

## CASE STUDY

# Bermuda grass pollen allergen implicated in clinically relevant cross-reactivity to multiple grains: A case report

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**Abstract**

**Background:** Anaphylaxis is a severe and potentially fatal type of allergic reaction and is characterized by the rapid development of symptoms in the respiratory and circulatory systems, possibly leading to death if not treated properly. Occupational anaphylaxis, which does not exhibit significant differences in pathogenesis from the nonoccupational form, develops in response to work-related triggers. However, the onset of occupational anaphylaxis can also be triggered by other factors. Therefore, an unexpected episode may occur due to exposure to a previously sensitized antigen or cross-reaction in the occupational environment, even if the direct trigger has been removed. Accordingly, it is difficult to diagnosis and treat such cases and ensure avoidance of potential triggers.

**Case presentation:** An adult male patient developed anaphylaxis following exposure to grass antigens while replacing and burying sewer pipes at a theme park. He later developed cross-reactivity to other grains. Despite symptomatic treatment, his total serum level of allergen-specific immunoglobulin E (Ig E) antibodies continuously increased, and thus, he was admitted with severe hypersensitivity, at which time his serum levels of Ig E antibodies specific for Bermuda grass, wheat, and rice had also increased.

**Conclusion:** In Korea, Bermuda grass is rarely seen and is generally found in athletic fields or theme parks. Following exposure to this relatively rare grass, our patient exhibited new anaphylactic responses to various external antigens. Therefore, we attribute his severe anaphylaxis to sensitization caused by Bermuda grass exposure and cross-reactive hypersensitivity to other grains.

**KEYWORDS**

Bermuda grass, occupational anaphylaxis, occupational asthma, Omalizumab, plant antigen

## 1 | BACKGROUND

Anaphylaxis is a severe, systemic, and potentially fatal rapid-onset type reaction involving both immunoglobulin E (Ig E) and non-Ig E-mediated allergic responses.

This reaction generally accompanies symptoms in skin and mucous membranes and rapidly affects the airway and respiratory and circulatory systems. Accordingly, prompt and appropriate treatment is required to avoid a fatal outcome.<sup>1</sup>

Recent studies have emphasized the potential abilities of workplace factors to trigger anaphylaxis, leading to the term "occupational anaphylaxis".<sup>2</sup> Although occupational and nonoccupational anaphylaxis do not exhibit significant pathological differences, the circumstances leading to the development of a severe allergic reaction are different.<sup>3</sup> Accordingly, it may be difficult to determine the cause of anaphylaxis, provide appropriate treatment, and avoid the triggering antigen(s). Particularly, workers sensitized to occupational allergens may be exposed to these allergens outside the workplace or may exhibit cross-reactivity to other antigens, leading to a continued risk of anaphylaxis even when the trigger has been removed from the workplace.

Previous reports have described the development of anaphylaxis in response to various occupational sensitizing agents as well as cross-reactivity. For example, a health care worker who exhibited symptoms of contact dermatitis in response to natural rubber latex gloves experienced an anaphylactic reaction by coming into contact with a natural rubber latex glove worn by a physician during a gynecologic examination.<sup>4</sup> Additionally, a health care worker who had become sensitized to the natural rubber in latex gloves developed anaphylaxis after eating raw manioc, an edible root belonging to the spurge family.<sup>5</sup> Regarding food-borne allergens, a fishmonger sensitized to creatine kinase in fish later developed various seafood-related allergic reactions,<sup>6</sup> and a worker at a spice factory was sensitized via inhalation and developed an anaphylactic reaction after consuming coriander.<sup>7</sup> However, none of these afore-mentioned cases reported persistent hypersensitivity or were refractory to symptomatic treatment.

In this report, we describe our experience with a case of occupational anaphylaxis in a patient following skin and inhalation exposure to Bermuda grass antigen while replacing and burying sewer pipes under the lawn of a theme park. This reaction potentially led to cross-reactivity to the consumption of grain-based carbohydrates and exposure to various external factors, with persistent severe hypersensitivity.

