

Title: 2016 thunderstorm-asthma epidemic in Melbourne, Australia: an analysis of patient characteristics associated with hospitalization

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## ABSTRACT

**Rationale:** On the 21<sup>st</sup> of November 2016 in Australia, a major thunderstorm-asthma epidemic struck Melbourne with an unprecedented number of emergency presentations, hospital admissions and fatalities.

**Objectives:** We identified affected patients who presented to The Royal Melbourne Hospital, an adult tertiary centre in North-West Melbourne. We aimed to characterize individual patient factors associated with hospital admission and identify biomarkers in patient subgroups that are at risk of being severely affected by thunderstorm-asthma.

**Methods:** Cross-sectional, retrospective analysis of demographics of 240 patients presenting to The Royal Melbourne Hospital on 21<sup>st</sup> to 22<sup>nd</sup> November 2016 post thunderstorm-asthma event and clinical characteristics of 70 of those patients who subsequently attended an outpatient clinic review.

**Results:** Patients were generally young adults (mean age 35 years), with seasonal rhinitis (96%) and universally (100%) sensitised to ryegrass pollen. Forty-four patients (63%) had a known diagnosis of asthma while 20% reported no previous diagnosis but had symptoms consistent with asthma. Patient characteristics associated with hospitalisation were: uncontrolled asthma symptoms in the month before the thunderstorm-asthma event, symptomatic allergic rhinitis, high blood eosinophilia and lower lung function.

**Conclusion:** Thunderstorm-asthma affects people with seasonal rhinitis, ryegrass sensitisation and can occur without prior history of asthma, with dramatic potential to inundate a healthcare system. Our data suggests that hospitalization, and thus a more severe thunderstorm-asthma exacerbation, was associated with a known history of asthma, prior uncontrolled asthma symptoms, allergic rhinitis, high eosinophil count and lower lung function. These factors may inform strategies to identify those most at risk of thunderstorm-asthma.

## INTRODUCTION

On 21<sup>st</sup> November 2016, Melbourne, Australia experienced a catastrophic thunderstorm-associated asthma event on a scale unprecedented worldwide, from which important lessons can be learnt. On that evening a powerful storm front passed over the greater Melbourne region from the west between 5pm and 6.30pm. The number of people developing symptoms of acute asthma, many for the first time, rapidly overwhelmed health resources. There were as many as 3460 excess respiratory-related emergency department (ED) presentations (556% above the three-yearly average of 766) and a 681% increase in asthma-related admissions (three-yearly average of 77) over 30 hours.<sup>1</sup> Tragically, this event culminated in 10 asthma deaths.<sup>2</sup>

Thunderstorm-asthma (TA) events are not unique to Australia, with previous reported outbreaks in United Kingdom, Italy and Canada.<sup>3-5</sup> All thunderstorm asthma events in Melbourne have occurred in November, coinciding with spring peak ryegrass pollen season.<sup>4</sup> It is largely accepted that TA events occur when a storm's strong wind gusts hit a populated centre with high concentrations of aeroallergens (pollen and/or fungal spores), sweeping the pollen grains near at-risk populations. It is also believed that the sudden increase in humidity provokes an osmotic shock, bursting the pollen grain into starch particles small enough to reach the lower airways.<sup>6,7</sup> Similar to an in-laboratory allergen challenge, TA events can initiate characteristic early and late obstructive asthmatic responses.<sup>8</sup> While it is believed that the early bronchospasm that occurs before the influx of inflammatory cells is responsive to bronchodilators, it is still uncertain why some patients developed acutely life-threatening exacerbation unresponsive to first line treatments by paramedics, warranting hospitalization. Mortality from TA is uncommon,<sup>8</sup> and the unparalleled scale and severity of this Melbourne event warrants careful analysis, especially as climate change and associated extreme weather patterns are predicted to increase the likelihood of future events.<sup>7</sup>

