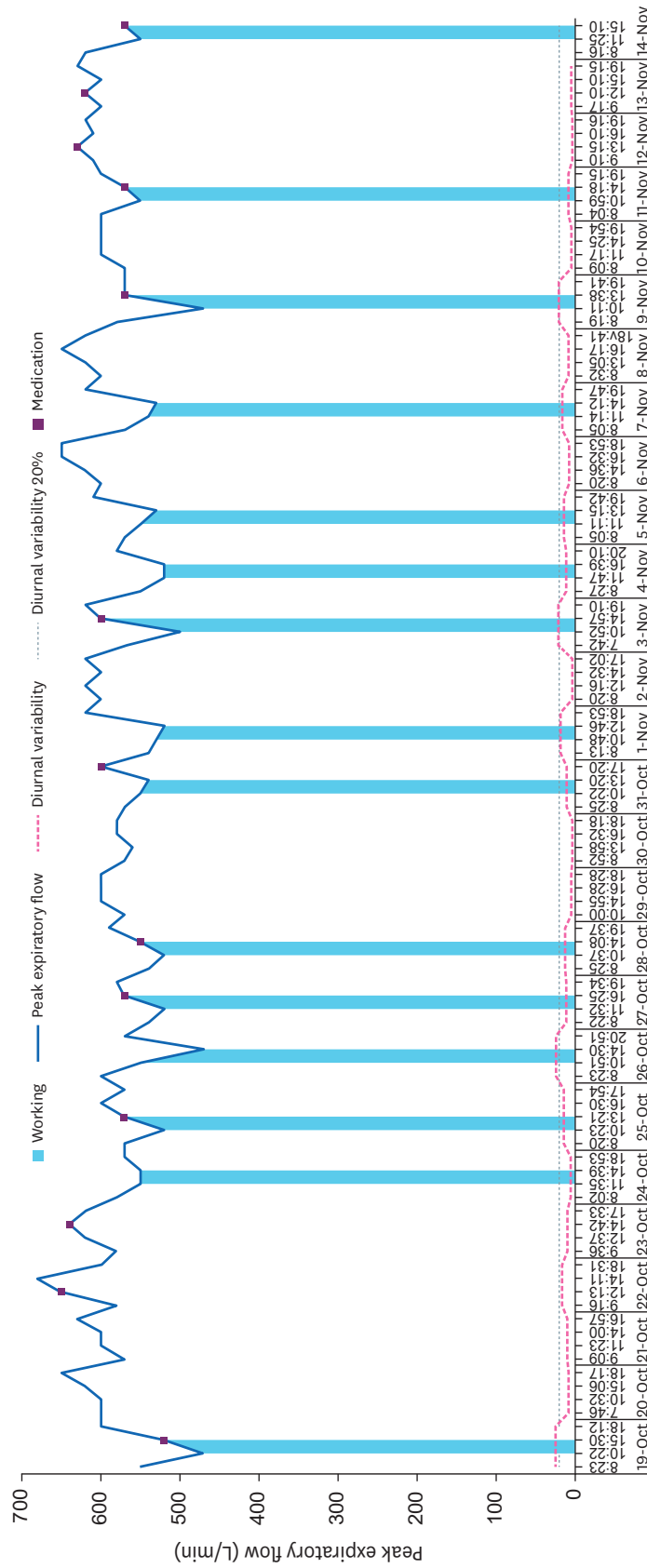


Fig. 5. Serial peak expiratory flow measurement from October 19, 2022, to November 14, 2022.

DISCUSSION AND CONCLUSION

The diagnosis of occupational asthma begins with confirming its presence. Asthma can be assessed using pulmonary function tests (PFT), which measure the reduced FEV₁/FVC ratio (< 70%) in baseline spirometry. The baseline spirometry of the present patient showed a normal FEV₁/FVC ratio of 85%. In the case of clinical features of asthma with normal baseline spirometry findings, a nonspecific bronchoprovocation challenge is recommended to demonstrate the reversibility of airflow limitation or airway hyperresponsiveness. The sensitivity of the methacholine bronchial provocation test using the non-deep inhalation method was approximately 100% when the cutoff point of PC20 for a positive conclusion was set to 8–16 mg/mL.⁶ Although its clinical usefulness has not been fully concluded, the grades of airway hyperresponsiveness determined by the methacholine bronchial provocation test have been proposed to be borderline (4–16 mg/mL), mild (1–4 mg/mL), moderate (0.25 mg/mL), and marked (< 0.25 mg/mL). The patient's PC20 was 2.60 mg/mL, which was a positive methacholine bronchial provocation test result, and the airway hyperresponsiveness was judged to be mild. This confirmed the patient's asthmatic status.