## 2 | CASE PRESENTATION

A 35-year-old man was referred to a tertiary hospital with persistent edema, rash, and dyspnea and a continuous increase in serum allergen-specific Ig E levels of several months of duration. For more than 10 years, the patient's occupational tasks had involved burying sewer pipes and installing manholes in Jeolla province, Korea. On an average, he spent more than 10 hours per day replacing sewer pipes buried under asphalt roads near urban residential areas and was exposed to asphalt, concrete, and sewage sludge.

From April to July 2015, he replaced sewer pipes buried under the lawn at a theme park, which differed from his usual working environment and led to his exposure to large

amounts of grass, rust, and pollen. At this time, he was also exposed to a foreign grass species (ie, not the general Korean *Zoysia* grass species). In July, he began to develop a cough while outside or working, which did not improve 1 month later upon completing his work at the theme park. In August, he received treatment for dyspnea, coughing, and edema over a 4-week period. Despite treatment, however, he continued to develop facial edema and dyspnea after consuming various types of foods, including apple, garlic, potato, brown rice, red bean-based snacks, ramen, corn, and sweet potato, and also experienced numbness in his leg. He reported abnormal blood test results at the time (data not available), and he was scheduled to undergo allergy and asthma testing.

However, on August 29, he experienced dyspnea and numbness of the leg while working on a nearby lawn and was admitted to the emergency room. On admission, he exhibited wheezing in left lower lung field and the following laboratory findings: white blood cell count (WBC),  $9.37 \times 10^3/\mu\text{L}$ ; neutrophils, 64.6%; lymphocytes, 27.0%; eosinophils, 0.4%; C-reactive protein (CRP), 0.01 mg/dL; thyroid-stimulating hormone (TSH), 1.46 IU; free T4, 1.17 ng/dL; and total T3, 1.33 mmol/L. Although chest radiography did not indicate abnormalities, chest computed tomography revealed mild thickening in both lower lung fields and severe fatty liver (Table 1).

On August 31, he continued to experience facial edema, leg numbness, and dyspnea, accompanied by the following laboratory values: WBC,  $14.26 \times 10^3/\mu\text{L}$ ; neutrophils,  $7.34 \times 10^3/\mu\text{L}$ ; eosinophils, 0.20%; Ig E  $>3000.0$  IU/mL; and aspartate aminotransferase/alanine aminotransferase (AST/ALT), 34/62 IU/L (normal range: AST 10 to 40 and ALT 7 to 56 IU/L). He was prescribed dexamethasone 5 mg and chlorpheniramine 4 mg intramuscular (IM), oral methylprednisolone (methylon®) 16 mg, oral antihistamine: fexonidine HCl 180 mg (Allegra®), levocetirizine 10 mg (Xyzal®), cimetidine 400 mg, and salbutamol sulfate (Ventolin evohaler®) 0.1 mg. Additionally, a pulmonary function test (PFT) and serum allergen-specific tests were conducted to identify the causes of allergy and asthma. The following antigens yielded positive results during serum allergen-specific Ig E testing (Table 2): *Dermatophagoides pteronyssinus*, class 2 (0.89 IU/mL); *Dermatophagoides farina*, class 2 (2.60 IU/mL); Bermuda grass, class 2; and redtop, class 2. Class 1 results were identified for orchard grass; white oak; rye; mugwort; mite farinae; and fescue meadow. His poor overall condition precluded skin-specific antigen and patch testing.

