

Epidemic thunderstorm asthma



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Epidemic thunderstorm asthma has been reported about a dozen times in the past 35 years; the threshold for reporting seems to be an increase of at least five to ten times in asthma presentations to emergency departments over a short time.^{1,2} Most cases have occurred in Australia (especially in Melbourne) and in the UK; one has occurred in North America and two smaller events have occurred in continental Europe (Italy). Many individuals are affected by these events but do not seek acute medical care.^{3,4} The accepted mechanism is extreme allergen exposure, predominantly grass pollen, in susceptible allergic individuals. The rapidity of onset and the speed of recovery (compared with usual emergency department asthma presentations) suggest these cases probably represent allergen-induced and mainly bronchospastic early asthmatic responses, an otherwise unusual precipitant for asthma exacerbation that is severe enough to merit emergency department requirement.² The early asthmatic responses is dependent on the degree of allergy (allergen specific IgE assessed by prick skin test or serology); the magnitude of (untreated) airway hyper-responsiveness; and the dose of allergen.⁵ Because the early asthmatic response can occasionally be life threatening,⁶ and sometimes fatal as in the Melbourne epidemic in 2016, rapid access to life-saving medical care is also important.

The largest and most catastrophic epidemic thunderstorm asthma event occurred in Melbourne on Nov 21, 2016. In *The Lancet Planetary Health*, Francis Thien and colleagues⁷ report a multidisciplinary review of environmental, weather, and patient risk factors, as well as the response of, and effect on, the local health-care system. These data support the early asthmatic response hypothesis—a huge allergen dose, which is the major determinant differentiating thunderstorm asthma from seasonal allergic asthma at other times, was documented by extremely high rye grass pollen concentrations, much of which was ruptured, along with the meteorological conditions (ie, a high gust front, high humidity, rain, and a sharp drop in temperature) favouring release and dispersal of inhalable allergen-rich starch granules. The high prevalence (approaching 100%) and magnitude (mean prick skin test wheal size 10–11 mm) of grass pollen sensitivity in

this population has been reported previously.^{8,9} More than 70% of the patients had untreated airway hyper-responsiveness;⁷ previous asthma with no symptoms in the past year; asthma-like symptoms not diagnosed; or seasonal allergic rhinitis with no asthma symptoms. Many of the individuals with current asthma seemed to have been undertreated. Since even life-threatening early asthmatic responses should respond well to prompt treatment, it is probable that most, if not all, of the ten fatalities (five in-hospital from hypoxic encephalopathy and five out-of-hospital) might have survived with immediate access to treatment.

The unusual age distribution, mainly individuals aged 20–59 years rather than very young or old individuals, represents a reversal of the usual U-shaped curve, where there are more admissions of young and elderly people, and has been observed or can be inferred from previous studies.² The mechanism might relate partly to concentrations of allergen-specific IgE that are highest between ages 20–39 years,¹⁰ correlating with the age distribution of allergic rhinitis.⁷ Other factors could be relevant; eg, individuals of this age might be more likely to be outdoors, and potentially more active (both of which factors would increase allergen load). Additionally, issues around treatment and adherence might be relevant. Although most patients did not have doctor-diagnosed asthma, 100% of the 35 critically ill patients admitted to an ICU had a previous diagnosis of asthma, 66% of whom were not using inhaled corticosteroids. An unusual observation was the high prevalence of Asian or Indian ethnicity among the patients, including six of the ten deaths. This high prevalence most likely reflects the very high prevalence of atopy and associated rhinitis and asthma in immigrant populations;¹¹ but other factors such as access to medical care and adherence might also be important.

Data such as these might help to mitigate the severity of future epidemic thunderstorm asthma events which, based on history, are likely to recur particularly in Melbourne. The cornerstone must be communication and education (including but not limited to patients, physicians, and emergency health-care services) surrounding important aspects of these events. Early warning based on environmental

factors—mainly pollen levels and meteorology, can alert emergency health services as well as direct at-risk individuals to remain indoors with windows shut, a practice which seems to reduce exposure.^{3,8} The major emphasis, however, should be directed to medical management of individuals at-risk. Since all fatal and life-threatening asthma occurred in individuals who currently had asthma, most of whom were not using inhaled corticosteroids, the need for enhanced treatment and treatment adherence in this group must be emphasised. Identification and treatment of any asthma in patients with allergic rhinitis patients who have respiratory symptoms should prove beneficial. Treatment of grass pollen allergic rhinitis in those with or without respiratory symptoms, particularly with nasal corticosteroids, might beneficially affect the lower airways; however, grass pollen immunotherapy for allergic rhinitis will control or prevent allergic asthma¹² and should be an important consideration.

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I declare no competing interests. I thank Jacquie Bramley for assistance in preparation of this manuscript.

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- 1 Dabrera G, Murray V, Emberlin J, et al. Thunderstorm asthma: an overview of the evidence base and implications for public health advice. *QJM* 2013; **106**: 207–17.
- 2 Cockcroft DW, Davis BE, Blais CM. Thunderstorm asthma: an allergen-induced early asthmatic response. *Ann Allergy Asthma Immunol* 2018; **120**: 120–23.
- 3 Girgis ST, Marks GB, Downs SH, Kolbe A, Car GN, Paton R. Thunderstorm-associated asthma in an inland town in south-eastern Australia. Who is at risk? *Eur Respir J* 2000; **16**: 3–8.
- 4 Clayton-Chubb D, Con D, Rangamuwa K, Taylor D, Thien F, Wadhwa V. Thunderstorm asthma—revealing a hidden at-risk population. *Intern Med J* 2018; published March 23. DOI:10.1111/imj.13800.
- 5 Cockcroft DW, Ruffin RE, Frith PA, et al. Determinants of allergen-induced asthma: Dose of allergen, circulating IgE antibody concentration, and bronchial responsiveness to inhaled histamine. *Am Rev Respir Dis* 1979; **120**: 1053–58.
- 6 Cartier A, Malo JL, Dolovich J. Occupational asthma in nurses handling psyllium. *Clin Allergy* 1987; **17**: 1–6.
- 7 Thien F, Beggs PJ, Csutoros D, et al. The Melbourne epidemic thunderstorm asthma event 2016: an investigation of environmental triggers, effect on health services, and patient risk factors. *Lancet Planetary Health* 2018 **2**: e255–63.
- 8 Lee J, Kronborg C, O’Hehir RE, Hew M. Who’s at risk of thunderstorm asthma? The ryegrass pollen trifecta and lessons learnt from the Melbourne thunderstorm epidemic. *Respir Med* 2017; **132**: 146–48.
- 9 Sutherland MF, Portelli EL, Collins AL, Rahman MA, McDonald CF. Patients with thunderstorm asthma or severe asthma in Melbourne: a comparison. *Med J Aust* 2017; **207**: 434–35.
- 10 De Amici M, Ciprandi G. The age impact on serum total and allergen-specific IgE. *Allergy Asthma Immunol Res* 2013; **5**: 170–74.
- 11 Leung RC, Carlin JB, Burdon JG, Czarny D. Asthma, allergy and atopy in Asian immigrants in Melbourne. *Med J Aust* 1994; **161**: 418–25.
- 12 Morjaria JB, Caruso M, Emma R, Russo C, Polosa R. Treatment of allergic rhinitis as a strategy for preventing asthma. *Curr Allergy Asthma Rep* 2018; **18**: 23.