For the diagnosis of occupational asthma, after confirmation of the asthma, it is necessary to confirm that occupational factors have caused the asthma. An individual's occupational history is crucial for diagnosing occupational asthma. In the case of our patient, there were no previous reports of asthma-related symptoms or a prior asthma diagnosis prior to beginning work at the new workplace. However, after starting a new job in an enclosed and poorly ventilated environment, the patient developed asthma-related symptoms including dyspnea, coughing, and sputum production. He reported that there were similar symptoms among his co-workers. The patient experienced relief from asthma-related symptoms when away from work. These factors in their occupational history raised suspicions of occupational asthma, but they were not sufficient to provide a definitive diagnosis.

Various algorithms have been proposed to diagnose occupational asthma.¹ Serial PEF is a widely accepted and commonly used method to diagnose occupational asthma by demonstrating workplace-related airway obstruction. The sensitivity and specificity of PEF were 64% and 77%, respectively, compared with the specific inhalation challenge. Compared to expert diagnosis, the sensitivity and specificity of PEF were determined to be 75% and 94%, respectively.⁷ The normal variation in PEF during the next 24 hours should be less than 15%. A variation of 20% or more, which is related to workplace exposure, is indicative of occupational asthma. There are many ways to calculate diurnal variability. The Korea Occupational Safety and Health Agency recommends a specific method for calculating diurnal variability, which was used in this study. The mean PEF variation on working days was 15%, but the highest variation was 24%, with 4 days of 4 weeks having a variation of over 20% related to workplace exposure. Conversely, no day showed variations exceeding 20% during the remaining days. A variation in PEF of 20% or more during working days compared to rest days supports the diagnosis of occupational asthma, with a reported sensitivity of 93% and specificity of 90%.⁷ A significant difference of more than 16 L/min in the average PEF between working and rest days can be used to diagnose occupational asthma with a sensitivity of 70%.⁸ The average PEF on working days was 558 L/min and on rest days, it was 609 L/min. The difference in the average PEF between working and rest days was 52 L/min. PEF deterioration during working days and improvement on rest days also suggest occupational asthma.⁷ The results of comprehensive PEF interpretations were sufficient to diagnose occupational asthma.

To properly diagnose and treat occupational asthma, it is important to identify the specific causative agents responsible for the condition. There are more than 350 agents, including a range of airborne substances in the workplace, that have been reported to cause occupational asthma. The most common causes are high-molecular-weight agents, such as flour, and low-molecular-weight agents, such as diisocyanates, which are responsible for approximately 20% of occupational asthma cases.⁹ Skin testing is usually the preferred method for the identification of potential allergens that cause symptoms, as it offers a more comprehensive examination of allergens. However, this may be limited by the need for patients to discontinue their medication. In contrast, *in vitro* testing is usually unaffected by medication. As the patient was receiving asthma treatment, discontinuation of the medication for skin testing was not feasible. For the patient, ssIgE levels for 92 allergens were investigated using MAST. Although the patient's immunological assessment revealed sensitivity to cats, it is unlikely that the onset or exacerbation of asthma was caused by cats based on the patient's clinical features, including the onset of asthma-related symptoms, changes in the workplace, and serial PEF results. No allergens with a class of +2 or higher related to the patient's workplace were detected. The detection of ssIgE unrelated to symptoms is common. ssIgE can be used to determine whether allergens contribute to asthma; however, the specific causative allergen may not always be identified. In these cases, the examination results must be considered in the context of the patient's clinical history, and the analysis of causation cannot be solely based on laboratory results.

