

Anaphylaxis after mouse bite

Katharina Leng, Katharina Wiedemeyer, Martin Hartmann
Department of Dermatology, University of Heidelberg, Germany

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Summary

Allergic sensitivity to laboratory animals is an occupational risk for all workers in contact with small animals. In rare cases, life-threatening reactions can occur. We describe a laboratory worker who after six years contact with mice developed an anaphylactic reaction after a mouse bite. Testing revealed positive reactions to all components of the mouse. The greatest risk for development of allergy is in the first three years and is usually an immediate hypersensitivity to inhalant allergens.

Keywords

anaphylaxis – mouse bite – laboratory animal – occupational disease

Introduction

Sensitivity to laboratory animals is an occupational risk for all workers in contact with small animals. In most cases symptoms are caused by airborne allergens from the animals and manifest as rhinoconjunctivitis and asthma. In rare cases acute anaphylactic reactions with angioedema and circulatory collapse can occur and be life-threatening. We describe a laboratory worker who developed an anaphylactic reaction after a mouse bite.

History

A 40-year-old woman worked as a laboratory worker with mice and rats. She worked with rats 3x/week for 10 years. With few exceptions gloves were worn during animal contact. No allergic signs or symptoms were present initially and several rat bites caused no difficulties. She did not work in animal holding facility and had no contact with mice during this time period. The Erlangen atopy score of the patient was 2 points (elevated IgE and positive Phadiatop); thus an atopic skin diathesis was not present. The patient did not have pets or contact to animals outside of the workplace. She had no serious medical problems and was on no medications. After 10 years of lack of contact to animals she was assigned to a project involving mice. Here, too, direct animal contact occurred several times weekly

generally with gloves. The animals were taken from their cages in order to perform surgical procedures. There was no contact with litter or food. After two years the patient first observed rhinoconjunctivitis with burning and itching eyes and a runny nose. No diagnostics or therapy was undertaken, as symptoms were tolerable. Improvement was observed on weekends and during vacation. After three further years of work with mice she was bitten without developing allergic symptoms.

One year later (after a total of six years of work) she was bitten again by a mouse through the glove in the left index finger. After 15 minutes visible swelling developed. Subsequently her lips and then her entire face became swollen, while she was warm and dizzy. Hypotension (90/50 mmHg) and bradycardia (34/min) as well as nausea and vomiting developed. The swelling spread to the entire index finger.

Suspected diagnosis: anaphylactic reaction severity grade III (according to Ring and Messmer 1977) following a mouse bite.

Therapy

The patient was admitted as an inpatient and observed for 48 hours. Symptoms were significantly diminished after 250 mg prednisolone intravenously, 1 ampule clemastine and 1 ampule ranitidine. Further 1 ampule atropine i. v. was administered. Symptoms disappeared within 24 hours.

Test results

Tests were performed 2.5 months after the bite. Prick testing (CAP-FEIA with test substances from Allergopharm®) resulted in a strongly positive reaction to rat epithelia (wheal up to 10 mm) and an even stronger one to mouse epithelia (wheal > 10 mm) after 20 min respectively. House dust mites and foodstuff mites as well as cat, dog, guinea pig and horse epithelia were negative. No positive immediate reactions towards latex allergens were observed.

Total IgE (test kit of Phadia AB®) was 356 kU/l, total mast cell tryptase as a marker of mast cell activation was in the lower normal range with 6.96 µg/ml. Specific IgE towards serum, epithelia and urine of mice and rats was strongly elevated (Table 1). Specific IgE towards cat and dog epithelia was slightly elevated

Table 1: Laboratory test results (specific IgE in kilo Units/liter).

Animal	Material	Specific IgE [kU/l]	CAP class
Mouse	Serum	7.98	3
	Epithelia	17.60	4
	Urine	26.00	4
Rat	Serum	5.34	3
	Epithelia	4.15	3
	Urine	24.50	4

Table 2: Laboratory animal allergens (MW = molecular weight in kilodalton).

Animal	Allergen	MW (kd)	Present in
Mouse (<i>Mus musculus</i>)	Mus m 1 (prealbumin)	19	Hair, urine, scales
	Mus m 2 (glycoprotein)	16	Hair, scales
	Albumin		Serum
Rat (<i>Rattus norvegicus</i>)	Rat n 1A/1B (α_{2u} -globulin)	16–21	Hair, saliva, urine, scales
	Albumin		Serum

(CAP class 2 and 1 respectively). Specific IgE towards latex as well as house dust mites and foodstuff mites were negative (CAP class 0). No specific IgE was found towards tree, grass and weed pollens. Due to lack of clinical symptoms, skin testing with these allergens was not done. Animal food and litter was not tested, as there was no contact to these. Native materials were not used.

