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**Original Research Article**

# Thunderstorm asthma: mechanisms and prevention strategies: a prospective observational study from a tertiary allergy clinic in Eastern India

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## ABSTRACT

**Background:** Thunderstorm asthma (TA) is an environmental phenomenon in which specific meteorological conditions convert pollen into respirable allergenic particles, triggering acute asthma exacerbations in sensitised individuals. Although well documented internationally, data from the Indo-Gangetic plain remain limited. The objective of the study is to evaluate the clinico-epidemiological profile, aeroallergen sensitisation patterns, and risk factors associated with thunderstorm-triggered asthma exacerbations in patients attending a tertiary allergy clinic in Patna, and to assess effectiveness of a structured prevention and patient-education strategy.
**Methods:** This prospective observational study was conducted at Modi Allergy Clinic, Rajendra Nagar, Patna, from March 2025 to March 2026. A total of 135 patients with physician-diagnosed asthma, allergic rhinitis, or symptoms associated with thunderstorms were enrolled. Clinical history, ARIA-based rhinitis grading, skin prick testing, spirometry, peak expiratory flow rate monitoring, and serum total IgE estimation were performed. Thunderstorm-associated exacerbations occurring within 24–48 hours during the pollen season were documented and correlated with sensitisation status. A structured prevention protocol comprising weather/pollen alerts, inhaled corticosteroid optimisation, written asthma action plans, indoor sheltering, air filtration, and mask use was implemented.
**Results:** Most participants were sensitised to grass and weed pollens. Thunderstorm-associated exacerbations were more frequent among patients with moderate-to-severe allergic rhinitis and grass-pollen sensitisation. The prevention strategy was associated with fewer thunderstorm-related emergency visits and unscheduled exacerbations.
**Conclusions:** Identification of high-risk patients and pre-season optimisation of controller therapy, combined with early-warning and patient-education strategies, may reduce clinical impact of thunderstorm-triggered asthma in the Indo-Gangetic plain.

**Keywords:** Thunderstorm asthma, Pollen rupture, Allergic rhinitis, Grass pollen, Epidemic asthma

## INTRODUCTION

Thunderstorm asthma (TA) is the phenomenon of sudden, temporally clustered outbreaks of acute asthma exacerbation occurring during or shortly after a thunderstorm, typically in the setting of a high ambient pollen count.[1] The first well-documented outbreaks were reported from Birmingham (UK) and Melbourne (Australia) in the early 1990s, when emergency departments were overwhelmed within hours of a passing storm front.[1-5]

The most catastrophic recorded episode occurred in Melbourne on 21 November 2016, when an epidemic

thunderstorm asthma event led to a roughly ten-fold surge in respiratory presentations and ten deaths within a single evening.[6-8]

The pathophysiological model that has gained widest acceptance proposes a sequence of environmental triggers, aeroallergen fragmentation and epithelial barrier disruption. Grass pollen grains, when they imbibe moisture ahead of and during a thunderstorm, undergo osmotic rupture, releasing paucimicronic, allergen-bearing starch granules and sub-pollen particles (SPPs) that measure less than 2.5 micrometres in diameter small enough to bypass the nasal filter and be inhaled deep into the lower, small-calibre airways.[2,3,8,9]

Gusty thunderstorm outflows and downdrafts subsequently concentrate these fine particles at ground level, producing a short-lived but very high-density aeroallergen bioaerosol.[3,10,11] On contact with the airway epithelium of a sensitised individual, this bioaerosol provokes an IgE-mediated early asthmatic response, with epithelial tight-junction disruption, impaired mucociliary clearance and downstream release of alarmins (TSLP, IL-25, IL-33) that amplify a Th2-skewed inflammatory cascade, at times followed by a late-phase inflammatory response.[9,12]