Previous epidemiological studies of TA have identified at-risk populations for asthma symptoms as those with poorly-controlled asthma, those failing to take regular inhaled corticosteroid preventer therapy and seasonal allergic rhinitis.<sup>5,9,10</sup> However, a major limitation in most previous studies on thunderstorm-asthma is lack of proper control groups that are required to draw conclusions on associations. More recently, results from telephone interviews following the 2016 event conducted by eight Melbourne metropolitan health services identified that individuals with current asthma or prior hospitalization in the previous 12 months are at higher risk of TA hospitalization.<sup>11</sup> However, this study did not investigate lung function or biomarkers that may be clinically important in the patients that were severely affected by the event. Understanding of subgroups that are at risk of being more severely affected by thunderstorm-asthma remains incomplete and an area that requires urgent attention. Our aims were to identify patient characteristics and clinically measurable variables and biomarkers associated with hospital admission compared to those who had been discharged from the ED.

## METHODS

### Study design

We conducted a cross-sectional, retrospective analysis of clinical characteristics of thunderstorm-asthma patients who presented to The Royal Melbourne Hospital (RMH), an adult metropolitan tertiary service in North-West Melbourne, who subsequently attended clinic review.

### **Study sample**

We reviewed records of all patients who attended RMH ED with TA from 5pm on 21<sup>st</sup> November to midnight 22<sup>nd</sup> November 2016 and subsequently contacted them by phone to offer an outpatient clinic review for evaluation and optimization of asthma care. The study was approved by the Human Research Ethics Committee of Melbourne Health. It should be noted that these same patients were contacted as part of the large telephone survey that has been published by Thein and colleagues<sup>2</sup> however the present study observes additional outcomes and physiological and biological markers from those who presented for outpatient review to the Royal Melbourne Hospital.

### **Study measurements**

At follow-up in the outpatient clinic, all patients were interviewed using a standardized template by a respiratory physician. Asthma was defined by a history compatible with intermittent symptoms of cough, dyspnoea or wheeze (or if presenting for the first time during this event, as acute cough, dyspnoea or wheeze after the passage of the thunderstorm on 21<sup>st</sup> November). If this diagnosis was made by a medical practitioner prior to the November hospital presentation, we defined this as previously-diagnosed asthma. Asthma control prior to the event was assessed retrospectively during the visit and defined as asthma symptoms more than twice weekly in the month preceding. The Sinus Outcome Test-22 (SNOT-22) and Asthma Control Questionnaires (ACQ) were applied, assessing symptoms and control of rhinitis and asthma respectively, with lower scores indicating better control. Clinical measurements – taken at follow-up – included spirometry (Vmax Encore®), fraction of exhaled nitric oxide (FeNO, Niox Vero®), blood eosinophil count, total IgE and specific-IgE to aeroallergens (Phadia 250, Thermo Fisher Scientific®): ryegrass, bermuda grass, *cladosporium*, *alternaria* and house dust mite (*dermatophagoides pteronyssinus*). Positive sensitization was defined as specific IgE more than 0.35 KUA/L and “very high” sensitization as IgE more than 17.5 KUA/L. All values above the maximum limit of detection were analysed with the limit value of 100 KUA/L.

### **Statistical analysis**

Data were analyzed with STATA 14 using non-parametric or parametric tests as appropriate to distributions. For the IgE variables with a truncated distribution (100 KUA/L cut-off), median was reported for the descriptive analyses. For categorical variables, counts and percentages were reported, and group differences were tested using Chi-square test.

## **RESULTS**

Two-hundred and forty patients presented to RMH with TA (the three-yearly November average for RMH ED asthma presentations was 28) in which 56% arrived at the ED in the first 6 hours after the storm (**Figure 1A**). Forty-three were admitted: 27 to ED short stay unit; 13 to respiratory wards (3 initially received non-invasive ventilation); 3 to ICU requiring intubation. The remainder were discharged directly from ED (n=128) or prior

to full medical assessment (n=69). There were no deaths at RMH. From paramedic notes, 29 patients arrived by ambulance, including 5 patients who were resuscitated by the paramedics with either adrenaline, invasive or non-invasive ventilation or chest compressions. The median time between the storm and patient emergency calls to ambulance Victoria was at 63 minutes for the resuscitated patients compared to 345 minutes for all other ambulances directed to RMH. Salbutamol was used prior to paramedic arrival in 76% of the cases (p=0.03). Of the 240 patients, 70 attended outpatient review, 49 declined or failed to attend, 45 had incorrect contact details, and 76 were otherwise uncontactable.