The shooting range can be classified into two types, indoor and outdoor, and further divided into firing and air gun categories. The patient worked in an indoor air gun shooting range. The indoor air gun shooting range is not widely known as a potential cause of asthma, but poor indoor air pollution is considered a causative agent. Air pollutants are substances that can be present in the air in the form of particles or gases and can adversely affect human health. Indoor air pollution can result from outdoor air pollution, tobacco smoke, heating and cooling systems, building materials, furniture, and electrical equipment. In addition, indoor air gun shooting ranges can contribute to the generation of air pollutants due to shooting activities and the use of chemicals during maintenance.

Indoor air gun shooting ranges may be exposed to increased levels of air pollutants.⁴ The 2019 indoor air quality data from the patients' workplaces revealed that all air pollutant levels met South Korea's Indoor Air Quality Control Act standards. However, these measurements were not taken during shooting or cleaning activities and may differ from the actual exposure levels of the patient. It is unfortunate that an on-site indoor pollution assessment was not carried out, and instead, an indirect assessment had to be relied upon for the exposure assessment. Airborne particulate levels during a shooting in indoor air gun shooting ranges have been observed to be 100–1,000 times higher than the normal background concentrations.¹⁰ Based on the current epidemiological evidence linking short-term air pollution exposure to asthma due to environmental exposure, it is more plausible to link a patient's occupational indoor air pollution exposure to the development of asthma because it is more frequent and intense compared to environmental exposure. The patient's workplace, which was an indoor air gun shooting range, lacked proper ventilation, potentially leading to elevated air pollutant levels and occupational asthma. Inadequate ventilation systems for indoor shooting ranges have been widely criticized. Inadequate ventilation can increase humidity levels, leading to increased condensation on surfaces and creating a moist environment that promotes mold growth. Moreover, stagnant air caused by inadequate ventilation can increase the concentration of mold spores in the air, thereby increasing

exposure to spores.¹¹ The lead concentration in the air in an indoor air gun shooting range without proper ventilation was $11.50 \mu\text{g}/\text{m}^3$, but this decreased to $6.46 \mu\text{g}/\text{m}^3$ when ventilation was introduced, indicating the presence of elevated levels of air pollutants in the indoor environment without proper ventilation.¹²

Air pollution, particularly exposure to elevated levels of indoor pollutants such as particulate matter, NO_2 , VOCs, and mold, has been linked to the onset of asthma symptoms.³ Evidence suggests that traffic-related air pollution can significantly increase the risk of childhood asthma.¹³ Studies have also shown a correlation between poor air quality and asthma onset and exacerbation.¹⁴ The mechanisms by which air pollution affects the onset and exacerbation of asthma are not well understood; however, oxidative stress and the generation of reactive oxygen species play major roles.¹⁵ There was a positive correlation between indoor dampness or mold and the onset and worsening of asthma.¹¹ Additionally, atopy and allergic rhinitis, known risk factors for asthma, may increase the likelihood of developing asthma, particularly in conjunction with exposure to indoor air pollutants.

In the present case, there was an observed latency period of approximately one month from occupational exposure to the onset of symptoms. Indoor air pollution, including particulate matter, can contribute to the development or worsening of asthma through various mechanisms, such as allergen sensitization and increased airway responsiveness. Immunologically mediated occupational asthma typically exhibits an asymptomatic latency period that can range from weeks to years, depending on factors such as exposure intensity, specific sensitizers, and individual susceptibility. While irritant-mediated occupational asthma is not as well established, certain forms, such as low-dose reactive airway dysfunction syndrome and irritant-induced asthma, have been reported to have latency periods. Therefore, it is reasonable to assume a one-month asymptomatic latency period regardless of the type of occupational asthma. The projectiles used in the air guns were composed of pure lead. Lead particles may be directly exposed through ejection while passing through the barrel or indirectly through contact with hands, clothes, and other surfaces. Lead particles may also be dispersed when lead projectiles hit a target, causing indirect exposure.

