

BRIEF REPORT

Occupational exposure to laboratory animals causing a severe exacerbation of atopic eczema

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ABSTRACT

A 24-year-old man with a long history of severe atopic eczema presented with a marked exacerbation requiring hospital admission. It emerged that his occupation as an animal house technician required him to work closely with laboratory animals, particularly mice and rats. Radioallergosorbent tests to mice allergens were markedly elevated. Avoidance of animal work, in conjunction with medical treatment, resulted in a marked improvement of his eczema.

Key words: allergy, contact dermatitis, contact urticaria, exacerbation, flare, irritation, mice, occupation, skin, work.

INTRODUCTION

Laboratory animal allergy is a well recognized condition, affecting individuals who have close contact with laboratory animals such as mice and rats. Studies have estimated that 10 to 23% of exposed workers report symptoms of allergy.¹ The most common symptoms are allergic conjunctivitis and allergic rhinitis.² Sensitisation generally occurs within the first 3 years of employment.⁵ Risk factors include an atopic background and high levels of allergen exposure. The allergens involved are usually mice and rat urinary proteins of low molecular weight.

CASE REPORT

A 24-year-old-man weighing 68 kg was admitted to hospital with a severe flare of his long standing atopic eczema. He also suffered from asthma and hay fever and had several previous hospital admissions for severe eczema. He had

been employed as a university animal house technician for the previous 3 years which had involved close contact with laboratory animals. Previous treatments included UV therapy, administered in his teen years prior to commencing employment, to which he had a good response. During flares, he also underwent multiple courses of oral antibiotics with weaning doses of oral prednisolone commencing at 25 mg and decreasing over several weeks. A year after commencing employment he was commenced on azathioprine because of worsening eczema. Although he initially responded to a dose of 100 mg daily, the eczema gradually worsened. After 2 years, azathioprine was discontinued and cyclosporin was initiated at 300 mg daily (Table 1). Nevertheless, his eczema had remained under only fair control, despite this aggressive medical therapy. There had been no significant deterioration in his asthma and hay fever.

On examination he was erythrodermic with evidence of multiple excoriations and secondary skin infection. He was admitted to hospital for wet dressings, prednisolone and oral antibiotics. Over the course of a week he demonstrated dramatic improvement, even more so than one would normally expect. This suggested the presence of an environmental allergen.

On further questioning regarding his occupational exposures, he noted that he was exposed to mice, rats, sheep and rabbits on a daily basis. Radioallergosorbent testing (RAST) demonstrated a strong reaction to mouse urine and a moderate reaction to mouse epithelium. The patient's serum IgE at the time was markedly elevated at 2821 (reference range 0–100) and eosinophil count was 2.69 (reference range 0.0–0.5 × 10⁹/L). Interestingly his eosinophil count was closely related to the changes of IgE levels and was a useful marker in monitoring his response to treatment (Table 2).

After discharge from hospital, the patient remained off work while a workers' compensation claim was submitted, this was successful. Eventually he was retrained and resumed work for the same employer in an administrative role. Since avoiding contact with laboratory animals his eczema has improved significantly, although he still

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Abbreviation:

RAST	Radioallergosorbent testing
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Table 1 Physical and systemic treatment regimens and response in our patient

Year	Treatment	Response
1997–2000	UV therapy	Good
2000	Intermittent prednisolone; weaning from 25 mg over 6 weeks	Good, but potential cumulative side effects from prednisolone
2001	Intermittent prednisolone, weaning from 25 mg over 6 weeks	Generally worse, azathioprine commenced as steroid sparing agent
2002	Commenced on Azathioprine 100 mg daily	Fair control
2003 (year of hospital admission and animal testing)	Azathioprine 100 mg daily	Poor control with occupational exposure still present
	Intermittent prednisolone, weaning from 25 mg over 6 weeks	Improvement seen once occupational allergy identified and avoided
2004	Cyclosporin 300 mg daily	Maintained adequate control with avoidance of occupational exposure
	Cyclosporin 300 mg daily	
2005	Cyclosporin reduced to 200 mg daily	Good
2006	Cyclosporin reduced to 100 mg daily	Good
2007	Cyclosporin ceased	Switched back to azathioprine to reduce long term toxicity of cyclosporin
	Azathioprine 100 mg daily	

Table 2 IgE levels relating to Eosinophils

	IgE level (0–100)	Eosinophils (0.0–0.5 × 10 ⁹)
October 1997	10	0.10
February 2003	410	1.74
August 2004	2821	2.69
November 2006	1302	0.87

requires immunosuppression with azathioprine. Patch tests were also performed, these showed a positive reaction to nickel. He had also experienced an exacerbation of his eczema at the site of his belt buckle.

The final diagnoses made with respect to his skin condition were: (i) contact urticaria to mice, of occupational relevance; (ii) atopic eczema; and (iii) allergic contact dermatitis to nickel, relevant to belt buckle.

DISCUSSION

Protein contact dermatitis was first described in 1976 by Hjörth and Roed-Petersen.⁴ Skin exposure to proteins may initially cause contact urticaria, which may be short-lived, but repeated exposures may induce an eczematous response. Occupational contact urticaria in Australia was recently reviewed by our group.⁵

Laboratory animal allergy is well documented, and is more likely to affect individuals with a history of atopy and a high level of animal allergen exposure.⁶ Reduction of allergy can be achieved by introduction of work practices and engineering controls designed to reduce potential exposures to animals.⁷ Such methods could consist of

environmental control, regular housekeeping routines and, finally, personal protective equipment in order to minimise exposure in a clean environment.^{7,8}

We report a case of a man with known and persisting severe atopic eczema who had become resistant to medical treatment. Recognition of his occupational exposures was the key to improving his clinical course. Although these events occurred some years ago, we believe that his case is instructive. It is important for dermatologists to be aware of the role of potential occupational allergens such as laboratory animals, even though these may normally be associated with inhalant allergy, in order to successfully improve patient outcomes.

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