The patient did not react abnormally to systemic steroid or salbutamol sulfate (Ventolin evohaler®) inhalation and exhibited no abnormal PFT findings, and it was difficult to conduct further evaluations, such as the mannitol provocation test. The patient continued to exhibit allergic facial edema and dyspnea. Lung examination revealed slight expiratory wheezing and a blood test conducted on September 1 revealed elevated laboratory values

TABLE 1 Medical history timeline

Dates	Relevant Past Medical History and intervention		
•April to July 2015	A 35-year-old man without specific medical history was exposed to large amount of grass, rust, and pollen while working at a theme park to replace sewer pipes buried under lawn.		
Dates	Summaries from initial and follow-up visits	Diagnostic testing	Intervention
•Since July 2015	•Development of a cough while outside or working for a month	•Scheduled to undergo allergy and asthma testing	•Symptomatic management for dyspnea, coughing, and edema over a 4-week period.
•August 29 •The emergency room	•Experience of dyspnea, numbness of the leg, and admission to the ER room	•Serum allergen-specific test •Pulmonary function test	•Antihistamine •Systemic steroid
•Since August •Outpatient clinic	•No abnormal PFT finding •No abnormal reaction to salbutamol sulfate (Ventolin evohaler®) inhalation •Continuation to exhibit allergic facial edema and dyspnea	•Elevation of total serum allergen-specific Ig E level (> 3000 IU/mL)	•Antihistamine •Systemic steroid
•Since September •Outpatient clinic	•Improvement in symptoms after ceasing outdoor activities •Exacerbation of facial edema. •Drop in total serum allergen-specific Ig E levels. •Unexpected anaphylactic reactions caused by various external triggers	•Bermuda Grass and some allergen-specific Ig E testing yield positive results <sup>a</sup>	•Omalizumab (Xolair®) •Antihistamine •Systemic steroid
•Since December The outpatient clinic of referral hospital	•Allergic sensitization was refractory to treatment •Development of facial edema and dyspnea after consuming foods containing multiple grains. •Transfer to referral hospital.	•Bermuda grass and some allergen-specific Ig E testing yield positive results	•Omalizumab (Xolair®) •Antihistamine Systemic steroid
•Early 2016	•Sustained anaphylactic symptom •Angioedema due to apitoxin Allergic sensitization to multigrain		•Omalizumab (Xolair®) •Antihistamine •Systemic steroid (danazol, deflazacort) Cyclosporine
•Since then	•Symptoms gradually improved •He has not developed another episode of severe anaphylaxis		•Cessation of systemic steroid •Omalizumab (Xolair®) Antihistamine

<sup>a</sup>The detailed notations are shown in Table 2

for WBC ( $19.26 \times 10^3/\mu\text{L}$ ; neutrophils  $13.63 \times 10^3/\mu\text{L}$ ; eosinophils 0.10%), IgE ( $>3000.0 \text{ IU/mL}$ ), and AST/ALT (38/61 IU/L [normal range: AST 10 to 40 and ALT 7 to 56 IU/L]). There was no significant improvement in the clinical symptoms despite the additional prescription of antihistamine (ebastine 10 mg); therefore, monthly injection of subcutaneous Omalizumab (Xolair®) was added to the previous regimen on September 10. At the time of the outpatient visit in October, the patient still showed allergic facial edema, with no complaint of deterioration in any symptoms. Lung examination revealed slight expiratory wheezing. The treatment plan did not change.

He was admitted to the local emergency clinic on November 2 for additional treatment of an allergic reaction (dyspnea) with an unclear etiology, and testing on November 5 revealed that his total serum allergen-specific Ig E level was 910 IU/mL and AST/ALT was 46/95 IU/L (normal

range: AST 10 to 40 and ALT 7 to 56 IU/L). He had allergic facial edema, and his lung examination revealed slight expiratory wheezing. On the following outpatient clinic visit on November 19, he reported improvement in his symptoms after ceasing his outdoor activities (other sewer pipe workplace job). A controlled tapering of methylprednisolone (methylon®) from 16 mg to 8 mg was thus attempted, although this led to worsening of edematous symptom. On December 3, the total serum allergen-specific Ig E level was measured at 822.2 IU/mL. The occurrence of acute anaphylaxis had decreased, but the patient continued to experience facial edema as well as hypertension and alopecia. He also experienced a worsening of rash and angioedema upon consuming grains during treatment and was repeatedly admitted to the emergency room for unexpected anaphylactic reactions caused by various external triggers.