Indoor air gun shooting ranges pose a lesser threat of lead exposure than indoor firing ranges, but elevated lead exposure levels have still been reported. The median blood lead level (BLL) of shooters using indoor air gun shooting ranges was $3.3 \mu\text{g}/\text{dL}$, with a range of $1.8\text{--}12.7 \mu\text{g}/\text{dL}$.¹⁶ The South Korean Ordinance of the Ministry of Employment and Labor sets the occupational exposure limit at less than $30 \mu\text{g}/\text{dL}$, but this does not guarantee that all workers are protected from health problems. According to data from 2017 to 2018, the average BLL among adults in the US was $0.86 \mu\text{g}/\text{dL}$, with the 95th percentile being $2.62 \mu\text{g}/\text{dL}$.¹⁷ The patient's BLL of $1.8 \mu\text{g}/\text{dL}$ is not considered high, but it is still higher than the average for the adult population and close to $2 \mu\text{g}/\text{dL}$. In Korea, the concentration of lead in the air at indoor air gun shooting ranges was $9.37 \mu\text{g}/\text{m}^3$, which is lower than the South Korean working environment standard of $50 \mu\text{g}/\text{m}^3$, but higher than South Korea's working environment standard ($0.5 \mu\text{g}/\text{m}^3$) and the American air quality standard ($1.5 \mu\text{g}/\text{m}^3$).¹² The working environment measurement data for the patient's workplace showed a TWA of lead of $0.0063 \text{ mg}/\text{m}^3$, making it challenging to establish individual-level evidence of lead exposure. Nevertheless, exposure of staff members to airborne lead concentrations of up to $7.14 \text{ mg}/\text{m}^3$ has occurred in indoor shooting ranges when sweeping the range.¹⁸

However, the relationship between lead levels and asthma has not yet been definitively established. Several studies have indicated that lead exposure may influence the development of allergies in type 1 T helper cells and type 2 T helper cells. It has also been explained that there is a connection between lead exposure and asthma, with the lead being capable of elevating the level of total IgE, and high levels of total IgE being associated with a higher likelihood of asthma development.¹⁹ It has been found that an elevated BLL is significantly related to asthma.²⁰

In conclusion, the patient was diagnosed with occupational asthma after confirmation of asthma through a PFT and a positive methacholine bronchial provocation test. The occupational history of the patient, which showed the development of asthma symptoms after starting work at a new workplace, and the higher PEF variation on working days compared to that on rest days supported the diagnosis of occupational asthma. It has been challenging to identify the exact occupational factors that lead to occupational asthma owing to the difficulty of conducting specific inhalation challenges related to indoor air pollution. However, this was not essential for the diagnosis of occupational asthma.¹ A comprehensive interpretation of these results is sufficient for the diagnosis of occupational asthma. The development of occupational asthma may have been influenced by a combination of factors, including poor indoor air pollution, exposure to lead, patient atopy, and allergic rhinitis. The patient's work-fitness was evaluated, and job alterations were recommended. The patient's symptoms improved after changing workplaces. Consequently, general ventilators were scheduled for installation within the range. This case report highlights the importance of considering occupational asthma as a possible diagnosis in indoor air gun shooting range workers presenting with respiratory symptoms, especially if these symptoms are present only during working hours. Research on indoor air pollution in indoor air gun ranges, methods to reduce indoor air pollution, and regulations on the installation of general ventilators and local exhaust systems are necessary for the future.

ACKNOWLEDGEMENTS

This study was supported by a 2022 research grant from Pusan National University Yangsan Hospital.

SUPPLEMENTARY MATERIAL

Supplementary Table 1

Daily records of the serial peak expiratory flow

[Click here to view](#)

REFERENCES

1. Tarlo SM, Lemiere C. Occupational asthma. *N Engl J Med* 2014;370(7):640-9.
[PUBMED](#) | [CROSSREF](#)
2. Karjalainen A, Kurppa K, Martikainen R, Karjalainen J, Klaukka T. Exploration of asthma risk by occupation--extended analysis of an incidence study of the Finnish population. *Scand J Work Environ Health* 2002;28(1):49-57.
[PUBMED](#) | [CROSSREF](#)

