

Thunderstorms: a risk factor for asthma attacks

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Sometimes thunder and wheezes come together. One of the last occasions when such a combination was detected was in London during the summer of 1994. At 18.00 hours on 24 June there was a heavy thunderstorm with about 50 ground strikes of lightning, a fall in temperature from 26.5°C to 18.7°C, a rise in humidity from 50% to 87%, and rainfall of 46 mm.¹ A few hours later there was a massive increase in the number of patients presenting at the accident and emergency departments of several hospitals with wheezing and shortness of breath. In total, 604 patients were seen in 12 accident and emergency departments compared with an expected number of 66.² Was there any causal link between these two notable and uncommon events?

Strong arguments in favour of the possibility that asthma outbreaks are caused by thunderstorms have been provided by researchers in Melbourne where both events have occurred simultaneously on several occasions.³ The outbreaks of asthma in Melbourne were of a similar relative size to that in London with about a tenfold increase in the number of patients presenting to the accident and emergency departments with asthma. Patients affected during these outbreaks were more likely to be sensitised to rye grass pollen than non-epidemic asthmatic patients, and subsequently showed well defined responses to inhalation challenge tests with isolated starch granule suspensions containing *Lol p IX* obtained from pollen grains. Thunderstorms were thought to play a part by providing the appropriate circumstances for the pollen grains to break and release hundreds of starch granules of a respirable size, and in vitro evidence for this hypothesis was provided.⁴ Although the Melbourne outbreaks were suited to the investigation of their mechanisms, well defined epidemiological evidence of an association between the occurrence of asthma outbreaks and thunderstorms was not investigated; in addition, the studies of the mechanisms were performed in a small number of subjects so some doubt over the causality of the association remains.⁵

The simultaneous occurrence of asthma outbreaks and thunderstorms was also noticed by Packe and Ayres when describing an outbreak of asthma which occurred in Birmingham in 1983.⁶ In this paper the authors cited papers published since the 1950s which showed that rainfall may favour the release of fungal spores into the atmosphere, suggesting that, after thunderstorms with heavy rain, the effect on spore release may outweigh the effect of rain on the removal of suspended particles.^{7,8}

Asthma outbreaks are not easily amenable to epidemiological investigation because they tend to be rare. When a suspected association is not testable because of the small number of outbreaks, one can focus on the usual variations of asthma and determine whether thunderstorms are a risk factor for asthma admissions in a large population. The study in this issue of *Thorax* by Newson *et al* is an important contribution to this latter approach.⁹

The authors looked for evidence of an independent association between the occurrence of thunderstorms and the number of asthma admissions in hospitals of several regional health authorities in England, and also determined whether that association was modified by the level of airborne grass pollen. With this approach the authors were

able to test the behaviour of a population of several million, including several hundreds of thousands of asthmatic subjects, on about 1200 different days. A log linear model with a modification to account for repeated measures was used, which is one of the statistical methods to be used with time series. This type of technique is widely available and has recently been reliably used in a large multinational European study on the short term health effects of air pollution.¹⁰ A contribution of the study, which is of interest for future epidemiological studies, is the measure of thunderstorms using records of lightning flashes as provided by the Meteorological Office. Each flash of lightning corresponds to a sferic, which consists of an electromagnetic signal emitted during the lightning discharge. According to Lee¹¹ this method provides much greater spatial resolution than records of ground based observers.

Using 15 hour lagged values of sferics an association was found between thunderstorms with strong activity (high sferic density) and asthma admissions for both children and adults (RR 1.25; 95% CI 1.15 to 1.36), but there was a smaller association with low sferic density compared with the absence of sferics. This association was geographically consistent as it was present in 12 of the 14 geographical areas for those aged 14 years and over and in 10 of the 14 areas for those aged 0–14 years.

The association of a high level of pollen (≥ 50 g/m³/day) with asthma admissions, assessed in only five geographical areas, was modified by the sferics. Whereas there was a non-significant increase of only 3% in asthma admissions on days with a high pollen count and no sferics, the increase in asthma admissions was approximately 31% when both high pollen counts and high sferic density coincided (RR 1.31; 95% CI 1.21 to 1.42). This pattern was again geographically consistent.