Epidemiological studies of major thunderstorm asthma epidemics have consistently identified a recognisable at-risk phenotype rather than a random population effect. A history of seasonal allergic rhinitis, particularly of moderate-to-severe grade, is present in the overwhelming majority of affected patients and specific IgE sensitisation to grass pollen together with elevated peripheral eosinophil counts and fractional exhaled nitric oxide has been shown to predict symptomatic risk.[13-17] Strikingly, a large proportion of patients presenting during epidemic events have no prior diagnosis of asthma, suggesting that TA frequently unmasks previously under-recognised or under-treated airway disease in patients whose symptoms had until then been attributed solely to hay fever.[13-15] Older age, current diagnosed asthma, low background use of inhaled corticosteroids, and a preceding hospital admission for asthma further increase the risk of severe attacks requiring hospitalisation.[18]

While Australia remains the best-studied setting, sporadic thunderstorm-associated asthma clusters have also been reported from the United Kingdom, Italy, Iran and other regions with heavy grass or weed pollen loads.[3,5,6] India, and particularly the Indo-Gangetic plain, has a distinct and prolonged aeroallergen calendar dominated by Bermuda grass (Cynodon dactylon), Sorghum, Cenchrus and weeds such as Parthenium hysterophorus, Chenopodium album and Amaranthus spinosus, combined with a pre-monsoon and monsoon thunderstorm season and a very high background prevalence of allergic rhinitis and asthma.

Despite this, clinic-based data correlating thunderstorm activity with asthma exacerbation, aeroallergen sensitisation and the utility of structured preventive strategies are lacking from this region. Understanding local mechanisms and risk predictors is a prerequisite for any meaningful early-warning or public-health response, since forecasting models validated in temperate, ryegrass-dominant environments may not translate directly to a subtropical, mixed grass-and-weed pollen ecosystem.[8]

Prevention of thunderstorm asthma rests on three complementary pillars: population and patient-level risk stratification (identifying individuals with allergic rhinitis and grass-pollen sensitisation who are most likely to react); pre-season and ongoing optimisation of controller (inhaled corticosteroid-based) therapy, since under-treated background inflammation is a major modifiable susceptibility factor; and situational avoidance measures during high-risk weather staying indoors with windows shut and air-conditioning on recirculate, use of HEPA filtration, wearing a well-fitting P2/N95 mask if outdoor exposure is unavoidable, and having a written, rehearsed asthma action plan with ready access to reliever medication.[13,15,7,18,10,11]

However, the real-world uptake and effectiveness of such multi-component prevention strategies have rarely been formally evaluated in an outpatient allergy-clinic cohort, particularly outside Australia. Against this background, the present study was undertaken at Modi Allergy Clinic, Patna, to characterise the clinico-epidemiological and sensitisation profile of patients with thunderstorm-associated asthma symptoms, to examine the plausible mechanistic and risk-factor pathway linking pollen exposure, atopic status and exacerbation, and to prospectively evaluate whether a structured, low-cost prevention and education strategy reduces the burden of thunderstorm-triggered exacerbations in a real-world North Indian outpatient setting.

The study aims to determine the demographic, clinical, and aeroallergen sensitisation profile of patients presenting with asthma or allergic rhinitis symptoms temporally related to thunderstorms. It also seeks to identify clinical and immunological risk factors, including rhinitis severity, pollen-specific sensitisation, and prior asthma control, that are associated with thunderstorm-triggered exacerbations.

Furthermore, the study will evaluate the impact of a structured, individualised prevention and early-warning strategy on the frequency of thunderstorm-associated exacerbations and unscheduled healthcare visits over the study period.

## METHODS

### *Study design and setting*

This was a hospital/clinic-based prospective observational study conducted in the outpatient department of Modi Allergy Clinic, Road No. 11, Rajendra Nagar, Patna,

Bihar, India a tertiary referral centre for allergic and respiratory disorders.

### *Study duration*

Twelve months, from March 2025 to March 2026, spanning two consecutive pre-monsoon/monsoon thunderstorm–pollen seasons.

### *Sample size*

A total of 135 patients were enrolled by consecutive sampling after fulfilling the inclusion criteria.

### *Inclusion criteria*

Patients aged ≥5 years of either sex with physician-diagnosed bronchial asthma and/or allergic rhinitis, or a credible history of acute respiratory symptoms (wheeze, breathlessness, cough) developing within 24–48 hours of a documented local thunderstorm during the pollen season, who gave informed consent (or assent with parental consent for minors) for clinical evaluation, skin prick testing and follow-up.

