

9. REISMAN R, MAURIELLO P, DAVIS G. A double-blind study of the effectiveness of high-efficiency particulate air (HEPA) filter in the treatment of patients with perennial allergic rhinitis and asthma. *J Allergy Clin Immunol* 1990;**85**:1050–1059.

Swimming-pool pneumonitis

E. González-Mancebo*, M. L. Díez Gomez, Z. Pulido, T. Alfaya, F. León

Key words: *Candida albicans*; fungi; hypersensitivity pneumonitis; indoor swimming pool.

● **HYPERSENSITIVITY** pneumonitis (HP) is well known worldwide, but we are constantly discovering more antigens involved in this disease. There are several cases of HP associated with sauna-takers (1–3), but there have been only two publications about HP related to indoor swimming pools (4, 5).

Our patient was a 43-year-old man who, 4 months before, had suffered an episode of fever of 40°C, headache, myalgia, nonproductive cough, chest tightness, and dyspnea. These symptoms appeared 4–6 h after he swam in a covered swimming pool, lasted for 24 h, and disappeared after acetaminophen administration. The patient remained asymptomatic for 1 month without using the swimming pool, but he presented the same symptoms when he used the pool again. After that, the patient had not visited the swimming pool for 3 months, and he had not suffered further episodes.

We performed microbiologic studies for mold growth with samples from this swimming pool. *Candida albicans* and *Aspergillus fumigatus* were isolated from swimming-pool environment samples.

Skin prick tests with a battery of common aeroallergens including *Alternaria tenuis*,

A. fumigatus, *Cladosporium herbarum*, and *Penicillium notatum* (Bial Aristegui, Bilbao, Spain) were carried out, with negative result.

Intradermal tests with *A. fumigatus* and *C. albicans* were also performed, yielding a positive result with *C. albicans* extract at 24 h.

Total serum IgE was 32 kU/l and specific IgE determinations to *A. tenuis*, *C. herbarum*, *P. notatum*, *A. fumigatus*, and *C. albicans* (Pharmacia CAP System, Sweden) were all negative.

Specific IgG antibodies were investigated by ELISA, with a positive result for *A. fumigatus* and *C. albicans* and a negative result for *Thermoactinomyces* spp. and *Micropolyspora* spp.

We performed a specific bronchial provocation test (PIPT) with 5 ml *A. fumigatus* extract (10 mg/ml) and 1 month later with 5 ml *C. albicans* extract (10 mg/ml) (6). PIPT with *A. fumigatus* extract was negative. PIPT with *C. albicans* extract was positive 30 h after challenge, reproducing the same symptoms in the patient. We also found medium and basal pulmonary field crackling in lung auscultation, leukocytosis of 11 900 cells/mm³ (neutrophils: 8900 cells/mm³), a decrease in *Pa*O₂ blood gasometry (*Pa*O₂ 67 mmHg), and chest radiography with basal and bilateral pulmonary interstitial infiltration. The patient required systemic corticosteroid and oxygen administration. We also performed PIPT with *C. albicans* on two healthy volunteers (swimming-pool workers) with negative result.

SDS–PAGE/IgG immunoblotting (7) and inhibition assays with *A. fumigatus* and *C. albicans* extracts were carried out. IgG immunoblotting showed bands with molecular masses of 30, 46, 55, 90, and >100 kDa in *A. fumigatus* extract and 46, 60, 90, and >100 kDa in *C. albicans* extract. Two of the proteins responsible for these reactivities (46 and >100 kDa) seemed to be the same protein in both extracts, since cross-reactivity was demonstrated by cross-inhibition.

In our patient, HP related to an indoor swimming-pool environment, and caused by *C. albicans*, was diagnosed. We could reproduce the clinical and laboratory findings through specific PIPT with the suspected agent. To the best of our knowledge, there is only one previous publication on *Candida* as the cause of HP (in a farmer exposed to wet hay [8]). The results of SDS–PAGE and IgG immunoblotting showed a 46-kDa protein in *A. fumigatus* and *C. albicans* extracts, already described in earlier studies (7, 9). There are no previous reports of the high-molecular-mass antigens (>100 kDa) that we observed in *A. fumigatus* and *C. albicans* and of the cross-reactivity among them. We also emphasize the value of a site visit in the diagnosis of HP (4). A previous publication describes endemic granulomatous pneumonitis in an indoor swimming pool and suggests the term “lifeguard lung” (5), but we note that this could be a problem that also affects users of swimming pools, who are not as frequently exposed to antigens.

*C/ Witerico 5, port 6, 3° D
28025 Madrid
Spain

Accepted for publication 3 April 2000
Allergy 2000; 55:782–783
Copyright © Munksgaard 2000
ISSN 0105-4538

References

1. METZGER WJ, PATTERSON R, FINK J, SEMERDJIAN R, ROBERTS M. Sauna-takers' disease. Hypersensitivity pneumonitis due to contaminated water in a home sauna. *JAMA* 1976;**236**:2209–2211.
2. EMBIL J, WARREN P, YAKRUS M, et al. Pulmonary illness associated with exposure to *Mycobacterium avium* complex in hot tub water. *Chest* 1997;**111**:813–816.
3. PATTERSON R. Studies of hypersensitivity lung disease with emphasis on a solid-phase radioimmunoassay as a potential diagnostic aid. *J Allergy Clin Immunol* 1978;**61**:216–219.