The findings reported by Newson *et al* deserve consideration from two distinct angles – namely, aetiology and public health. They are important from an aetiological perspective because they provide additional evidence in support of a causal relationship between thunderstorms and attacks of asthma. Unfortunately the categorisation of sferics resulted in very small numbers in the high sferic density group for which an association was observed, and the criteria for stratification of sferics was not given. The relationship between thunderstorms, pollen counts, and asthma admissions was carefully adjusted for day-of-the-week and seasonal effects as well as for the potential impact of administrative changes in the area covered by some health authorities. Could other non-reported factors have confounded the reported association? Viral infections have been found to be a risk factor for asthma attacks both in children¹² and adults¹³ and, at least in theory, may have been seasonally correlated with the distribution of thunderstorms. Whether adjustment for the season could have involved partial adjustment for viruses is difficult to say. Air pollution may also be considered as a potential confounder if days with high sferic densities tended to display higher air pollution levels than days without sferics. Air pollution has been found to be associated with asthma admissions in the UK¹⁴ and elsewhere,¹⁵ although the strength of this association is substantially weaker than the one reported for thunderstorms. Although the authors

point out that the asthma outbreak that occurred in London in June 1994 did not coincide with high air pollution levels, in the current study adjustment for air pollution was not provided. However, since air pollution has been found to increase bronchoconstriction in asthmatic subjects after controlled exposure to allergens,^{16,17} pollutants could in theory have had a double role as both a confounder and a synergistic factor, and this complexity may have made the authors reluctant to include air pollution in the time series models.

Another relevant issue on aetiology is whether the presence of thunderstorms is an indirect or a related variable to other meteorological factors more directly linked to the mechanisms that supposedly trigger asthma attacks. As previously mentioned, in vitro experiments by Suphioglu *et al* have shown that pollen grains are ruptured in rain water by osmotic shock, each grain releasing hundreds of starch granules of a respirable size (<3 µm) which contain Lol p IX.⁴ A measure of osmotic shock may perhaps have been able to reveal a more direct mechanism and thus a larger relative risk. During the London outbreak marked changes were noted in humidity, temperature, and rainfall, and we do not know to what extent inclusion of these variables in the study by Newson *et al* could have provided different results. Future studies based on the same approach are needed to investigate other thunderstorm-associated variables potentially involved in triggering asthma attacks.

The importance of the role of thunderstorms in asthma attacks from a public health perspective was demonstrated by the large outbreak that occurred in London when, in a few hours, many casualty departments were overwhelmed by patients with asthma attacks, some having run out of medicines. For this reason predicting the occurrence of thunderstorms likely to cause asthma outbreaks may be important for public education. Unfortunately, the present study tell us little about the relation between thunderstorms and asthma outbreaks since there was no indication of days with an unusually high occurrence of asthma during the period of the study. However, the occurrence of asthma outbreaks cannot be totally ruled out since the study included only hospital admissions for asthma and not visits to the accident and emergency departments. Most reported asthma outbreaks have shown that a substantial proportion of affected people were able to return home after treatment at the emergency department without needing to be admitted. In the London outbreak only 16% of those seen at the emergency departments were admitted.²

Is the reported association sufficiently relevant to recommend warning systems to predict thunderstorms likely to affect asthmatic subjects and to establish appropriate informative strategies for the patients? Newson and co-authors speculate that, assuming high pollen counts amplify

the high spheric effect as much as the low spheric effect, the simultaneous presence of both high pollen levels and high spheric density may increase the number of asthma admissions by 45% in children and nearly double it in adults. In the National Institutes of Health review on the global strategy for asthma management and prevention published in January 1995 it was recognised that, although adverse weather conditions have been associated with asthma exacerbations, these factors have not been examined systematically and in depth.¹⁸ Taking into account the evidence provided by the researchers in Melbourne, the study by Newson *et al* has provided additional evidence that thunderstorms increase the risk of asthma exacerbations and future reviews should certainly consider this point.

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Review article

Thunderstorm-asthma and pollen allergy

Thunderstorms have been linked to asthma epidemics, especially during the pollen seasons, and there are descriptions of asthma outbreaks associated with thunderstorms, which occurred in several cities, prevalently in Europe (Birmingham and London in the UK and Napoli in Italy) and Australia (Melbourne and Wagga Wagga). Pollen grains can be carried by thunderstorm at ground level, where pollen rupture would be increased with release of allergenic biological aerosols of paucimicronic size, derived from the cytoplasm and which can penetrate deep into lower airways. In other words, there is evidence that under wet conditions or during thunderstorms, pollen grains may, after rupture by osmotic shock, release into the atmosphere part of their content, including respirable, allergen-carrying cytoplasmic starch granules (0.5–2.5 µm) or other paucimicronic components that can reach lower airways inducing asthma reactions in pollinosis patients. The thunderstorm-asthma outbreaks are characterized, at the beginning of thunderstorms by a rapid increase of visits for asthma in general practitioner or hospital emergency departments. Subjects without asthma symptoms, but affected by seasonal rhinitis can experience an asthma attack. No unusual levels of air pollution were noted at the time of the epidemics, but there was a strong association with high atmospheric concentrations of pollen grains such as grasses or other allergenic plant species. However, subjects affected by pollen allergy should be informed about a possible risk of asthma attack at the beginning of a thunderstorm during pollen season.

