
Case report

Nonoccupationally induced allergy to monkey

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Background: A previous study described 2 patients with occupationally induced asthma caused by exposure to monkey. We describe a patient who developed asthma, rhinitis, and conjunctivitis owing to sensitivity to a pet monkey. The patient had a long history of upper and lower allergic airway disease. When 2 pet macaque monkeys were introduced into the home, the patient noticed increasing coughing, wheezing, and shortness of breath. During a particularly long exposure to the monkey in a car, the patient developed severe wheezing, rhinitis, and conjunctival edema.

Objective: To demonstrate that a nonoccupational exposure to monkey can lead to allergic sensitivity.

Methods: Commercially obtained macaque monkey hair was defatted in acetone and extracted. A total of 5 $\mu\text{g/mL}$ of extract protein was adsorbed onto microtiter plates, and an alkaline phosphatase enzyme-linked immunosorbent assay was performed on serum samples from 4 individuals: 2 controls, 1 individual with suspected sensitivity, and the patient. A positive result was an optical density greater than twice that of the control.

Results: The patient's serum had an optical density greater than twice that of the control. The antibody levels of 2 control subjects—the patient's wife and one of the investigators—were elevated but not positive. The investigator had manifested asthma after exposure.

Conclusions: This is the first report of sensitivity to monkey occurring in a nonoccupational setting.

Ann Allergy Asthma Immunol. 2004;93:402–403.

INTRODUCTION

Sensitivity to monkey consisting of asthma symptoms has been previously described in a psychologist and a research assistant heavily exposed to monkeys in a laboratory setting.¹ We describe a patient who developed rhinitis and asthma after intense exposure to the family pet, a macaque monkey. The Saint Louis University Institutional Review Board approved the study.

CASE REPORT

The patient is a 49-year-old man with a 20-year history of upper and lower airway allergic disease. He had positive skin test reactions to dog, cat, and house dust mite and was therapeutically administered a nasal steroid, an antihistamine, an inhaled corticosteroid, and an inhaled long-acting β_2 -agonist. He had worsening of his symptoms while married to a woman who owned several cats. After his divorce and removal of the cats, his symptoms improved, until 4 years ago when he married a woman who kept 2 pet macaque monkeys. He developed upper and lower respiratory tract symptoms on a long-term basis. When 1 pet monkey was permanently removed from the home he improved somewhat, until a 30-minute exposure in a closed car on the way to the veterinarian. At that time, the patient experienced severe wheezing, rhinitis, and conjunctival edema.

His past medical history was significant only for allergic rhinitis and asthma. He was taking no additional prescriptions or over-the-counter medications. He denied any history of urticaria or drug, food, or latex sensitivity. His family history is significant for a son with allergic rhinitis and asthma. His social history revealed no tobacco or alcohol use. He worked as a truck driver. Physical examination revealed a swollen, pale nasal mucosa with no evidence of polyps. His lungs were clear on auscultation, without evidence of wheezing or rhonchi. Pulmonary function testing revealed a forced expiratory volume in 1 second of 106% of normal and a forced expiratory flow between 25% and 75% of 97% of normal. The remainder of his physical examination findings were within normal limits.

A home visit revealed a clean house with carpeted floors and an open cage in the living room where the monkey slept. The monkey was frequently out of its cage and had free range of the house. It was recommended that the patient keep the monkey out of the bedroom to minimize his contact with it. Interestingly, one of the investigators with known sensitivity to multiple animals required albuterol therapy after having contact with the monkey during the home visit.

METHODS

Because of the fear of simian virus, we elected not to skin test the patient with antigens from the monkey. Instead, an enzyme-linked immunosorbent assay (ELISA) was developed to demonstrate IgE to the monkey. Two grams of hair was obtained from the pet monkey during the home visit. After a protein assay revealed inadequate protein in an extract of the sample, 45 g of monkey hair was commercially obtained

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Supported by the Clement J. Sullivan Research Fund.

Received for publication January 14, 2004.

Accepted for publication in revised form July 9, 2004.

(Buckshire Corp, Perkasi, PA). The hair was defatted twice in acetone and then dried at room temperature. The hair was then soaked at 4° C in phosphate-buffered saline for 3 days. The sample was then filtered and lyophilized. The extract was reconstituted, dialyzed against water for 24 hours, lyophilized again, and reconstituted to 2,286 µg/mL of protein in water. An alkaline phosphatase ELISA was then performed.

