

Sudden Asphyxic Asthma Following Exposure to High Concentrations of Dermatophagoides Group I Allergen

Paul J. Hannaway, M.D.

The duration of relapsing asthma that precedes a near-fatal or fatal asthma episode is often prolonged.¹⁻³ However, a subgroup of patients, who are often younger atopic asthmatics, can experience unexpected respiratory failure or what is now called sudden asphyxic asthma.^{4,5} Assumed risk factors for both gradual and sudden-onset catastrophic asthma include: increased bronchial hyperactivity,⁶ wide fluctuations in pulmonary function tests,^{7,8} corticosteroid withdrawal,⁹ improper use of β agonist drugs,¹⁰ underestimation of the disease severity, aberrant psychosocial factors,¹¹ and a history of seizures or life-threatening asthma.^{12,13} Exposure to the aeroallergen, *A. alternaria*, has recently been identified as an additional risk factor for acute respiratory arrest in children and young adults with mold-sensitive asthma.¹⁴

Whereas the risk factor posed by the house dust mite for emergency asthma care and the development of chronic asthma is well documented,^{15,16} there are no published reports coupling direct exposure to high concentrations of house dust mites to sudden asphyxic or near fatal asthma. The following case report describes a young adolescent male with chronic, IgE-mediated, dust mite-sensitive asthma who developed sudden asphyxic asthma and acute respiratory failure following prolonged exposure to very high concentrations of *Dermatophagoides* group I allergens.

CASE REPORT

S.H. is a 15-year-old male with an 11-year history of chronic, IgE-mediated asthma. Pertinent past his-

tory includes two short term hospitalizations at age 9 and 11 for viral syndrome induced asthma. Initial allergy evaluation at age 11 yielded evidence of reversible airway obstruction, a serum IgE of 407 IU/ml, and total eosinophils of 437/mm³.

Prick tests were moderately positive (5 to 10-mm wheals) to house dust, cat antigen and trees, grasses, and weeds common to Eastern New England. The patient was then placed on a program of daily anti-inflammatory therapy, (inhaled beclomethasone), daily theophylline, prn inhaled β agonists and appropriate environmental controls.

On May 29, 1987, while riding on the Monorail in Disneyland S.H. suddenly developed acute abdominal cramps, nausea, and vomiting followed by facial edema and upper airway obstruction requiring CPR and on-site endotracheal intubation by local EMTs. Upon admission to a local hospital he had facial edema, upper airway obstruction, a pH of 7.07, and a pCO₂ of 67. He responded promptly to critical care therapy and was extubated in 12 hours. Exhaustive analysis of events leading to this acute anaphylactic (nonasthma), event and respiratory arrest revealed that he had ingested five baby aspirins 4 hours before this episode. The patient was then labeled as a high risk, aspirin-sensitive asthmatic and a long-term asthma crisis plan, including the need for compliance with asthma medications, Epi-Pen availability, and prompt use of oral steroids in relapsing asthma was implemented.

Over the next 5 years (1987-1992), he averaged three to four annual asthma relapses that were usually triggered by upper respiratory infections in the fall and winter months. He required one short-term hospitalization in March 1989 for apparent viral-syndrome induced asthma. During these relapses, he exhibited signs

