

A high school gym-induced disease

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ABSTRACT

Physical exercise may induce upper and lower airway symptoms such as rhinitis and asthma. Rhinitis symptoms are often neglected although runny nose and nasal congestion may interfere with performance of the affected individual. A detailed history regarding locality and time of symptoms is of most significance for taking the appropriate diagnostic measures and identifying, as in this case, an uncommon form of allergic rhinitis to airborne spores from moulds.

School sport should contribute to social, cognitive, emotional and physical development of children and adolescents. However, in rare cases school sport may also induce clinical symptoms and give rise to morbidity.

Here we present a high school student with nasal symptoms which were first diagnosed as exercise-induced rhinitis.¹ Interestingly, nasal secretion and obstruction exclusively developed in the high school gym; outdoor physical exercise was tolerated without any symptoms. This “gym-induced” rhinitis was neglected for several months, although the symptoms interfered with the student’s performance. One reason for neglect of rhinitis is difficulties in definition, classification and diagnosis of this heterogeneous disease. The clinical spectrum ranges from allergic hay fever in the summer months to “skier’s or runner’s nose”, the latter being induced by forced inhalation of cold, dry air with subsequent nasal secretion (rhinorrhoea) and/or obstruction (nasal congestion).²

CASE REPORT

During the preceding 2 years a 16-year-old high school student had developed pruritus, sneezing, watery secretion and nasal obstruction approximately 45 minutes after the start of the physical education class in the high school gym. These nasal symptoms interfered with his performance, especially in the second part of the 1.5 h class. The symptoms varied but slowly progressed in intensity, leading additionally to development of conjunctival injection and itchiness in recent months. Twice he had also experienced acute angioedema of the eyelids, while asthma symptoms did not occur. During outdoor sport or recreational activities he did not develop rhinitis or any other symptoms. Besides the high school gym no other gyms were frequented. He had not received symptomatic treatment for rhinitis.

Due to onset of nasal symptoms during physical exercise, primarily exercise-induced rhinitis had been assumed by the treating family physician and school physician, respectively. After onset of conjunctivitis and angioedema of the eyelids the

patient was referred to our outpatient allergy clinic. The co-occurrence of eye symptoms and the restriction of symptoms to the school gym, however, made exercise-induced rhinitis unlikely and complete allergological work-up of the patient was initiated.

Routine laboratory parameters including serum levels for C4 and C1-inhibitor as well as C1-inhibitor function (to rule out hereditary angioedema) and total serum immunoglobulin E (IgE) were within normal limits. Natural rubber latex-specific IgE (eg material of gymnastic mats) was negative. Screening prick testing with inhaled allergens such as pollen, cat and dog dander, house dust mites and common food allergens including cow’s milk, egg, finned fish and hazel nut revealed IgE-mediated sensitisations for birch, alder, wheat and grass pollen. Prick testing with additional aeroallergens (moulds, horse dander, storage mites, and rubber latex) was negative. Due to the well-known limited sensitivity of mould prick tests, additionally intradermal tests with a panel of fungi including *Alternaria alternata*, *Aspergillus fumigatus*, *Aureobasidium pullulans*, *Cladosporium herbarum*, *Fusarium roseum*, *Mucor mucedo*, and *Penicillium notatum* were performed and showed a strongly positive reaction to *Alternaria alternata* (after 20 minutes wheal diameter 8 mm, erythema 15 mm). Elevated *Alternaria alternata*-specific IgE (m6, CAP System, Pharmacia Diagnostics, Uppsala, Sweden) of 2.5 kU/l was measured. Further questioning revealed that the high school gymnasium was built in 1970; the brickwork suffered damage by water including visible wet and mouldy spots. Dust was collected from the floor of the high school gym, and several fungal species including *Alternaria alternata*, *Aspergillus fumigatus*, *Penicillium* species, and *Aureobasidium pullulans* were identified.

Considering the typical clinical symptoms developing in a timely and local relation to exposure to *Alternaria* spores, detection of *Alternaria alternata*-specific IgE and positive intradermal tests, diagnosis of acute intermittent allergic rhinitis to *Alternaria alternata* was established. Meanwhile, the old school gym was torn down and has been replaced by a new construction. Since then the patient has not developed any symptoms during physical education class.