#### Characteristics of those who attended the clinical review.

The demographics of the 70 patients completing clinical review are recorded in **Table 1**. Reviewed patients were slightly younger compared to the patient group that were not able to be followed-up (mean age 35 vs 35.9 years, p<0.01). Gender distribution was comparable between the groups (p=0.62).

Of the 70 reviewed patients, 44 (63%) had previously-diagnosed asthma and the majority (79%) had active symptoms in the preceding year. Previous asthma hospitalization was described in 34% and previous TA exacerbation in 16%. No previous asthma diagnosis was recorded in 26 (37%) patients. From the overall group, 12/70 patients (17%) had previous history of symptoms suggestive of asthma consistent with 'undiagnosed asthma', whilst 14/70 (20%) had developed asthma symptoms for the first time on 21<sup>st</sup> November. While 46 (66%) described uncontrolled asthma symptoms in the month leading to the event, only 12 (17%) claimed to be using inhaled corticosteroid (ICS) preventer therapy prior to the thunderstorm, and 31 (44%) were using short-acting bronchodilators alone.

At clinic review (median 36 days post-event) patients were still experiencing asthma symptoms (mean ACQ of 1.20) and symptomatic allergic rhinitis (mean SNOT-22 score of 29). Spirometry at clinic review was within normal limits (median FEV<sub>1</sub> 92.5% pre-bronchodilator, 98.5% post-bronchodilator). Only 15 patients (21%) demonstrated significant bronchodilator reversibility ( $\geq 12\%$  and  $\geq 200\text{ml}$  change in FEV<sub>1</sub>).

#### Characteristics associated with hospitalization among those who attended the clinical review.

Nineteen (27%) individuals were admitted to hospital from the ED whilst 51 (73%) were discharged from ED (**Table 1**). In the hospitalized patients, 8 (42%) were born overseas compared to 36 (71%) of the non-admitted patients. Whilst a prior asthma diagnosis was evident in similar proportions of both admitted and non-admitted patients, 17 (90%) of those admitted to hospital had uncontrolled asthma symptoms in the previous month compared to 29 (55%) of those who were not admitted to hospital (p=0.01). Those admitted to hospital also appeared to be a more severe subgroup, having a much higher rate of previous hospital admission for asthma (7 (54%) of the hospitalized patients versus 8 (26%) of the non-hospitalized patients (p=0.07)). Those admitted patients also had a significantly higher SNOT-22 score indicating more severe rhinitis symptoms (p<0.001). No difference in medication use was observed prior to hospital admission between hospitalized and non-hospitalized patients.

Biomarkers taken at clinic review showed that median total IgE titres were elevated at 292 kU/L (normal <120), with a trend towards having higher titres if hospitalized

( $p=0.055$ ). Strikingly, 100% of patients were sensitised to ryegrass, with allergen-specific-IgE with median values classified in the very high range. Sensitisation to Bermuda grass, *alternaria* and *cladosporium* were present in 96%, 13% and 9% of patients respectively. Fungal sensitisation was low and was not associated with hospitalization, in contrast to previous TA events.<sup>12,13</sup> Median blood eosinophil counts were elevated overall at  $0.30 \times 10^9/L$ , being higher in those who were hospitalized ( $0.40$  v  $0.30$ ,  $p=0.03$ ). Hospitalized patients had significantly lower post-bronchodilator FEV<sub>1</sub> than non-hospitalized, (post-bronchodilator FEV<sub>1</sub> 96.3% v 99.7%,  $p<0.001$ ).

## DISCUSSION

Our findings confirm universal sensitivity to ryegrass and, consistent with previous literature,<sup>3,5</sup> a very high prevalence of allergic rhinitis in TA patients, suggesting this is a predictor of susceptibility, as is the low use of inhaled asthma preventer medications overall. This report extends these previous observations by observing potential risk factors for hospital admission compared to emergency presentation after outpatient evaluation, highlighting opportunities for prevention of severe attacks.

