



Short communication

Who's at risk of thunderstorm asthma? The ryegrass pollen *trifecta* and lessons learnt from the Melbourne thunderstorm epidemicJoy Lee^{a,c,*}, Caroline Kronborg^a, Robyn E. O'Hehir^{a,b}, Mark Hew^{a,c}^a Allergy, Asthma & Clinical Immunology, The Alfred Hospital, Melbourne, Australia^b Allergy, Immunology & Respiratory Medicine, Central Clinical School, Monash University, Melbourne, Australia^c School of Public Health & Preventive Medicine, Monash University, Melbourne, Australia

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ABSTRACT

The Melbourne thunderstorm asthma epidemic in November 2016 was unprecedented in scale and impact. We systematically reviewed our hospital's patients with thunderstorm asthma to identify key risk factors. Of 85 adult patients assessed, the majority (60%) had no prior diagnosis of asthma. However, allergic rhinitis during the grass pollen season was almost universal (99%), as were ryegrass pollen sensitization (100%) and exposure to the outdoor environment during the thunderstorm (94%). Airborne pollen levels on the thunderstorm day were extreme. We conclude that ryegrass pollen sensitization, clinical allergic rhinitis, and acute allergen exposure constitute a risk-factor '*trifecta*' for thunderstorm asthma.

1. Introduction

Thunderstorms have triggered sudden asthma epidemics throughout the world [1]. The evidence mostly suggests an allergic aetiology due to plant pollens and fungal spores [1]. Importantly, thunderstorm asthma can affect individuals *without* prior asthma [2]. This unpredictable element hinders effective targeting of protective measures for thunderstorm asthma.

On the 21st of November 2016 at 1700 h, a springtime thunderstorm struck Melbourne, Australia, and triggered the most devastating thunderstorm asthma epidemic to date [3]. In a city of nearly five million, ambulances and emergency departments were overwhelmed by 3500 presentations, with many times that number treated urgently in the community [4]. Thirty-five patients required intensive care admission, and nine deaths are under coronial investigation.

We systematically examined our cohort of patients at Alfred Health, Melbourne, to identify key susceptibility factors.

2. Methods

We reviewed records of all patients aged ≥ 16 years with symptoms suggestive of asthma (dyspnoea, respiratory distress, cough or wheeze) presenting to the emergency departments of Alfred Health between 1700 h 21st November, and 1700 h, 23rd November 2016. For patients

discharged from Emergency, we attempted repeated telephone contact to offer review at our specialist asthma and allergy clinic. All patients admitted with thunderstorm asthma were scheduled for review.

A detailed asthma history was obtained. On spirometry, an obstructive ventilatory defect was defined by an FEV₁/FVC ratio below the lower limit of normal (for age, weight and height). A significant bronchodilator response was defined as an increase in FEV₁ by $\geq 12\%$ AND ≥ 200 ml following bronchodilator.

We defined 'seasonal allergic rhinitis' as the presence of rhinitis symptoms during Melbourne's spring (September to November); 'perennial allergic rhinitis' as perennial symptoms at a similar intensity; and 'perennial rhinitis with seasonal exacerbation' as perennial symptoms with at least fifty percent worsening during Melbourne's spring.

Patients were designated atopic if they had at least one wheal ≥ 3 mm on skin prick to twenty aeroallergens (Stallergenes-Greer[®], Antony, France) or a serum allergen-specific IgE > 0.34 kUA/L (ImmunoCap[®] Abacus ALS, Brisbane, Australia) to at least one of: Ryegrass pollen, Bermuda grass pollen, *Alternaria* and *Cladosporium*. During the thunderstorm, Ryegrass was in pollination. *Cladosporium* and *Alternaria* fungal spores have previously been detected in the atmosphere during the season.

Patients were asked their location before and during the thunderstorm (between 1700 and 2000 h). The pollen count was obtained from 'Melbourne Pollen Count' (<http://www.melbournepollen.com.au/>)

Abbreviations: CI, Confidence Interval; FEV₁, Forced expiratory volume during the first second; FVC, Forced vital capacity; FER, Forced expiratory ratio; IgE, immunoglobulin E; SD, Standard deviation; SPT, skin prick test; RAST, Radioallergosorbent test

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Table 1
Baseline demographics.

Demographics	n = 85	
Age, mean (range, SD) years	40	16–81, 16
Gender, n (%) Male	51	60%
Smoking status, n (%)		
Never	53	62%
Ex-smoker	27	32%
Current	5	6%
Disposition from Emergency department		
Home	72	85%
Short stay unit or general ward	11	13%
Intensive care	2	2%
Asthma history		
No previous asthma	30	35%
Undiagnosed asthma	21	25%
Known asthma	34	40%
		n = 34
Current asthma	19	56%
Childhood asthma	11	32%
Previous thunderstorm asthma (November 2010)	4	12%
Allergic Rhinitis		
Symptoms during ryegrass pollen season (September–November)	84	99%
Seasonal allergic rhinitis only	75	88%
Perennial allergic rhinitis with seasonal exacerbation	6	7%
Perennial rhinitis without seasonal exacerbation	3	4%
Allergic rhinitis clinical severity		
Mild	26	31%
Moderate	42	50%
Severe	16	19%
Allergic Sensitization		
Serum (n = 68)		
Ryegrass	68	100%
Bermuda grass	63	94%
<i>Alternaria</i>	5	7%
<i>Cladosporium</i>	3	4%
Skin prick test (n = 75)		
Ryegrass	73	97%
Timothy grass	68	91%
Plantain grass	49	65%
Bahia grass	70	95%
Ragweed	49	66%
Cypress tree	35	47%
Wattle tree	33	44%
Birch tree	33	44%
Plane tree	27	36%
Cat dander	27	36%
Dog dander	11	15%
Horse dander	9	12%
House dust mite (<i>D. pteronyssinus</i>)	50	67%
<i>Alternaria</i>	9	12%
<i>Cladosporium</i>	4	5%
<i>Aspergillus</i>	6	7%
<i>Penicillium</i>	1	1%
<i>Trichophyton</i>	8	11%
Outdoors exposure during thunderstorm		
Exposed	80	94%
Physically outdoors	57	71%
Indoors with open windows	23	29%
Not exposed	5	6%

Abbreviations: n = number, SD = standard deviation, IgE = immunoglobulin E.