DISCUSSION

Onset of rhinitis including watery nasal secretion as the prominent clinical feature during sport activities usually implies the diagnosis of a non-allergic, exercise-induced rhinitis.¹ Formerly the term “vasomotor rhinitis” was often used, but this implies that the underlying cause is a vascular or neurological dysfunction of the mucosa,

whereas there are no sound data to support this notion.³ The disease is “non-allergic” when allergy has not been proven by proper allergy examination (history, skin test, measurement of allergen-specific IgE antibodies).

Allergic rhinitis is defined as a symptomatic hypersensitivity reaction of the nasal mucosa induced by IgE-mediated immunological response to allergen exposure.⁴ Symptoms of allergic rhinitis include nasal itching, sneezing, rhinorrhoea, and nasal obstruction. It is subdivided into intermittent (symptoms are present fewer than 4 days a week or for less than 4 weeks) or persistent disease (symptoms are present more than 4 days a week and for more than 4 weeks). Diagnosis of allergic rhinitis is based on a characteristic history of allergy symptoms and results of allergological testing, including in vitro and in vivo detection of allergen-specific IgE. Key to correct diagnosis remains an accurate and detailed history of potential allergen exposure.

Compared with pollen, house dust mites or animal dander allergy, establishing the diagnosis of a mould allergy may be more challenging. Diagnosis relies on positive skin testing with commercially available mould extracts and a detailed history suggestive of mould allergy, for example, limited occurrence of symptoms in specific buildings or in a damp basement, or symptoms when working at a compost pile. Most patients sensitised to mould spores do not exclusively react in skin tests to mould extracts but show polysensitisation to several inhaled allergens, as was the case in our patient, with sensitisation to several tree and grass pollens without seasonal symptoms.⁵ The complementary determination of *Alternaria*-specific IgE supported the diagnosis in our case. However, in a great number of patients with mould allergy correlation of serum IgE and skin tests is lacking. One major problem in the diagnosis and therapy of mould allergy is the production of standardised allergen products, including skin test solutions, solid-phase-bound allergens for IgE detection and therapeutic vaccines, because the raw material, namely the culture of fungi, contains more mycelium than spores and not all relevant allergens of spores may be found within the mycelium.⁶ Furthermore, composition of allergen extracts may change during repeated subcultures of fungi.⁷ Possibly, development of recombinant fungal allergens may solve this problem in the future.

Of most significance for the diagnosis of allergic rhinitis to *Alternaria* is a relevant exposure. Fungi are naturally occurring in our environment and mould spores are ubiquitously found as indoor and outdoor biologicals dispersed in the atmosphere. If mould concentration exceeds a critical limit, the large number of airborne spores may cause allergic diseases. Mould spores are small in size (3–10 µm), penetrate deeply into the respiratory tract, and can provoke rhinitis as well as asthma.⁸ For unknown reasons, children seem to be sensitised to mould spores more often than adults.⁹ Humidity and organic substrates are necessary for optimal mould growth. Warm indoor air in a gym absorbs more water than the cooler outdoor air and insufficient ventilation may consequently induce condensation, particularly around windows and corners. In our case an increased degree of moisture in the gym was particularly observed at sites of water damage and leakage which had not been readily repaired and sufficiently dried. Striking in our patient was the latency of allergen exposure, that is, the time between entering the gym and the onset of rhinitis symptoms.

What is already known on this topic

Nasal secretion and obstruction occurring during physical activity is a well-known phenomenon called exercise-induced rhinitis.

What this study adds

In case of rhinitic symptoms strictly limited to indoor activities, unusual causes like IgE-mediated allergy to indoor allergens such as mould have to be considered.

It is tempting to speculate that only swirling of dust particles induced by sporting activities of a whole group of students led to a relevant concentration of airborne spores entering the respiratory tract by inhalation.

The patient was advised against sporting activities in the mould-contaminated school gymnasium because permanent and recurrent airway exposure to a relevant allergen may lead to further progression of airway symptoms and the development of allergic asthma. Our patient was also informed about the natural habitat of *Alternaria* species, especially the prevalence on food such as fruits and vegetables and outdoor plants. Patients with *Alternaria* sensitisation may develop symptoms from May to September when working in the garden (eg mowing the lawn) or during recreational activities in the meadows. A peak of symptoms may be observed in July and August, when *Alternaria* produces a great amount of spores which are then dispersed by the wind. Patients with *Alternaria*-induced rhinitis or asthma may receive an *Alternaria*-specific immunotherapy; however, indication and management should be reserved for experienced allergologists.^{5 10}

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