Reprint requests to Dr. Paul J. Hannaway, 114 Highland Ave, Salem, MA 01970

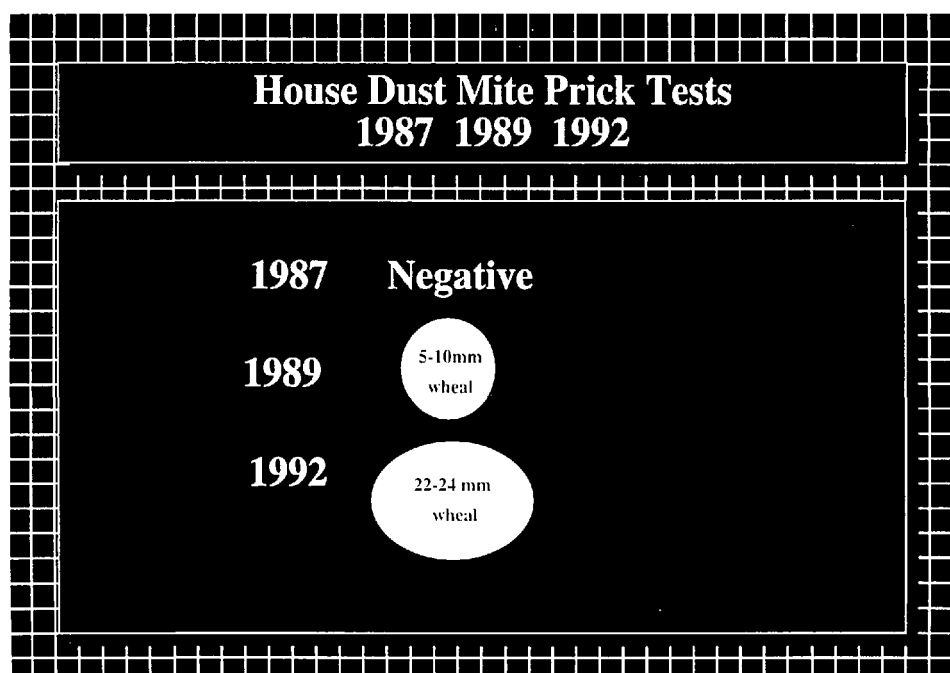


Figure 1. House Dust Mite Prick Tests 1987, 1989, and 1992

of extremely labile asthma as his FEV₁ would decrease to 30–55% of predicted levels during an asthma relapse and promptly return to 90–100% of predicted levels with rescue bronchodilators and oral prednisone.

On July 4, 1992, while visiting the home of a friend for a “sleep over,” he spent approximately 5 hours in a basement family room watching TV. In retrospect this carpeted room in a home with a stone foundation was described as being very damp and dusty. Shortly after retiring to an upstairs bedroom and attempting to sleep on a carpeted floor he experienced an acute asthma attack unresponsive to inhaled albuterol and oral theophylline. Within minutes after developing progressively severe shortness of breath he requested transport to a local hospital. En-route to the hospital he became unresponsive and upon arrival at the emergency room he was apneic and cyanotic, with a pulse rate of 20, a pH of 6.96, and a pCO₂ of 96. He responded quickly to intubation and appropriate critical care. ABGs returned to normal in approximately 12 hours and he was extubated in 24 hours. On office follow up 1 week later he was asymptomatic with normal PFTs and chest exam.

Retrospective analysis of events leading up to this episode of sudden asphyxic asthma was as follows: Patient had been relatively symptom free for several weeks and had been complying with a program of inhaled flunisolide, daily theophylline, and prn inhaled β -agonists. He denied having any asthma symptoms whatsoever until minutes preceding his respiratory arrest. He had been followed at regular intervals for several years and theophylline levels were consistently

therapeutic. There was no history of ASA ingestion, (salicylate levels not performed), illicit drug use, undue animal exposure, passive tobacco smoke exposure, (patient a nonsmoker), overuse of β agonists or URI type symptoms.

Prick tests to common inhalants and a few food antigens 1 month after his respiratory arrest showed increasing levels of skin reactivity. Specific prick tests worthy of note were house-dust mite antigens, *D. farinae* = 24-mm wheal and *D. pteronyssinus* = 22-mm wheal. RAST levels to these mite allergens revealed class V/VI sensitivity (Smith-Kline Labs). Tree, grass and weed pollens, and dog and cat antigens demonstrated wheal reactions approximating 10–15 mm in diameter. Food allergens, including MSG and bisulfite tests, were negative. Chart review revealed the patient’s prick test to *Der f* 1 antigen as negative in 1987 and moderately positive (5–10 mm wheal) to *Der f* 1 and *Der p* 1 in 1989. See Figure 1.

In view of this striking increase in skin sensitivity to house-dust mite antigen over a period of 3 years, the home the patient had been visiting and his own home was subjected to indoor allergen analysis. Dust samples were collected from both homes with the ALK Indoor Allergen Analysis Kit and analyzed at Vespa Laboratories.¹⁷ The dust sample from a sofa and carpet in his friend’s basement family room yielded very high levels of house-dust mite allergen—72u/gm of Group I allergen (42.6u/gm *Der p* 1 and 29.6u/gm *Der f* 1). The upstairs bedroom carpet had a level of 11.8 u/gm of Group I allergen (4.5u/gm *Der p* 1 and 7.3u/gm *Der f* 1). The following levels were recorded in the patient’s

home; family room-Group I allergen = 4.6u/gm (*Der f* I), bedroom mattress-7.7 u/gm (*Der f* I), and bedroom carpet-25.2u/gm (*Der f* I). See Figure 2.