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There is evidence that thunderstorms can be associated with allergic asthma epidemics in pollinosis patients (1–16) during pollen seasons, and there is argument in favour of the possibility that thunderstorms concentrate at ground level pollen grains that release in atmosphere allergenic particles of respirable size after their rupture by osmotic shock (1, 2).

It has been demonstrated that changes in the weather, such as rain or humidity, may induce hydration of pollen grains and sometimes also their fragmentation, which generates atmospheric biological aerosols carrying allergens (8, 11, 15).

During the first phase of a thunderstorm, pollinosis subjects may inhale a high concentration of allergenic material dispersed in atmosphere, which can induce asthmatic reactions and sometimes even severe ones (2, 6, 12, 13).

Fortunately, although it can induce severe asthma, outbreaks associated with thunderstorms are neither frequent nor responsible for high entity of disease exacerbations (12, 14, 15). However, the mechanisms involved in the release of allergens from pollens during thunderstorm should be known so that pollinosis patients can receive information about the risk of an asthma attack also in subjects affected only by seasonal allergic rhinitis.

Although thunderstorm-associated asthma outbreaks are not frequent, it is possible to observe in clinical practice single cases of patients with deterioration of the allergic respiratory symptoms during a thunderstorm (17, 18) and this possibility should be considered, because the frequency of thunderstorms is recently increased in some geographical areas, particularly in temperate and sub-tropical climate.

Allergenic pollen in the atmosphere

Although representing only a small proportion of the airborne particles present in the atmosphere, pollen grains can be causative agents of allergic respiratory responses in pollen allergic subjects, and pollinosis is now a public health problem (19).

Following Thommen's postulates (20), to be allergenic, the pollen grains

- must contain antigens able to elicit a specific IgE-mediated response in atopic subjects;
- must be produced in high quantities;
- must be buoyant to be carried long distances;
- should be produced by plants that grow in abundance.

Therefore, plants of major importance in pollen allergy are mainly those that rely on the wind as the carrier of the pollen and are defined anemophilous with allergens released by airborne pollens. In practice, only a few species, such as grass, pellitory, ragweed, mugwort, birch, olive, cypress, come into consideration as playing a major role in causing pollinosis, because they release in atmosphere large quantities of pollen during their flowering period.

In the European Community countries between 8% and 35% of young adults show IgE serum antibodies to grass pollen allergens (21) and the cost of pollen allergy in terms of impaired work fitness, sick leave, consulting physicians and drugs, is very high.

During natural pollination, mature pollen grains are dehydrated when they are released by anthers at the dispersal time. Once the pollen grains come into contact with wet surface, they absorb water undergoing rapid metabolic changes jointly with ultrastructural modifications (Fig. 1). The pollinic allergens could be located in the pollen walls and/or in the cytoplasm and are rapidly released when the pollen grains come into contact with the oral, nasal or conjunctival mucosa, thereby inducing the appearance of pollinosis symptoms in sensitized patients (22, 23). Sometimes, the cytoplasmic allergens are in the membrane surrounding starch granules and are released into the atmosphere when the pollen bursts under osmotic shock and can create a respirable allergenic aerosol.

In particular, it has been observed (22) that fresh birch pollen can rupture in high humidity conditions, releasing an aerosol characterized by fragments of pollen cytoplasm ranging in size from 30 μm to 4 μm and containing *Bet v 1* allergen. Taylor et al. (23) observed that about 65% of pollen grains grew a pollen tube up to 300- μm long prior to rupture and released their cytoplasmic



Figure 1. *Parietaria* pollen bursting under osmotic shock with release of cytoplasmic fragments carrying allergens.

content in the high humidity context. The particles released, such as fragmented pollen cytoplasm, form an ultrafine aerosol. The same authors observed that grass anthers should be a site of pollen rupture and a source of fine particulate aerosols that contain pollen allergens (23).

The concentration of allergenic pollen influences the degree of symptoms, but the relationship between allergen exposure, inflammation of airways and clinical symptoms is complex, and factors other than allergens are involved (24). In particular, air pollution may contribute to the asthmatic activity as gaseous air pollution affects airways by inducing inflammation (25–28) and subjects living in urban areas tend to be more affected by plant-derived respiratory disorders than those living in rural areas (29, 30), and pollen allergens release is modified by exposure of pollen to environmental pollution (25–27).