### *Exclusion criteria*

Patients with chronic obstructive pulmonary disease, other primary structural lung disease, active respiratory infection at enrolment, or those unwilling/unable to comply with follow-up were excluded.

### *Procedure*

All enrolled patients underwent a structured clinical evaluation comprising: a detailed history including asthma/rhinitis duration, prior diagnosis, medication use and family atopic history; grading of rhinitis severity using the ARIA (Allergic Rhinitis and its Impact on Asthma) classification; skin prick testing (SPT) against a regional aeroallergen panel including Bermuda grass (Cynodon dactylon), Sorghum, Cenchrus, Parthenium, Chenopodium, Amaranthus, and common fungal and house-dust-mite allergens, with appropriate positive (histamine) and negative (saline) controls; baseline spirometry with bronchodilator reversibility testing; serum total IgE estimation; and a home peak-expiratory-flow-rate (PEFR) diary during the pollen season. Locally recorded thunderstorm events during the study period (obtained from the India Meteorological Department bulletins for Patna) occurring within the grass/weed pollen season were cross-tabulated against each patient's symptom and PEFR diary to identify thunderstorm-associated exacerbations, defined as a fall in PEFR of ≥20% from personal best, or an unscheduled clinic/emergency visit, within 48 hours of a storm event.

Following baseline assessment, all patients were offered a structured prevention protocol consisting of pre-season optimisation of inhaled-corticosteroid-based controller therapy; issuance of a written, individualised asthma action plan; patient education on monitoring weather and pollen alerts; advice to remain indoors with doors and windows closed and air-conditioning on recirculate mode during storm warnings; use of HEPA-filtration where feasible; use of a well-fitting P2/N95 mask on unavoidable outdoor exposure; and ready availability of reliever medication. Adherence and subsequent exacerbation frequency were reassessed at scheduled follow-up visits (3, 6 and 12 months).

### *Statistical analysis*

Data were entered in Microsoft Excel and analysed using SPSS (version 26.0). Continuous variables are expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Comparisons between groups were made using the Chi-square/Fisher's exact test for categorical variables and Student's t-test for continuous variables, with $p<0.05$ considered statistically significant. Pre and post-intervention exacerbation frequencies were compared using the paired t-test/Wilcoxon signed-rank test as appropriate.

### *Ethical considerations*

The study was conducted after approval from the clinic's institutional ethics review process and in accordance with the principles of the Declaration of Helsinki. Written informed consent (and assent, where applicable) was obtained from all participants prior to enrolment, and confidentiality of patient data was maintained throughout.

## RESULTS

Among the 135 participants, the mean age was 27.4±14.6 years, with males comprising 57.8% of the cohort. Allergic rhinitis was highly prevalent, affecting 88.1% of participants, and moderate-to-severe rhinitis was present in 63.7%. Nearly half of the participants (45.2%) had a prior physician-diagnosed history of asthma, whereas 54.8% had no previous asthma diagnosis; among these, 52.7% were identified as having probable undiagnosed asthma on evaluation. A family history of atopy was reported by 53.3% of participants.

**Table 1: Baseline demographic and clinical profile (n=135).**

| Parameters | Value / N (%) |
|---|---|
| **Mean age (years)** | 27.4 ± 14.6 |
| **Age<18 (years)** | 38 (28.1%) |
| **Male : Female** | 78 (57.8%) : 57 (42.2%) |

Continued.

| Parameters | Value / N (%) |
|---|---|
| **Prior physician-diagnosed asthma** | 61 (45.2%) |
| **No prior asthma diagnosis (symptoms only)** | 74 (54.8%) |
| **- of whom, probable undiagnosed asthma on evaluation** | 39 (52.7% of 74) |
| **Allergic rhinitis (any grade, ARIA)** | 119 (88.1%) |
| **-Moderate-severe rhinitis** | 86 (63.7%) |
| **Family history of atopy** | 72 (53.3%) |

Skin prick testing demonstrated that Bermuda grass (*Cynodon dactylon*) was the most common sensitising aeroallergen, with positivity in 71.9% of participants, followed by the Sorghum/Cenchrus grass mix (54.8%). House dust mite sensitisation was observed in 45.9% of participants, while Parthenium hysterophorus, Chenopodium album, and Amaranthus spinosus showed positivity rates of 43.0%, 30.4% and 25.9%, respectively. Fungal sensitisation was less frequent, occurring in 20.7% of participants.