Whilst previous reports indicate that more than half of patients who present with TA do not have a previous asthma diagnosis,<sup>2</sup> an important finding of this study was that 2/3 of those admitted had a previous asthma diagnosis and over 90% of them had asthma symptoms in the month preceding the TA event. Our findings are supported by previous data on the 2016 Melbourne TA event which indicated that all 35 patients hospitalized in ICU had previously known doctor-diagnosed asthma,<sup>2,14</sup> suggesting known history of asthma as a risk factor for more severe TA.

Poor asthma control was associated with increased risk of hospital admission and, evidently, these patients were sub-optimally treated, providing opportunities for intervention. The large proportion receiving no medication or short-acting  $\beta$ -agonists alone, despite recent uncontrolled symptoms, strengthens a case for recommending ICS for patients with asthma and seasonal allergic rhinitis. Such a preventive approach is very important if they have ryegrass-induced seasonal allergic rhinitis and a previous history of TA. Although TA epidemics are uncommon, it also suggests the possibility that pollen-associated asthma exacerbations might be underreported. A previous Canadian study demonstrated that the number of emergency visits in children were increased by 35% during summer thunderstorm activity.<sup>15</sup> These findings highlight that patients with seasonal asthma warrant careful assessment during pollen season.

Given that most patients were symptomatic of asthma in the lead up to the event (66%) regardless of whether they had a previous diagnosis of asthma, and almost all had allergic rhinitis (96%), it may be that targeting recognition of symptoms of asthma and allergic rhinitis in public health campaigning prior to pollen season could be more effective at preventing morbidity overall. Symptomatic patients would be directed towards local healthcare providers for further evaluation and management, including education on medication adherence. Moreover, despite TA events being sporadic, education of providers on the importance of screening, assessing and optimising seasonal asthma and rhinitis treatment may be an important tool in reducing pollen allergy burden even in the absence of thunderstorms (**FIGURE 2**).<sup>16</sup>

Consistent with previous studies investigating the Melbourne 2016 TA event, our study found that ryegrass reported allergy was universal in TA patients but did not confer an increased risk of hospital admission.<sup>11</sup> In contrast to other studies however,<sup>11</sup> we found that more symptomatic allergic rhinitis (higher SNOT-22 score) was associated with hospital admission. The discrepancy may be based on the screening tools used in each of the studies. The SNOT-22 score used for this study is validated for assessing symptom control in allergic rhinitis, compared to tools used in other studies which looked at disease severity. As there are no studies comparing severity as opposed to control of disease in allergic rhinitis, we offer this finding as an area of further research.

Our data suggest type-2, eosinophilic inflammation with high blood eosinophilia, uncontrolled allergic rhinitis, high levels of total IgE, high ryegrass-specific IgE sensitization and lower lung function may constitute a specific phenotype susceptible to severe thunderstorm-associated exacerbations. This is a novel finding and is consistent with in-laboratory allergen challenges where airway hyperresponsiveness and allergic sensitization correlate with the severity of bronchoconstriction.<sup>17</sup> Based on previous TA event reports, it has been suggested that the early asthmatic response likely triggered most of the TA emergency visits (**FIGURE 1B**).<sup>18</sup> The acute response is followed by an inflammatory (late) phase which requires corticosteroid treatment in the ED. In our cohort, **Figure 1A** demonstrates that 56% of patients presented before midnight suggesting that both early and late responses were contributing to ED visits. Furthermore, 44% of patient assessed at clinic used bronchodilators prior to the event, suggesting that bronchodilators might have ameliorated the initial bronchospasm in some patients presenting later to the hospital. Nevertheless, there were 5 patients who developed acutely severe exacerbation requiring resuscitation by paramedics within the first hours of the storm passage. From our clinical assessment, all had active uncontrolled asthma prior to the storm suggesting that inflamed and hyperactive airways might have contributed to the severity of the bronchospasm. This is consistent with previous studies suggesting that the degree of airways hyperactivity induced by allergen challenges correlate with the magnitude of the inflammatory response.<sup>18,19,20</sup>

Our study is limited by the relatively small sample size and potential for selection bias with the low response rate, as well as recall bias given the retrospective analysis. Loss to follow-up was partly due to incorrect or incomplete contact details recorded for patients during the acute event making patients untraceable. Further, there was a reluctance by patients to attend for clinical evaluation of their symptoms post the TA event, thus contributing to the small sample size. These factors were similarly encountered in the few studies that did report on outpatient clinic assessment of TA patients,<sup>11,21</sup> and perhaps reflect a larger issue in the community of individuals under-recognising the seriousness and subsequent implications of uncontrolled asthma.