**TABLE 2** Changes in serum allergen-specific immunoglobulin E (Ig E) antibody levels and results of laboratory testing performed for the differential diagnosis

Serum allergen-specific Ig-E antibody	02/Sep/15	15/Nov/15	03/Dec/15	05/Jan/16
Total allergen	3000 (IU/mL)	910.1 (IU/mL)	822.2 (IU/mL)	2430 (IU/mL)
Bermuda grass	Class 2	—	—	Class 3 (4.09 IU/mL)
Redtop	Class 2	—	—	
<i>D. farinae</i>	Class 2(2.60 IU/mL)	—	—	Class 2 (2.40 IU/mL)
<i>D. pteronyssinus</i>	Class 2(0.89 IU/mL)	—	—	WNL* (0.34 IU/mL)
White oak	Class 1	—	—	—
Rye, cultivated	Class 1	—	—	Class 2
Mugwort	Class 1	—	—	—
House dust	Negative	—	—	Negative
Fescue meadow	Class 1	—	—	—
Wheat	—	—	—	Class 2 (2.47 IU/mL)
Rice	—	—	—	Class 2 (4.42 IU/mL)
ECP*	—	—	—	WNL (9.82 ug/L)
Tryptase	—	—	—	WNL (5.64 ug/L)
C1 inactivator	—	—	—	WNL (33.4 mg/ dL)

Despite continued treatment for anaphylaxis, the total allergen-specific and Bermuda grass-specific Ig E antibody levels continued to increase. Bermuda grass, a non-native species, is rarely planted in South Korea and generally carries a low chance of antigen sensitization.

WNL, within normal limits; ECP, eosinophil cationic protein.

Generally, his allergic sensitization was refractory to treatment, leading to his admission to a tertiary referral hospital. He repeatedly developed facial edema and dyspnea after consuming foods containing apple, garlic, potato, brown rice, red bean, ramen, corn, and sweet potato. Laboratory tests conducted on December 15 revealed an increase in the WBC count to  $13.4 \times 10^3/\mu\text{L}$ : neutrophils, 58.3%; lymphocytes, 33.4%; eosinophils, 1.0%; and AST/ALT, 59/157 IU/L (normal range: AST 10 to 40 and ALT 7 to 56 IU/L). The total serum IgE level had increased to 2430 IU/mL, and specific antigen testing revealed the following specific Ig E levels (normal ranges): *Dermatophagoides farinae*, 2.40 (0-0.35) IU/mL; wheat, 2.47 (0-0.35) IU/mL; Bermuda grass, 4.09 (0-0.35) IU/mL; rice, 4.42 (0-0.35) IU/mL; *Dermatophagoides pteronyssinus*, 0.34 (0-0.35) IU/mL; and eosinophil cationic protein (ECP), 9.82 (0-13.5)  $\mu\text{g/L}$  (Table 2). Tryptase and C1 inactivator testing was conducted to address the differential diagnosis, which included other diseases that may cause anaphylaxis, and yielded values of 5.64 (0-11.4)  $\mu\text{g/L}$  and 33.4 (21.0-39.0) mg/dL, respectively. Again, the patient's poor overall condition precluded skin-specific antigen and patch testing.

Although the patient continued receiving 300 mg Omalizumab (Xolair®) subcutaneously every 4-6 weeks, antihistamine and decongestant (fexonidine 180 mg, levocetirizine 5 mg, ebastine 10 mg, pseudoephedrine 120 mg), immunosuppressant (cyclosporine 100 mg), and systemic steroid treatments, his symptom did not improve significantly. Hence, the antihistamine and systemic steroid were changed to danazol 100 mg and deflazacort 12 mg. In March 2016, however, he was stung by a bee and developed angioedema. Testing on April 7, 2016 revealed an increase in honey allergen-specific Ig E level to 21.20 IU/mL. Accordingly, antihistamine and antileukotriene medication treatment was maintained to address sensitization to grass pollen and house dust mites, various food grains (including rice), and food-induced and idiopathic anaphylaxis. Omalizumab 300 mg was injected at 4- to 6-week intervals, and allergen immunotherapy for grass pollen was administered. The patient's symptoms gradually improved, and systemic steroid therapy was stopped. He has not developed another episode of severe anaphylaxis requiring admission to the emergency room.