Pollen grains penetrate into the upper respiratory tract but, because of their size, which is always greater than 10 μm of diameter, rarely reach the bronchial regions. However, bronchial asthma and its equivalents, such as irritative cough, are not infrequent in people affected by pollen-induced allergy.

Several hypotheses have been proposed to account for pollinosis-related symptoms in the lower respiratory tract at the bronchial level (19, 27):

- Absorption of allergen in the nose and subsequent transportation to the lower airways.
- Reflex mechanisms inducing bronchial involvement after a nasal reaction.
- Inhalation and penetration of small particles carrying pollen allergen into the lower respiratory region.

The discovery of airborne-allergen-carrying particles much smaller than pollen grains, such as those released by pollens during weather perturbations, particularly thunderstorm and rainfall, provided a possible explanation. In other words, the existence of allergen-carrying airborne particles much smaller than pollen grains (paucimicronic particles) can explain bronchial symptoms affecting subjects during the pollen season or a thunderstorm (1, 6–10) (Table 1).

Thanks to their size; these paucimicronic particles can penetrate deep into the airways inducing asthma in sensitized atopic subjects.

Table 1. Airborne small (paucimicronic) allergen-carrying particles

Starch granules and other components of the cytoplasm of pollen grains, released into the atmosphere under wet conditions, and responsible for thunderstorm-associated bronchial asthma.
Nonpollen plant parts from inflorescences, leaves or Ubisch bodies (spheroidal structures that develop in the anthers of many higher plants and that contain allergens).
Nonplant particulate matter (allergens transferred through physical contact or by leaching from the surface of the pollen grain to other airborne small particles).
Pollen fragments (are not frequently found and can derive from rupture of pollen grains during thunderstorms).

In the context of paucimicronic particles, there are orbicules, small granules (1–5 µm) or droplets developed from anther tissues, loaded with allergens and there is suggestion that they play a role in allergic asthma contributing to form a respirable aerosol during the pollen season (27, 31–33).

Thunderstorms and allergic asthma epidemics in pollinosis subjects during pollen season

Rainfall is usually known to remove pollen from the air but that is not always correct, because studies have revealed that allergens leave the pollen surface almost instantly, usually within seconds, on contact with water.

Thunderstorm-asthma outbreaks have been described in various cities such as Birmingham (UK) (3), London (UK) (7, 10), Melbourne (Australia) (4), Wagga Wagga (Australia) (14) and Naples (Italy) (16), but there are case reports in other cities (17, 18).

One of the first observations regarding thunderstorms and asthma outbreaks was provided by Packe and Ayres (3) at the East Birmingham Hospital, Birmingham, UK, on 6 and 7 July 1983. These authors describe a striking increase in the number of asthma emergency room visit admissions during the hours of a thunderstorm. In a period of 36 h, 26 asthma cases were treated in the emergency room, compared with a daily average of two or three cases in the days preceding the outbreak.

Another asthma outbreak occurred in London coinciding with a heavy thunderstorm on the 24 June 1994, when a large increase was observed in the number of visits for asthma at the emergency departments of London and the southwest of England, and several patients examined, who were not known to be asthmatics or were affected only by seasonal rhinitis, experienced an asthma attack (7, 10).

The epidemic had a sudden onset on 24 June 1994; 640 patients with asthma or other airways disease attended during 30 h from 18.00 on 24 June, nearly 10 times the expected number. Over half (365) the patients were aged 21–40. A history of hay fever was recorded in 403 patients; for 283 patients, this was the first known attack of asthma; a history of chronic obstructive airways disease was recorded in 12 patients. In all, 104 patients were admitted (including 5 to an intensive care unit). A total of 604 patients with wheezing and shortness of breath were seen in several departments, compared with an expected number of 66.6 (7).

The results confirm asthma epidemic, with almost 10 times the usual number of patients presenting during 30 h and an excess of 574 patients attributable to the epidemic (7, 10). The outbreak was not restricted to the London area, although the number of patients presenting to accident and emergency departments on the night of 24 June 1994 was greater in the Thames regions than in other regions in England. Moreover, not all affected

patients attended hospital and this epidemic was the largest outbreak ever recorded.

Other asthma outbreaks during thunderstorms were described in Melbourne, Australia (4), where two large asthma outbreaks coincided with thunderstorms. In addition, these events were followed by a rapid increase in hospital or general practitioner visits for asthma. Taking into account the Melbourne experience, a similar mechanism could have been involved, although other factors may have also contributed.