Notwithstanding this, our study showed consistent findings with larger studies on the Melbourne 2016 TA event.<sup>2,11,22</sup> Our study, however, was novel in terms of assessing patients in an outpatient clinic setting to investigate clinical biomarkers which may be useful to risk stratify patients in the future. We found trends of raised biomarkers including blood eosinophilia and total IgE were associated with more severe events and hospitalisation suggesting that these markers deserve validation in a prospective study. Interestingly, recent publications have demonstrated a positive association between

specific IgE levels and the risk of upper and lower airways allergen-related symptoms with pollen and house dust mites.<sup>23,24</sup> Approaches to find biomarkers in at-risk populations for TA is highly relevant. Our results suggest that there was a trend of increased IgE in hospitalized patients. We were not able to assess the role of ryegrass-specific IgE since results were truncated at a cut-off of 100 KUA/L.

Since future episodes of the weather events leading to thunderstorm asthma are highly likely, patients and healthcare providers need to be aware of the risk of asthma symptoms in individuals with allergic rhinitis. Our study suggests that asthma symptoms in those with seasonal allergic rhinitis are a likely indicator of risk in the setting of a TA event and so should be identified and treated. Treatment options for allergic rhinitis for which some evidence of efficacy in asthma exists includes intranasal corticosteroids and montelukast, although there has been some debate about the former and neither of these agents have been studied in a prospective manner in seasonal allergic asthma due to grass pollen sensitisation.<sup>25,26,27</sup> Recommended treatments for episodic asthma include seasonal inhaled asthma preventive inhaled corticosteroids with as needed short-acting beta 2 agonist, or as needed ICS/LABA (long-acting beta2-agonist) combination therapy, particularly in those likely to have poor adherence to regular preventer use.<sup>28,29</sup> Allergen immunotherapy has efficacy in allergic rhinoconjunctivitis due to grass pollens, and a recently published retrospective analysis provides evidence that sensitized individuals were protected from TA by grass pollen sublingual tablet immunotherapy, reinforcing the potential role of immunotherapy in TA.<sup>30,31,32</sup>

## CONCLUSION

The Melbourne TA event illustrates how climatic conditions can affect a large population of susceptible individuals, leading to a surge in hospital presentations and admission for acute asthma. In this retrospective analysis, hospitalisation following a TA event was associated with poor asthma control earlier in the hay fever season, active allergic rhinitis and blood eosinophilia. The risk factors for admission identified in this study warrant further validation in case-controlled and prospective studies to inform targeting of preventive campaigns. It is likely that climate change and associated extreme weather patterns will increase the likelihood of future events; nonetheless the experience learned in recent events enables us to inform practitioners of these factors as potential risk factors in the identification and management of TA.



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**Competing interest statement:**

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**Table 1: Demographics, medical history and potential risk factors for hospitalization of the 70 patients reviewed in clinic**