### 3 | DISCUSSION AND CONCLUSIONS

Occupational anaphylaxis or anaphylaxis in response to causes and conditions attributable to a particular work environment is pathologically similar to general anaphylaxis. However, occupational anaphylaxis is generally Ig E-mediated, and the route and intensity of antigen exposure differ from that of nonoccupational anaphylaxis. For occupational anaphylaxis, exposure via ingestion is rare, compared to accidental exposure via inhalation, skin contact, animal sting/bites, and needle injuries, which may allow sensitization to low doses of allergen.<sup>2</sup> Although repeated exposure to the triggering antigen in a workplace setting can result in a rapid progression to allergic reaction, a high frequency of exposure may lead to clinical tolerance of the allergen.<sup>8</sup> Furthermore, allergic reactions attributable to the same cause may exhibit different sensitization patterns, depending on the site of exposure,<sup>9</sup> as well as cofactors such as temperature and intensity of activity.

In a study analyzing Korean occupational asthma between 1992 and 2006, 218 cases were compensated as occupational asthma by the Korea Workers' Compensation and Welfare Service (COMWEL) and 286 cases were reported as occupational asthma by Occupational Safety and Health Research Institute of Korea Occupational Safety and Health Agency (OSHRI, KOSHA) implemented surveillance system. Based on these data, the annual mean incidence rate of workers' occupational asthma was estimated at 1.6/million workers according to compensation data and 3.5/million on the basis of surveillance data. The most frequently reported causative agent was isocyanate followed by reactive dye in dyeing factories. Chemicals, metals, and dust were also found as causative agents, but other causes, such as plants, were not common in compensated occupational asthma.<sup>10</sup> They also emphasized that the compensation and voluntary surveillance data may underestimate the incidence of occupational asthma. Another study analyzed the data from work-related asthma surveillance between 2004 and 2009 in Korea and reported 236 cases of occupational asthma. The most frequently reported causative agent was isocyanate (46.6%) followed by flour/grain (8.5%) and metals (5.9%) and one case of plant antigen (0.4%) was reported during the surveillance.<sup>11</sup> A systemic review of 21 publications on occupational asthma revealed that the overall median population attributable risk (PAR) for occupational asthma was 17.6%.<sup>12</sup> These findings underscore the need for further preventive measures to reduce the incidence of occupational asthma and thus anaphylaxis.

As noted earlier, sensitization to occupational antigens may lead to reactions outside of the workplace, or vice versa, and antigen cross-reactivity may lead to the development of a