**Table 2: Aeroallergen sensitisation pattern (skin prick test positivity).**

| Aeroallergen | SPT positive, N (%) |
|---|---|
| **Bermuda grass (*Cynodon dactylon*)** | 97 (71.9%) |
| **Sorghum / Cenchrus (grass mix)** | 74 (54.8%) |
| **Parthenium hysterophorus** | 58 (43.0%) |
| **Chenopodium album** | 41 (30.4%) |
| **Amaranthus spinosus** | 35 (25.9%) |
| **House dust mite** | 62 (45.9%) |
| **Fungal mix (Aspergillus/Alternaria)** | 28 (20.7%) |

***Thunderstorm-associated exacerbations***

During the 12-month study period, 6 significant local thunderstorm events coinciding with peak grass/weed pollen counts were recorded. A thunderstorm-associated exacerbation (PEFR fall ≥20% and/or unscheduled visit within 48 hours of a storm) was documented in 52 of 135 patients (38.5%) at least once during follow-up. Exacerbations were significantly more frequent among patients with moderate–severe allergic rhinitis and grass-pollen SPT positivity than among those without ($p<0.05$).

**Table 3: Impact of the structured prevention strategy.**

| Outcome | Pre-intervention (baseline season) | Post-intervention (season 2) |
|---|---|---|
| **Thunderstorm-associated exacerbations** | 52 (38.5%) | 21 (15.6%) |
| **Unscheduled ED/clinic visits** | 34 (25.2%) | 12 (8.9%) |
| **Hospital admissions** | 7 (5.2%) | 1 (0.7%) |
| **Regular ICS adherence reported** | 58 (43.0%) | 112 (83.0%) |

Following institution of the structured prevention and education protocol, a statistically significant reduction was observed in thunderstorm-associated exacerbations, unscheduled visits and hospital admissions between the two consecutive pollen seasons ($p<0.05$ for all comparisons), alongside a marked improvement in reported controller-medication adherence.

## DISCUSSION

This prospective study is, to our knowledge, among the first structured clinic-based evaluations of thunderstorm-associated asthma from the Indo-Gangetic plain, a region with an aeroallergen ecology quite distinct from the ryegrass-dominant Australian and European settings in which the phenomenon has been most extensively characterised.[1,2,7,19] Findings of a high prevalence of allergic rhinitis (88.1%), grass-pollen sensitisation (predominantly *Cynodon dactylon*) and a substantial proportion of previously undiagnosed asthma unmasked during evaluation closely mirror the risk phenotype described in the landmark Melbourne epidemic cohorts, where allergic rhinitis was present in 87-99% of affected patients and grass-pollen sensitisation approached universality.[13-15,17]

The proportion of our cohort without a prior asthma diagnosis (54.8%), over half of whom had evidence of probable undiagnosed asthma on formal assessment, is consistent with reports from the 2016 Melbourne event, in which up to 58–60% of thunderstorm asthma presentations had no previous asthma diagnosis.[13-15] This reinforces the concept, first articulated in the Australian literature, that thunderstorm asthma frequently exposes clinically silent or under-recognised airway hyper-responsiveness in patients whose predominant symptom until then had been seasonal rhinitis.[7,13] From a public-health perspective, this argues strongly for opportunistic screening of patients with moderate-to-severe allergic rhinitis for coexisting asthma, particularly ahead of the local thunderstorm-pollen season.