Demographics and medical history	All (n=70)	Non-hospitalized (n=51)	Hospitalized (n=19)	P value
Age (years), mean (SD)	35 (10)	37 (10)	31 (10)	0.048 <sup>#</sup>
Female, n (%)	36 (51)	25 (49)	11 (58)	0.60 <sup>^</sup>
Country of birth				
Australia, n (%)	26 (37)	15 (29)	11 (58)	0.05 <sup>^</sup>
Overseas, n (%)	44 (63)	36 (71)	8 (42)	
Smoking History, n (%)				
Never-smoker	48 (69)	35 (69)	13 (68)	0.53 <sup>^</sup>
Ex-smoker	7 (10)	4 (8)	3 (16)	
Current smoker	15 (21)	12 (24)	3 (16)	
Potential risk factors for hospitalization				
Location, n (%) <sup>*</sup>				
Outdoors	35 (51)	24 (49)	11 (58)	0.71 <sup>^</sup>
Inside, windows open	23 (34)	17 (35)	6 (32)	
Inside, windows closed	10 (15)	8 (16)	2 (11)	
Allergic rhinitis history, n (%)	67 (96)	50 (98)	17 (90)	0.12 <sup>^</sup>
Asthma history, n (%)				
Previous asthma diagnosis	44 (63)	31 (61)	13 (68)	0.56 <sup>^</sup>
Undiagnosed asthma	12 (17)	7 (14)	5 (26)	0.21
No previous asthma <sup>§</sup>	14 (20)	13 (26)	1 (5)	0.06
Uncontrolled asthma symptoms in month prior to storm <sup>§</sup>	46 (66)	29 (57)	17 (90)	0.01 <sup>^</sup>
Medication prior to storm	(n=70)	(n=51)	(n=19)	
Asthma medication, n (%)				
None	27 (39)	23 (45)	4 (21 )	0.066
SABA or SAMA only	31 (44)	21 (41)	10 (53)	0.39
ICS + SABA/SAMA	12 (17)	7 (14)	5 (26)	0.21
Nasal steroids	19 (27)	16 (31)	3 (16)	0.19

Clinical measurements	(n=70)	(n=51)	(n=19)	
ACQ, mean (SD)	1.20 (1)	1.00 (1)	1.50 (1)	0.11 <sup>#</sup>
SNOT-22, mean (SD)	29 (20)	27 (19)	33 (23)	<0.001 <sup>#</sup>
Blood eosinophil count (×10 <sup>9</sup> /L) median (IQR)‡‡	0.30 (0.20)	0.30 (0.40)	0.40 (0.40)	0.03 <sup>†</sup>
IgE (KU/L) median (IQR)	292 (524)	210 (393)	360 (712)	0.055 <sup>†</sup>
Specific IgE to ryegrass (KUA/L) median (IQR)	85.20 (51.90)	70.4 (60.35)	100 (23.40)	-
FeNO (ppb)	59 (41)	59 (43)	45 (32)	0.21 <sup>#</sup>
Lung Function, mean (SD)				
% predicted post-bronchodilator FEV1	98.5 (16.3)	99.7 (16.8)	96.3 (15.6)	<0.001 <sup>#</sup>
% reversibility of FEV1	9.1 (14.1)	9.5 (15.4)	7.9 (9.6)	<0.001 <sup>#</sup>
Number with significant reversibility‡, n (%)	15	10/50	5/18	0.49 <sup>^</sup>

ACQ=Asthma Control Questionnaire; FeNO=fractional exhaled nitric oxide; FEV1=forced expiratory volume in 1 second; FVC=forced vital capacity; ICS=inhaled corticosteroids; IgE=immunoglobulin E; IQR=interquartile range; NSAID=Non-steroidal anti-inflammatory drug; SABA=short acting beta-2-agonist; SAMA=short acting antimuscarinic; SNOT-22=sinonasal outcome test-22.

<sup>#</sup> Z test

\*Percentage of those with known data.

<sup>^</sup> Chi<sup>2</sup> test

<sup>†</sup>Non-parametric (Mann-Whitney) test.

‡Significant reversibility is ≥200ml AND ≥12% increase between pre- and post-bronchodilator spirometric measurements.

‡‡Blood eosinophil counts, IgE levels, FeNO and pulmonary function were measured at the time of clinic review.