Thus in the 5 hours immediately preceding sudden asphyxic asthma and acute respiratory arrest this atopic adolescent with strongly positive skin and RAST tests to house-dust mite allergen was exposed to a 15-fold higher concentration of *Dermatophagoides* group I allergen in his friend's family room than he would have been exposed to in his own family's TV room.

Following this bout of sudden asphyxic asthma the patient was placed on high dose flunisolide (3000u/gm per day), daily theophylline, prn inhaled β agonists, and environmental controls were thoroughly reviewed. Immunotherapy was briefly considered but deemed to be too risky.

DISCUSSION

Several investigators have described a subgroup of patients with stable, mild to moderate asthma who, despite the absence of adverse psychosocial factors, seizures, oral steroid-dependency, or other risk factors for near fatal or fatal asthma, develop "out of a clear blue sky" sudden asphyxic asthma.¹⁻⁵ There are three patterns of onset (and resolution) of this form of asthma: (1) rapid (minutes to less than 3 hours), (2) delayed (a few hours), and (3) prolonged (days to weeks).^{1,14,18-20} In one report, nine of 10 patients with rapid onset (<3 hours) and resolution (<13 hours) of sudden asphyxic asthma were younger male asthmatics whose episodes were triggered by allergens or emotions.¹ Slow recovery was more commonly observed in older women with a prior episode of acute respiratory failure in whom relapses were triggered by a respiratory infection. In another series of 11 patients (predominately females 9:2) patients experienced sudden and unexpected respiratory arrest within 90 minutes after worsening of symptoms.¹⁴

An increasing incidence of sudden asphyxic asthma may be the reason near fatal or fatal asthma is now more likely to occur outside of hospital.^{21,22} A recent Australian study found that only 11% (13/116) of asthma deaths occurred in-hospital and the terminal episode was sudden in onset (within minutes) in 58% of decedents.²³ One-third of these 116 asthma deaths occurred in patients with mild or moderate asthma.

In a Cardiff survey 25% of deaths occurred within 30 minutes after the attack began and nearly two-thirds occurred within 8 hours.²⁴ A Massachusetts survey from 1974-1988 found a more than 2-fold increase in asthma mortality in the 1980s and a significant reversal in the site of asthma death.²⁵ From 1974-1978 two-thirds of asthma deaths in Massachusetts residents under age 50 occurred in hospital whereas after 1984, two-thirds of asthma decedents died at home or en route to hospital.

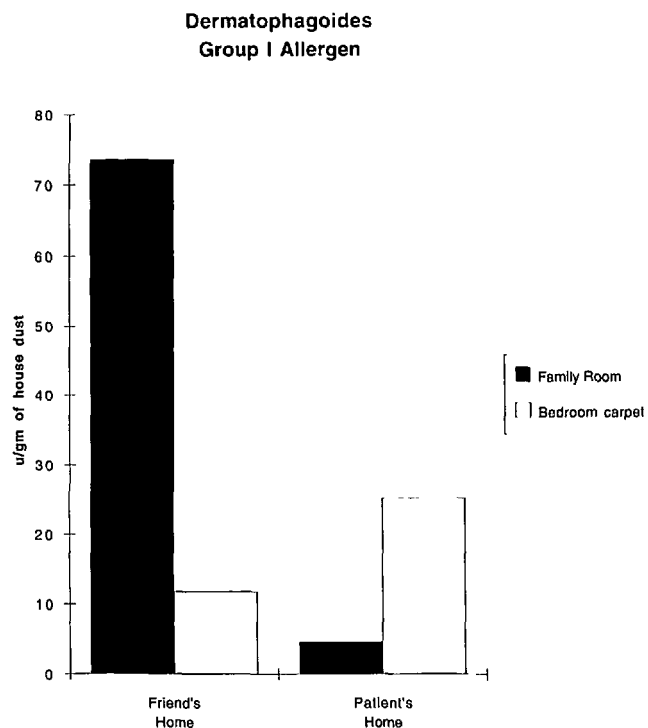


Figure 2. House-dust mite allergen levels as recorded from patient's home and friends home.