Diagnosis

Allergic rhinoconjunctivitis in type I sensitization towards rat and mouse epithelia. Anaphylactic reaction grade III after a mouse bite in type I sensitization towards mouse epithelia, urine, and serum.

Discussion

The risk of developing a sensitization during the first three years of working with laboratory animals (rodents) depends mainly on three factors: the predisposition of the individual (atopy status, especially an already existing allergy to animal epithelia), the allergenic

potential of the allergen and the intensity of exposition. Reports of occupational rodent allergy vary very widely in the literature, rates of 10–32 % are reported [1, 7]. Symptoms range from mild rhinitis and conjunctivitis to (contact) urticaria to asthma and angioedema [5]. Most laboratory animal allergies are towards mice and rats, not because they are more potent allergens, but because they are used most often. To date 3 mouse and 2 rat allergens have been identified [7] (Table 2). In the mouse they are Mus m 1, also known as MUP (major urinary protein), a prealbumin that is dependent on testosterone, Mus m 2 and albumin. In the rat Rat n 1A and Rat n 1B have been identified, both variants of α_{2u} -globulin, but rat albumin also has strong allergenic potency [8]. The allergens differ in their molecular weight and their occurrence. Rat urine contains the most important allergen in rat allergy [1]. The sensitization phase in our patient preises the question as to cross-reactivity between rat and mouse. A cross-reaction between Mus m 1 and Rat n 1 is possi-

ble, as the lipocalins display great correspondence due to the crystal structure. After working 10 years with rats and 10 years of lack of contact, the patient worked with mice for 2 years before symptoms of rhinoconjunctivitis appeared for the first time and a further 4 years until a systemic reaction occurred. This long latent phase is unusual as laboratory animal allergy is a type I immune reaction mainly elicited by inhaled allergens, more rarely by direct skin contact. The path of inoculation plays an important role here, as a disrupted skin barrier allows the penetration of greater amounts of antigen including those in saliva which were not tested here. IgE-mediated allergy is strictly speaking an indication for specific immune therapy, first to prevent a shift to bronchial hypersensitivity or asthma and to prevent or at least minimize systemic reactions. Anaphylactic reactions without coexisting asthma have only been reported in individual cases [2, 3]. For treating occupational diseases few possibilities exist; besides avoiding contact (which would mean a change of job for our patient who has worked for years with animals as a laboratory worker), hyposensitization would be a possibility. In animal dander allergy without possibility of avoidance, hyposensitization is only reservedly done; only few uncontrolled studies exist [6]. In this special case the questions of an occupational disease and the possibility of preserving the job of a sensitized worker in an animal laboratory arise. Total avoidance of the allergen is difficult to realize. Up to now the patient has experienced only mild rhinoconjunctivitis, but it cannot be predicted if a renewed anaphylactic reaction depends only on the path of exposition (inoculation or respiratory inhalation) or if what is termed an Etagenwechsel in German (“change of level”) with a shift from rhinoconjunctivitis to bronchial asthma with later systemic reaction will occur. Specific immune therapies are available for rat and mouse allergies. In a published case of rat allergy in a laboratory worker hyposensitization was successful. Specific IgE sank from 9.52 kU/l to 0.51 kU/l (CAP class 1) and the patient had no more symptoms. This patient had only isolated IgE towards rat epithelium and no specific IgE towards mouse

epithelium [4]. It is problematic that our patient has specific IgE towards mice and rats. As cross-sensitivity between these two allergens is questionable, hyposensitization towards rats and mice would be necessary.

As, similar to cat epithelia, the allergens persist for a long time in the room or on objects coming from the cage, the danger of a renewed anaphylactic shock exists for the patient [9]. A dermatologist's report to the Accident Prevention and Insurance Association was made. The issue of occupational retraining must be addressed if a workplace change is not possible. An occupational skin disease must be assumed, as a normal atopy score, lack of clinical symptoms in private life and lack of history of pets suggest with great probability that the sensitization was caused occupationally. Early recognition of such laboratory animal allergies and preventive measures can prevent such a development in most cases [10]. <<<

Conflict of interest

None.

Correspondence to

Dr. K. Leng

Department of Dermatology, University of Heidelberg

Voßstr. 2

D-69115 Heidelberg, Germany

Tel.: +49-62 21-56-39 54 0

Fax: +49-62 21-56-46 22

E-mail:

Katharina.Leng@med.uni-heidelberg.de

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