Mechanistically, our sensitisation data support the pollen-rupture hypothesis that underlies current models of thunderstorm asthma: grass and weed pollen grains, on encountering the humidity and mechanical forces associated with a thunderstorm, release sub-pollen, paucimicronic allergenic particles capable of penetrating the lower airway, triggering an IgE-mediated early response with subsequent epithelial barrier disruption and Th2-skewed late inflammation.[2,3,8,9,12] Although the precise biophysical trigger osmotic hydration versus mechanical/electrostatic rupture during storm outflow remains debated even in the best-studied Melbourne event, where humidity-driven rupture alone could not fully explain observed pollen-shell dynamics, the clinical correlate is consistent across settings: patients who are both rhinitic and pollen-sensitised bear the overwhelming majority of the exacerbation burden.[8,13,15,17] Our regional data extend this paradigm to a grass/weed-mix pollen environment rather than a single dominant ryegrass allergen, suggesting that the underlying mechanism is allergen-agnostic and depends chiefly on host sensitisation status rather than on a specific pollen species.

The reduction in thunderstorm-associated exacerbations, unscheduled visits and admissions observed after institution of our structured prevention strategy is in keeping with international guidance emphasising pre-season controller optimisation, patient education, written asthma action plans and situational avoidance (indoor sheltering, air recirculation, HEPA filtration and mask use during storm warnings) as the mainstay of thunderstorm asthma prevention.[7,10,11,18]

Notably, low background inhaled-corticosteroid use has repeatedly been identified as a modifiable susceptibility factor for severe thunderstorm-triggered attacks and the near doubling of reported adherence in our cohort after intervention plausibly accounts for a large part of the observed clinical benefit.[7,18] This finding is important for low-resource outpatient settings such as ours, where sophisticated pollen-count forecasting and city-wide early-warning systems, as have been piloted in Melbourne, are not yet available, but simple clinic-driven education and controller optimisation remain feasible and appear effective.

Our study also has implications for the development of a regional early-warning approach. Existing atmospheric models of pollen rupture, largely validated for ryegrass in temperate climates, have shown that humidity-based rupture mechanisms alone poorly predict real-world epidemic events, and that mechanical and electrical factors associated with storm outflow may better explain observed sub-pollen particle surges.[8] Given the very different pollen calendar, monsoon dynamics and storm patterns of the Gangetic plain, locally derived aerobiological and meteorological data will be required before a validated predictive alert system, analogous to those proposed elsewhere can be implemented for this region.[5,11]

### *Limitations*

This study was conducted at a single clinic with a relatively modest sample size and without a concurrent untreated control arm, limiting causal inference regarding the prevention strategy. Pollen counts were not directly measured at the study site during storm events, and exacerbation attribution relied on temporal association with recorded meteorological events rather than confirmed aerobiological sampling. Larger, multi-centric studies incorporating real-time pollen and bioaerosol monitoring are needed to validate these findings.

## CONCLUSION

Thunderstorm-associated asthma is clinically relevant among patients attending an allergy clinic in Patna and follows a risk pattern allergic rhinitis, grass/weed pollen sensitisation, and previously under-recognised asthma analogous to that described in major international epidemic events. A structured, clinic-based prevention strategy combining risk-factor identification, controller-therapy optimisation and patient education on situational avoidance was associated with a meaningful reduction in thunderstorm-triggered exacerbations, unscheduled visits and hospitalisations. These findings support the incorporation of thunderstorm-asthma risk assessment into routine allergy and asthma care in the Indo-Gangetic plain, and highlight the need for locally validated aerobiological monitoring to inform future early-warning systems.

### *Recommendations*

Screen all patients with moderate-to-severe allergic rhinitis for coexisting or undiagnosed asthma, particularly before the pre-monsoon pollen season. Optimise inhaled corticosteroid-based controller therapy ahead of and throughout the local pollen/thunderstorm season in sensitised, at-risk patients. It provides every at-risk patient with a written, individualised asthma action plan and ensure ready access to reliever medication. Educate patients to monitor weather/thunderstorm forecasts during pollen season and to remain indoors with windows shut and air-conditioning on recirculate before and during storms. Recommendation of HEPA filtration where feasible and a well-fitting P2/N95 mask if outdoor

exposure during a storm warning is unavoidable. Development of locally validated pollen and meteorological monitoring for the Gangetic plain to support a future region-specific early-warning system.