Young adults, especially the 10-14-year-old group, are more predisposed to sudden asphyxic asthma.²⁶ The reasons for this are unclear but increasing atopic sensitivity is one of several possibilities.^{27,28} The linkage between outdoor aeroallergens and near fatal or fatal asthma is mostly circumstantial, yet epidemiological studies on asthma morbidity and mortality imply that seasonal increases in mold and pollen exposure may be an important, but unrecognized trigger of near fatal or fatal asthma. Arnold reviewed 261 episodes of severe asthma requiring admission to Oxford teaching hospitals and found the majority of admissions were young atopic asthmatics who presented with rapidly (< than 1 hour) deteriorating asthma.²⁹ The British Thoracic Society compared atopic and nonatopic patients dying of asthma.³⁰ Those in the atopic group were more likely to experience a sudden asphyxic episode in the spring and fall as 12 of 18 patients who died suddenly did so in May-June and September-October. An increased incidence of asthma mortality was noted in May, June, and July in another series of 30 childhood asthma deaths.³¹

Weiss found a higher asthma death rate in the United States from June through August in 15 to 30-year-old asthmatics suggesting that outdoor aeroallergens may be responsible for this seasonal pattern of asthma mortality.³² Miller and Strunk reported a higher rate of

catastrophic asthma in May-June and September-October.³³ In one Swiss study outdoor aeroallergen exposure was presumed to be the likely trigger in 3 of 10 patients with sudden "out of a clear blue sky" asphyxic asthma.¹

A. alternaria aeroallergen has been strongly linked to catastrophic asthma in 11 patients who experienced 18 episodes of respiratory arrest, two of which were fatal, between 1980 and 1989 during the peak period of outdoor mold exposure.¹⁴ Other anecdotal reports note acute respiratory arrest after direct weed exposure³⁴ and attempts to remove an old carpet.³⁵ Platts-Mills has described several inner-city asthma fatalities that occurred in cockroach-infested homes.³⁶ Although studies have convincingly shown a strong correlation between emergency room visits for asthma and specific IgE-mediated sensitivity to house-dust mite, atmospheric pollens, and mold spores,³⁷⁻³⁹ published case-control data linking sudden asphyxic asthma to the house-dust mite is surprisingly scant.

This case report, which describes an adolescent male with significant dust mite allergy who developed sudden asphyxic asthma after prolonged exposure to high concentrations of *Dermatophagoides* group I allergen, suggests that inadvertent exposure to high concentrations of dust mite allergens should be added to the list of risk factors for sudden asphyxic or fatal asthma. Practitioners, particularly asthma specialists, who encounter such patients should retrospectively evaluate and quantitatively assay indoor environments in which near fatal or fatal asthma attacks occur. Over the past decade, investigators have focused on adverse psychosocial factors, bronchial hyperreactivity, noncompliance, and the potential of pharmacologic therapy as predisposing factors for the observed increase in asthma morbidity and mortality. Little attention has been paid to the potential role of indoor and outdoor aeroallergens.

The rise in asthma morbidity and mortality noted in most, if not all, developed countries in the 1980s has triggered scores of published reports on fatal or near fatal asthma. United States epidemiological studies have focused on asthma deaths and death rates by geographic locale, age, race, and sex of decedents. Unlike other developed nations, such as Canada, Great Britain, Scandinavia, Australia, and New Zealand, the United States has no established conduit which gathers and analyzes specific case control data such as that reported above. The puzzle posed by the striking increase in asthma morbidity and mortality in the United States will not be solved until some type of national registry is established. Such a registry could collect and analyze case control data to uncover the causes of near fatal or fatal asthma and determine if near-fatal or fatal asthma is being triggered by exposure to high concentrations of house-dust mite, molds, pollens, cockroach, or animal allergens.

PROLOGUE

In October 1992, after much prodding, the patient and his family moved to a newer home that permitted stricter environmental controls. Dust mite levels (Group I allergens) in his new bedroom in November 1992 were less than 0.2 $\mu\text{g}/\text{gm}$ per gram. The patient was followed at regular intervals (q 8 weeks) from November 92 to March 93 and was essentially symptom-free, i.e., no ER visits, good exercise tolerance, no school loss, and no need for prednisone. During the same period last year, (1991-1992), he required several emergency visits and four pulses of prednisone. In early March 1993, he was asymptomatic with normal PFTs. Inhaled flunisolide was lowered to 1500 $\mu\text{g}/\text{g}$ per day and he was continued on daily theophylline and prn inhaled β agonists.

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