[index.php/forecast](#)).

Ethics approval was obtained (Alfred ref. 20/17). Data analysis was performed using SPSS version 22 (IBM, Armonk, NY). Categorical variables are presented as percentages (frequency) and continuous variables as mean values with standard deviation.

3. Results

From 1188 emergency records, 243 patients had asthma symptoms. Of these, 168/243 (68%) were contactable, but 37 declined appointments. Of 131 scheduled appointments, 15 patients cancelled and 29 failed to attend; 87 attended clinic between 22nd December 2016 and 1st June 2017, at which two were found not to have thunderstorm asthma. Eighty-five clinic patients with thunderstorm asthma are described in Table 1.

Of nineteen patients with known current asthma, nine had an inhaled corticosteroid preventer, four (45%) of whom reported poor adherence (usage < five days/week).

Spirometry—obtained out of the ryegrass pollen season—demonstrated airflow obstruction in only seventeen patients (20%), and a bronchodilator response in only nine (11%).

Eighty-four (99%) of 85 patients had allergic rhinitis symptoms spanning the ryegrass pollen season. Eighty-one (95%) of 85 patients had skin testing, serum specific IgE, or both performed. Of these, 100% were sensitized to ryegrass pollen. For ryegrass pollen, the mean skin prick diameter was 11 ± 6 mm and the serum specific IgE was 55 ± 34 kU/L (range 1–100kU/L). Total serum IgE was 459 ± 546 kU/L (range 24–2229kU/L).

Eighty (94%) of 85 patients reported outdoor exposure between 1700 and 2000 h on 21st November 2016. Airborne pollen levels were ‘extreme’ (102grains/m³) on the day.

4. Discussion

Ryegrass pollen sensitization, clinical allergic rhinitis, and acute allergen exposure are a risk factor *trifecta* for Melbourne thunderstorm asthma [5,6]. Our results have important implications for thunderstorm asthma prevention, not only in Melbourne but also for other regions across the globe where temperate ryegrass (*Lolium perenne*) is cultivated.

Firstly, we confirm that allergic rhinitis and ryegrass pollen sensitization clearly define the adult population at risk for thunderstorm asthma. In Melbourne, allergic rhinitis affects up to 17% of the population, and should now be considered a condition with potentially serious consequences [5,6]. Ryegrass pollen sensitization should be confirmed and appropriate treatment offered, which may include nasal corticosteroids and specific allergen immunotherapy [3].

Secondly, exposure to the outdoors is the critical trigger for thunderstorm asthma. It is hypothesised ryegrass pollen grains (> 35 µm in diameter) are ruptured by storm moisture into respirable 3 µm granules [7,8]. Thunderstorm downdrafts draw these granules to ground level mimicking aerosol challenges [9,10]. Early warning systems should be developed to alert allergic rhinitis patients to stay indoors with windows shut before the onset potential thunderstorm epidemics.

Our patients had a higher rate of allergic rhinitis (99%) than reported previously [7]. We are also the first to show universal (100%) ryegrass pollen sensitization among those tested with a combination of skin testing and serum specific IgE. In Melbourne, Bermuda sensitization often accompanies ryegrass sensitization, explaining its high prevalence. Fungal sensitization was not relevant for this event.

Our outdoor exposure rate (94%) exceeds that of a previous questionnaire-based case-control study (38%) [2], and may be explained by the more detailed history available during clinical consultation.

A prior diagnosis of asthma, or detectable airflow obstruction outside of the pollen season, was found in the minority. Although important, they are insensitive markers of risk. Asthma was underdiagnosed and undertreated in our cohort, exposing gaps in community asthma management [11].

By Australian rates, active smoking was under-represented in our cohort, and is probably not an important contributor to thunderstorm asthma.

Our study has the usual constraints of an uncontrolled cohort, and is

subject to recall bias for outdoors exposure history. Not all patients attended clinic, and four patients did not undergo allergen testing.

Sensitization, clinical allergic rhinitis, and outdoor exposure to ryegrass pollen are critical contributors to thunderstorm asthma risk, and each requires preventive public health measures. Further investigation should focus on identifying more specific markers of susceptibility within this at-risk group.

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Author statement

JL, ROH and MH conceived and designed the study. JL, CK, MH collected data. JL and MH interpreted the data and were responsible for the first draft to which all authors contributed. All authors approved the final version.

Competing interests

Mark Hew has undertaken contracted research for AstraZeneca, Sanofi, Novartis, & GlaxoSmithKline; delivered Educational talks for GlaxoSmithKline, AstraZeneca & Novartis; Participated on advisory boards/consultancies for AstraZeneca, GSK & Seqirus; for all of which his employer (Alfred Health) has been reimbursed.

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Alfred Health Research and Ethics department.

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