Serum samples were obtained from 4 individuals: the patient, a nonsensitive exposed individual (the patient's wife), an atopic nonexposed subject (one of the investigators), and a nonatopic nonexposed control. All samples were run in triplicate, with washing and dilutions performed in phosphate-buffered saline/Tween 20. Five micrograms per milliliter of protein was adsorbed on Nunc-Immuno plates (Fisher Scientific, Pittsburgh, PA) for 3 hours at room temperature, washed, and then blocked for 1 hour with 3% bovine serum albumin at room temperature. The plates were washed, and unknown serum samples at 1:25, 1:50, and 1:100 dilutions were added. The plates were incubated overnight at 4° C and then washed. Goat anti-human IgE (1:1,000) (ICN Biomedicals, Irvine, CA) was added. The plates were incubated for 2 hours and then washed. Alkaline phosphatase-conjugated rabbit anti-goat IgG (1:1,000) (ICN Biomedicals) was added and incubated for 2 hours. After the plates were again washed, *p*-nitrophenyl phosphate in diethanolamine buffer was added. Optical density (OD) was read at 405 nm after 30 minutes. The sample was considered positive if the ratio of patient or subject OD divided by control OD was greater than or equal to 2.

RESULTS

The patient's OD units were 2.1 times those of the nonatopic control, indicating anti-monkey IgE (Table 1). Two other subjects are also shown to have elevated anti-monkey IgE levels compared with the control: a known atopic individual (one of the investigators) and an exposed, nonsensitive person (the patient's wife).

DISCUSSION

Because there is a close relationship between humans and monkeys, both being members of the primate family, there is a question as to whether allergy to monkey could develop in humans. There is 1 report¹ in the literature of allergy to monkey occurring as a result of an occupational exposure in a university psychologist and a research assistant who studied monkeys. These patients developed asthma associated with the workplace and were found to have positive skin test reactions to an extract of monkey hair.

We believe that this is the first case of nonoccupational sensitization and allergy to monkey in humans. Because of the fear of simian virus, we did not skin test our patient with monkey antigen and instead developed an *in vitro* test (ELISA) for detecting IgE anti-monkey antibody. The results demonstrated a significant elevation of IgE anti-monkey antibody levels in our patient. We hope to identify the particular monkey allergens in future studies. The patient's wife

Table 1. Optical Densities (ODs) of ELISA Measuring IgE Anti-Monkey Antibodies in Serum Samples from 4 Individuals

	OD _{405 nm}	Ratio*
Patient	1.108	2.10
Sensitive subject	0.970	1.85
Exposed subject	0.965	1.84
Nonatopic control	0.524	...

Abbreviation: ELISA, enzyme-linked immunosorbent assay.

* Patient or subject OD divided by nonatopic control OD. A ratio of 2 or greater was considered positive.

and an atopic investigator also had elevated IgE anti-monkey antibody levels, which were less than those of the patient but more than those of a control. Interestingly, the atopic investigator known to be sensitive to multiple animals developed mild bronchospasm in the presence of the monkey, whereas the nonatopic wife developed no symptoms.

The possibility of fungal or house dust mite contaminants in the hair producing a positive ELISA result is very remote. The patient is not sensitive to fungi, and the minute amount of house dust mite antigen in the extract would not be enough to produce a positive result. Preparations of commercial epithelial extracts are obtained from unwashed hair and epithelia.

The patient's known animal sensitivities plus the clinical improvement with the removal of 1 monkey would indicate that sensitivity to monkey allergens, as determined by the positive ELISA result, was the cause of his symptoms.

The United States has a vigorous trade in pet monkeys. Although the importation of monkeys for pet trade was banned in 1975, thousands of monkeys are being sold annually, and their popularity is increasing. It is impossible to provide exact statistics on the trade of pet monkeys. The US government does not maintain a database because the trade is not fully monitored by any single government agency. However, based on the number of primates held by members of the Simian Society of America and the number reported to states that require registering, it is estimated that there are 11,000 to 15,000 nonhuman primates in private sector possession.² It is the feeling of experts in the field that primates are wild animals and do not belong in peoples' homes. The possibility of developing allergic sensitivity is yet another reason to keep primates out of the home environment.

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