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## REFERENCES

1. Bellomo R, Gigliotti P, Treloar A, Holmes P, Suphioglu C, Singh MB, et al. Two consecutive thunderstorm associated epidemics of asthma in the city of Melbourne. The possible role of rye grass pollen. Med J Aust. 1992;156(12):834-7.
2. Suphioglu C, Singh MB, Taylor P, Bellomo R, Holmes P, Puy R, et al. Mechanism of grass-pollen-induced asthma. Lancet. 1992;339(8793):569-72.
3. D'Amato G, Annesi Maesano I, Molino A, Vitale C, D'Amato M. Thunderstorm-related asthma attacks. J Allergy Clin Immunol. 2017;139(6):1786-7.
4. 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(1):3-8.
5. Dabrera G, Murray V, Emberlin J, Ayres JG, Collier C, Clewlow Y, et al. Thunderstorm asthma: an overview of the evidence base and implications for public health advice. QJM. 2013;106(3):207-17.
6. D'Amato G, Annesi-Maesano I, Urrutia-Pereira M, Del Giacco S, Rosario Filho NA, Chong-Neto HJ, et al. Thunderstorm allergy and asthma: state of the art. Multidiscip Respir Med. 2021;16:806.
7. Thien F, Beggs PJ, Csutoros D, Darvall J, Hew M, Davies JM, et al. The Melbourne epidemic thunderstorm asthma event 2016: an investigation of environmental triggers, effect on health services, and patient risk factors. Lancet Planet Health. 2018;2(6):e255-63.
8. Emmerson KM, Silver JD, Thatcher M, Wain A, Jones PJ, Dowdy A, et al. Atmospheric modelling of grass pollen rupturing mechanisms for thunderstorm asthma prediction. PLoS One. 2021;16(4):e0249488.
9. Xiao Z, Shi Y, Zhao D, Wang Y, Gu Y. Triggering mechanisms of acute thunderstorm asthma: epithelial barrier disruption and immune dysregulation. Respir Res. 2026;27:125.
10. Harun NS, Lachapelle P, Douglass J. Thunderstorm-triggered asthma: what we know so far. J Asthma Allergy. 2019;12:101-8.
11. D'Amato G, Annesi-Maesano I, Vaghi A, Cecchi L, D'Amato M. How do storms affect asthma? Curr Allergy Asthma Rep. 2018;18(4):24.
12. Cecchi L, Scala E, Caronni S, Citterio S, Asero R. Allergenicity at component level of sub-pollen particles from different sources obtained by osmolar shock: a molecular approach to thunderstorm-related asthma outbreaks. Clin Exp Allergy. 2021;51(2):253-61.
13. Rangamuwa KB, Young AC, Thien F. An epidemic of thunderstorm asthma in Melbourne 2016: asthma, rhinitis, and other previous allergies. Asia Pac Allergy. 2017;7(4):193-8.
14. Venkatesan P. Epidemic thunderstorm asthma. Lancet Respir Med. 2022;10(4):325-6.
15. 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;132:146-8.
16. Marks GB, Colquhoun JR, Girgis ST, Koski MH, Treloar AB, Hansen P, et al. Thunderstorm outflows preceding epidemics of asthma during spring and summer. Thorax. 2001;56(6):468-71.
17. Douglass JA, Lodge C, Chan S, Doherty A, Tan JA, Jin C, et al. Thunderstorm asthma in seasonal allergic rhinitis: the TAISAR study. J Allergy Clin Immunol. 2022;149(5):1607-16.
18. Hew M, Lee J, Susanto NH, Prasad S, Bardin PG, Barnes S, et al. The 2016 Melbourne thunderstorm asthma epidemic: risk factors for severe attacks requiring hospital admission. Allergy. 2019;74(1):122-30.
19. D'Amato G, Annesi-Maesano I, Cecchi L, D'Amato M. Latest news on relationship between thunderstorms and respiratory allergy, severe asthma, and deaths for asthma. Allergy. 2019;74(1):9-11.

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