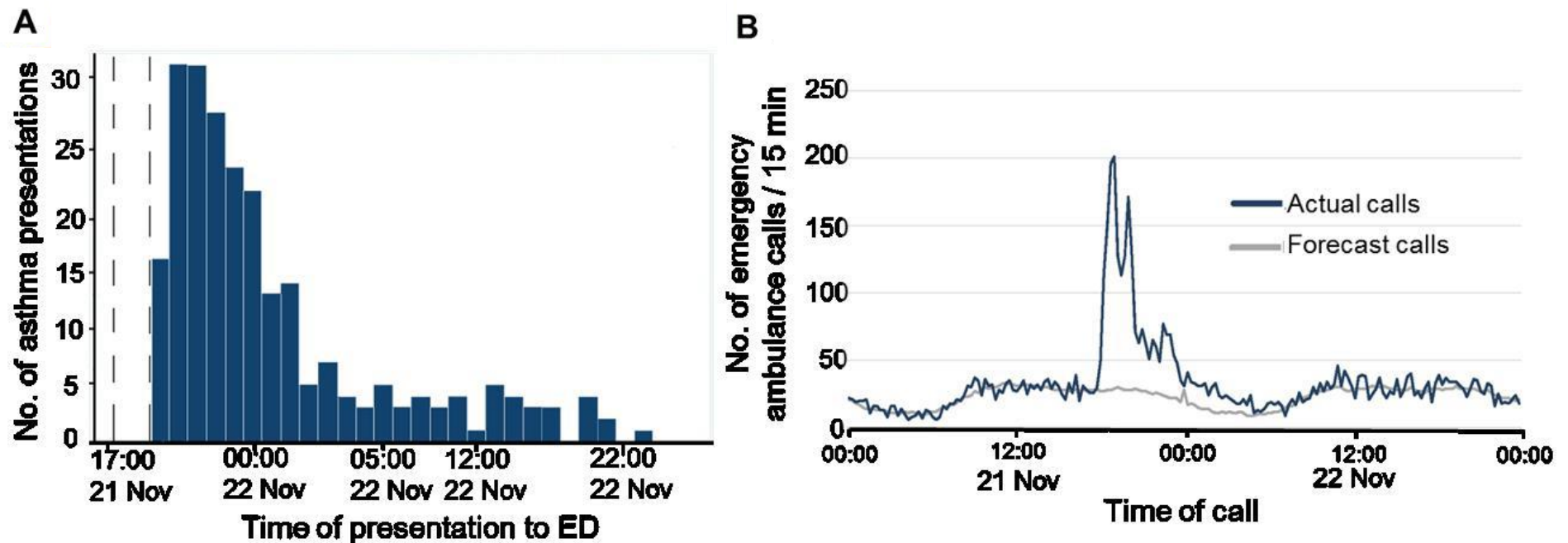
\$Those with no previously known asthma diagnosis or symptoms, which developed symptoms of asthma only for the first time on 21st November 2016.

§ Twice weekly asthma symptoms or more in the month preceding the thunderstorm-asthma event.

**Figure 1**

(A) Timing of thunderstorm asthma presentations to RMH ED. Vertical lines show timing of transit of storm over the Melbourne metropolitan area.

(B) Timing of calls to Victorian emergency services (Courtesy of State of Victoria: Review of response to the thunderstorm asthma event of 21-22 November 2016 - Final Report)



**Figure 2: Asthma and Allergic Rhinitis Management Strategy**

<b>CURRENT ASTHMA and ALLERGIC RHINITIS</b> <ul style="list-style-type: none"><li>• Review asthma control and treatment<ul style="list-style-type: none"><li>• If regular ICS are not otherwise indicated but history of uncontrolled <b>seasonal asthma</b> is present, consider regular use of ICS at a minimum during pollen season.</li><li>• If patient ever had asthma precipitated by a thunderstorm (or similar storm event) then advise ICS over pollen season</li></ul></li><li>• Updated Asthma action plan to include seasonal asthma</li><li>• Review allergic rhinitis control and treatment</li><li>• Outdoor allergen avoidance strategies</li></ul>	<b>ALLERGIC RHINITIS and NO ASTHMA</b> <ul style="list-style-type: none"><li>• Review allergic rhinitis control and treatment</li><li>• Asses for asthma symptoms and if present evaluate variable airflow obstruction</li><li>• Outdoor allergen avoidance strategies</li><li>• Possible consideration of pollen desensitisation immunotherapy before pollen season</li></ul>
<b>ASTHMA HISTORY and ALLERGIC RHINITIS</b> <ul style="list-style-type: none"><li>• Ensure asthma is quiescent and controlled</li><li>• Updated Asthma action plan to ICS as needed strategy</li><li>• Review allergic rhinitis control and treatment</li><li>• Outdoor allergen avoidance strategies</li></ul>	<b>NO ALLERGIC RHINITIS and NEVER ASTHMA</b> <ul style="list-style-type: none"><li>• Very low risk for epidemic thunderstorm asthma</li></ul>

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