new allergic reaction to food,<sup>3</sup> particularly if the initial sensitization occurred via inhalation or skin exposure. Pollen-food syndrome, a widely known form of cross-reactivity that may develop without direct sensitization,<sup>13</sup> results from cross-reactivity between pollen-specific Ig E and homologous proteins found in fruits and vegetables. Cases of cross-reactivity anaphylaxis in response to various foods (eg, rice, Rosaceae plants, beer, and peanuts) have been reported in lipid transfer protein (LTP)-allergic patients, although these cases were not attributable to occupational factors. In the present case, the first anaphylactic symptom occurred between July and August when there were wide exposure to grass allergens. The symptoms were relieved after the patient quit outdoor work in the early stage of treatment. Although his symptoms did not recover sufficiently during the high grass allergen season between May and September,<sup>14</sup> anaphylactic symptoms developed continuously after being exposed to various grains. He had experienced several anaphylactic attacks and was newly sensitized to apitoxin despite receiving omalizumab, high-dose glucocorticoid, and immunosuppressant. There were high levels of Ig E antibodies specific to most allergens he had been exposed to, indicating that his severe allergic reactions were potentially attributable to cross-reactivity and presumably due to the hypersensitivity to other antigens caused by existing antigens. Although the provocation and skin tests required for diagnostic confirmation were not performed due to severe sensitization, the patient exhibited an elevated serum level of Bermuda grass-specific Ig E antibodies, which is an uncommon finding in a construction worker in Korea. A previous study reported that the rates of sensitization to pollen allergens of *Dermatophagoides farina* and *Dermatophagoides pteronyssinus* have increased since 1999 and reached a level of 40% in 2008. Similarly, the rates of sensitization to alder, oak, mugwort, ragweed, Japanese hop, and timothy grass pollen have also increased significantly since 1999. However, the rate of sensitization to Bermuda grass pollen has remained at approximately 5%, and a follow-up study of antigens found that this pollen type is associated with a relatively low risk of sensitization.<sup>15</sup> A recent study for Korean allergic pollens calendar collected allergen data between 1997 and 2009 and revealed that Bermuda grass pollen was a possible grass allergen in Korea, although no Bermuda grass pollen was included in their calendar. Such an omission might be due to the difficulty in discriminating individual grass pollens<sup>14</sup> and could be the reason that Bermuda grass (*Cynodon Dactylon*) has not yet been reported as a causative agent for asthma or anaphylaxis.

A 2006 study reported that 95.6% of grasses cultivated in South Korea are of the *Zoysia* species.<sup>16</sup> However, because *Zoysia* grass species only remain green for a short period of time, foreign grass species, such as Bermuda grass, are often planted in theme parks and athletic fields. Accordingly, the patient in this case

report was likely sensitized to Bermuda grass while working at the theme park, especially as he conducted his work during early summer, when this grass pollinates. Furthermore, the nature of his work, which involved the repeated removal and replacement of the lawn, likely facilitated the sensitization by exposing him to a high concentration of Bermuda grass material. Given the timing of the specific Ig E antibody production after working in a lawn environment and lack of a prior history of grain-related allergy, the exposure to Bermuda grass, while working at a theme park, was identified as the likely cause of anaphylaxis and cross-reactivity.

In conclusion, although the exact pathogenesis of anaphylaxis has not been clarified, this condition is known to be potentially fatal in the absence of proper treatment. Additionally, the management of anaphylaxis requires avoiding exposure to the allergen and receiving immediate treatment for symptoms. Accordingly, a thorough identification of the exposure route and frequency, progression, cofactors, and potential sensitization scenario is required to manage occupational anaphylaxis. Still, cross-reactivity may lead to an attack, even in the absence of direct contact with the suspected antigen. Thus, allergen exposure and anaphylaxis onset are therefore possible in a nonworkplace environment even after removing occupational exposure.

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## DISCLOSURE

*Ethical approval and consent to participate:* This study utilized workers' compensation data obtained from KOSHA and did not include identifiable personal information. This article was exempted from Institutional Review Board review of OSHRI, KOSHA *Consent for publication:* We have obtained consent to publish from the participant to report individual data. *Availability of supporting data:* The present study utilized workers' compensation data, which were formally obtained from KOSHA, and did not include identifiable personal information. *Competing interest:* The authors have no potential conflicts of interest that are relevant to this article. *Approval of the research protocol:* N/A. *Informed consent:* Written informed consent was obtained from the patient for publication of this case report and any accompanying data. *Registry and registration no. of the study/trial:* This study utilized workers' compensation data obtained from KOSHA and did not include identifiable personal information. *Animal studies:* N/A.

## CONFLICT OF INTEREST

The authors have no potential conflicts of interest that are relevant to this article.

## AUTHORS' CONTRIBUTION

KYJ and LJH contributed to the concept and design of this study. KYJ performed the analysis of the medical records. KYJ and LJH drafted the manuscript. All authors have read and approved of the final manuscript.

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