4. MORENO-ANCILLO A, VICENTE J, GOMEZ L, et al. Hypersensitivity pneumonitis related to a covered and heated swimming pool environment. *Int Arch Allergy Immunol* 1997;**114**:205–206.
5. ROSE CS, MARTYNY JW, NEWMAN LS, et al. “Lifeguard lung”: endemic granulomatous pneumonitis in an indoor swimming pool. *Am J Public Health* 1998;**88**:1795–1800.
6. HINOJOSA M, FRAJ J, DE LA HOZ B, ALCAZAR R, SUEIRO A. Hypersensitivity pneumonitis in workers exposed to esparto grass (*Stipa tenacissima*) fibers. *J Allergy Clin Immunol* 1996;**98**:985–991.
7. QUIRCE S, HINOJOSA M, BLANCO R, CESPON C, YOLDI M. *Aspergillus fumigatus* is the causative agent of hypersensitivity pneumonitis caused by esparto dust. *J Allergy Clin Immunol* 1998;**102**:147–148.
8. ANDO M, YOSHIDA K, NAKASHIMA H, SUGIHARA Y, KASHIDA Y. Role of *Candida albicans* in chronic hypersensitivity pneumonitis. *Chest* 1994;**105**:317–318.
9. ITO K, ISHIGURO A, KANBE T, TANAKA K, TORII S. Characterization of IgE-binding epitopes on *Candida albicans* enolase. *Clin Exp Allergy* 1995;**25**:529–535.

with two parallel ski tracks and the difference in altitude 51 m, where a controlled setting simulating an arctic environment at -11°C (relative humidity 80–100%) could be arranged. Six male, nonsmoking military conscripts, aged 18–21 years (weight 60–90 kg, height 175–190 cm, and 12-min running test result 2200–2960 m), were recruited on a voluntary basis from the garrison of Kajaani, Finland.

The subjects were encouraged to ski at a submaximal speed. The heart rate means during the exercise were 140–158/min, as monitored with a wireless heart rate meter (Polar Electro). The nasal volume (at 0–5 cm from the nostril) was registered with acoustic rhinometry (Rhin 2000), and the nasal flow at the gradient pressure of 75 Pa with active anterior rhinomanometry (Rhinotest MP 500) with face mask and foam-rubber nose pieces. The nasal secretions were aspirated with a small plastic

Nasal function of skiers in cold weather

T. Pirilä*, H. Kiukaanniemi, K. Jokinen

Key words: acoustic rhinometry; cold exposure; nasal secretion; rhinomanometry.

● IN the literature, controversial results can be found concerning nasal congestion in reaction to cold exposure. An increase or no significant change in nasal resistance has been noted after breathing of cold air either through a face mask (1) or in a climate chamber (2, 3).

The nose maintains its patency during exercise in a cold environment.

We studied nasal reactions in a 1200-m-long ski-tunnel (Huurre Ski-Tunnel, Vuokatti, Finland),

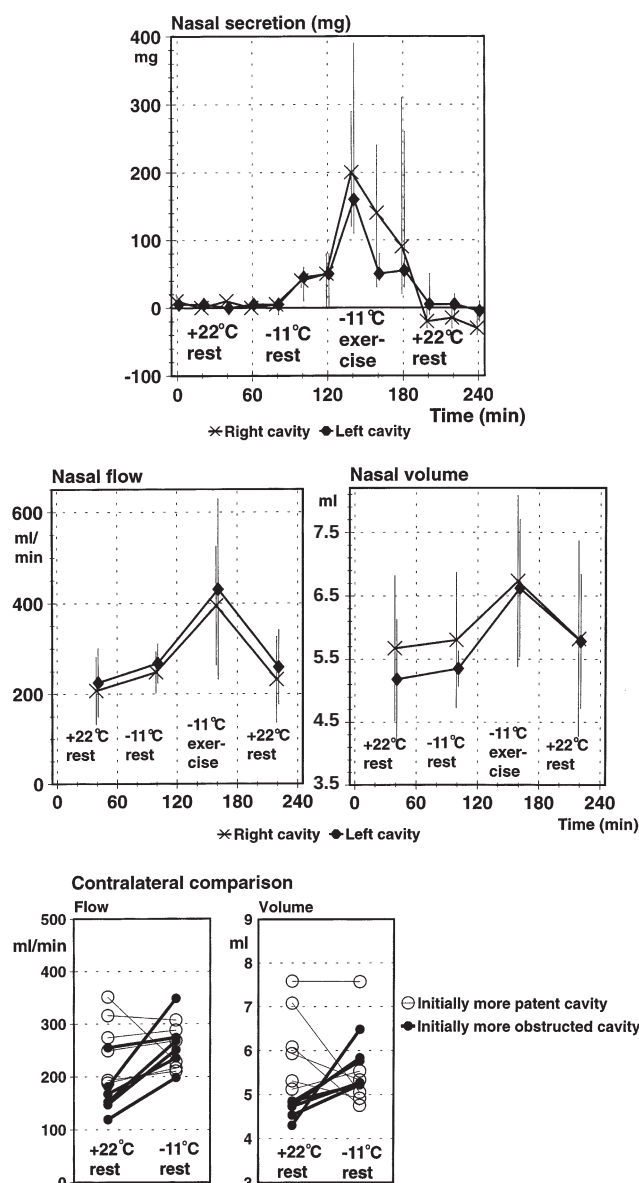


Figure 1. Nasal secretion response and changes in objective patency parameters after exposure to cold and skiing exercise.