3. Vardoulakis S, Giagloglou E, Steinle S, Davis A, Sleuwenhoek A, Galea KS, et al. indoor exposure to selected air pollutants in the home environment: a systematic review. *Int J Environ Res Public Health* 2020;17(23):8972.
[PUBMED](#) | [CROSSREF](#)
4. Beaucham C, Page E, Alarcon WA, Calvert GM, Methner M, Schoonover TM, et al. Indoor firing ranges and elevated blood lead levels - United States, 2002–2013. *MMWR Morb Mortal Wkly Rep* 2014.63(16):347-51.
[PUBMED](#)
5. Laidlaw MA, Filippelli G, Mielke H, Gulson B, Ball AS. Lead exposure at firing ranges-a review. *Environ Health* 2017;16(1):34.
[PUBMED](#) | [CROSSREF](#)
6. Cockcroft DW. Direct challenge tests: Airway hyperresponsiveness in asthma: its measurement and clinical significance. *Chest* 2010;138(2 Suppl):18S-24S.
[PUBMED](#) | [CROSSREF](#)
7. Tarlo SM, Balmes J, Balkissoon R, Beach J, Beckett W, Bernstein D, et al. Diagnosis and management of work-related asthma: American College of Chest Physicians Consensus Statement. *Chest* 2008;134(3 Suppl):1S-41S.
[PUBMED](#) | [CROSSREF](#)
8. Anees W, Blainey D, Moore VC, Robertson K, Burge PS. Differentiating occupational asthmatics from non-occupational asthmatics and irritant-exposed workers. *Occup Med (Lond)* 2011;61(3):190-5.
[PUBMED](#) | [CROSSREF](#)
9. McDonald JC, Keynes HL, Meredith SK. Reported incidence of occupational asthma in the United Kingdom, 1989–97. *Occup Environ Med* 2000;57(12):823-9.
[PUBMED](#) | [CROSSREF](#)
10. Alcock R, Wajrak M, Oosthuizen J. Assessment of the effectiveness of ventilation controls in managing airborne and surface lead levels at a newly commissioned indoor shooting range. *Int J Environ Res Public Health* 2022;19(18):11711.
[PUBMED](#) | [CROSSREF](#)
11. Mendell MJ, Mirer AG, Cheung K, Tong M, Douwes J. Respiratory and allergic health effects of dampness, mold, and dampness-related agents: a review of the epidemiologic evidence. *Environ Health Perspect* 2011;119(6):748-56.
[PUBMED](#) | [CROSSREF](#)
12. Lee CH. Lead concentration in air and urinary lead concentration of trainees in indoor shooting range. Asan: Soon Chun Hyang University; 2019.
13. Khreis H, de Hoogh K, Nieuwenhuijsen MJ. Full-chain health impact assessment of traffic-related air pollution and childhood asthma. *Environ Int* 2018;114:365-75.
[PUBMED](#) | [CROSSREF](#)
14. Lang DM, Polansky M. Patterns of asthma mortality in Philadelphia from 1969 to 1991. *N Engl J Med* 1994;331(23):1542-6.
[PUBMED](#) | [CROSSREF](#)
15. Alexis NE, Carlsten C. Interplay of air pollution and asthma immunopathogenesis: a focused review of diesel exhaust and ozone. *Int Immunopharmacol* 2014;23(1):347-55.
[PUBMED](#) | [CROSSREF](#)
16. Demmeler M, Nowak D, Schierl R. High blood lead levels in recreational indoor-shooters. *Int Arch Occup Environ Health* 2009;82(4):539-42.
[PUBMED](#) | [CROSSREF](#)
17. Centers for Disease Control and Prevention. National report on human exposure to environmental chemicals. <https://www.cdc.gov/exposurereport/index.html>. Updated 2023. Accessed December 15, 2022.
18. Ochsmann E, Göen T, Schaller KH, Drexler H. Lead--still a health threat for marksmen. *Int J Hyg Environ Health* 2009;212(5):557-61.
[PUBMED](#) | [CROSSREF](#)
19. Sunyer J, Antó JM, Castellsagué J, Soriano JB, Roca J; The Spanish Group of the European Study of Asthma. Total serum IgE is associated with asthma independently of specific IgE levels. *Eur Respir J* 1996;9(9):1880-4.
[PUBMED](#) | [CROSSREF](#)
20. Zeng X, Xu X, Zheng X, Reponen T, Chen A, Huo X. Heavy metals in PM2.5 and in blood, and children's respiratory symptoms and asthma from an e-waste recycling area. *Environ Pollut* 2016;210:346-53.
[PUBMED](#) | [CROSSREF](#)