Other asthma outbreaks occurred in Wagga Wagga (southern Australia) on 30 October 1997 (14) and in Naples, Italy on 4 June 2004 (16).

In Wagga Wagga, 215 asthmatic subjects attended the local emergency department, 41 of whom required admission to hospital. Marks et al. (15) demonstrated that the arrival of a thunderstorm outflow was accompanied by a large increase in the concentration of ruptured pollen grains in ambient air and they observed that in a large region of south eastern Australia, the incidence of excess hospital attendances for asthma during late spring and summer was strongly linked to the occurrence of thunderstorm outflows.

It seems likely that the outflow of cold air associated with a thunderstorm rather than electrical activity, thunder or rain alone, is responsible for the observed event.

During the episode of thunderstorm-associated asthma registered in Naples on 4 June 2004 (between 1.30 and 2.00AM), six adults (three women and three men between 38 and 60 years old) and a girl of 11 years old had attacks of severe bronchial asthma, which was nearly fatal in one case. All patients received treatment in emergency departments and one was admitted to an intensive care unit for very severe bronchial obstruction and acute respiratory insufficiency.

All subjects were outdoors when the thunderstorm struck. The most severe case, a 60-year-old woman sensitized only to *Parietaria* pollen allergens, soon began to show symptoms of intense dyspnoea, which gradually worsened. She was taken to hospital where she was intubated and given high intravenous doses of corticosteroids. She was discharged a few days later. She had previously suffered from seasonal asthma, but had been asthma-free for the past few years and did not need continuous therapy. None of the other six subjects regularly took antiallergic and/or antiasthma drugs. Four had a history of asthma, whereas two had a history of only rhinitis.

We found that all seven patients were sensitized with allergic respiratory symptoms upon exposure to *Parietaria* pollen but were not sensitized to grasses (16).

Parietaria is an urticacea that is widespread in the Naples area with a spring and summer pollen season in part contemporaneous with that of grasses (34). During the thunderstorm, the concentration of airborne *Parietaria* pollen grains was particularly high with a peak of

144 grains/m³ being recorded on June 3. Air pollution levels for both gaseous and particulate components based on the hourly concentrations of nitric dioxide, ozone and respirable particulate matter were not particularly high in Naples on June 3 and 4.

Subjects with sensitization to *Parietaria*, who were indoors in Naples with the windows closed during the night between 3 and 4 June, did not experience asthma attacks. There was evidence that thunderstorm-induced asthma was related to the *Parietaria* pollen allergens. The recertification code for this document is 590588.

Possible mechanisms for thunderstorm asthma

What is most frequently hypothesized is that dry updrafts entrain whole pollens into the high humidity at the cloud base of a thunderstorm where pollens may rupture and cold downdrafts carry pollen fragments to ground level. In other words, at the onset of a thunderstorm, pollen fragments are carried to ground level where outflows distribute them. As a consequence, there is high respirable allergen load in the air.

Taylor et al. (23) hypothesized that the turbulent front of the advancing outflow releases more pollen from flowering grasses, and then may entrain them into the cloud base. Strong electric fields develop in the thunderstorm. Positive ions are released from the ground and attach to particles and electric charge may enhance pollen rupture.

Grass pollens after rupture by osmotic shock during thunderstorms release large amounts of paucimicronic allergenic particles, i.e. cytoplasmic starch granules containing grass allergens. Other pollens, such as those of *Parietaria*, which have no starch granule in the cytoplasm can release other paucimicronic cytoplasmic components carrying allergens. Because of their very small size, these microparticles can penetrate the lower airways inducing the appearance of bronchial allergic symptoms.

Suphioglu et al. (1) showed that rye grass pollen grains contain a large quantity of starch granules coated with the allergens. While levels of chemical air pollution on epidemic days were below or similar to levels in a control period. This observation led to the hypothesis that pollen grains are ruptured in rainwater by osmotic shock, with each grain releasing around 700 starch granules small enough to penetrate the airways and trigger asthma attacks in previously sensitized subjects. Although much remains to be discovered about the relationship between an increase in the number of asthma attacks and thunderstorms, reasonable evidence exists in favour of a casual link between them (35). If this is the case, asthmatics sensitized to pollen allergens may be at risk of suffering asthma attacks during thunderstorms. Depending on the size of the population at risk, thunderstorm-associated asthma outbreaks may threaten the operative capacity of health services, as was the case in London. Therefore, it could be of interest to establish whether some risk factors may predict the occurrence of

asthma outbreaks in such a way that early-warning systems can be developed. With this approach, and using the same data set alluded above, Newson et al. identified 56 asthma epidemics defined as periods of exceptionally high asthma admission counts compared with predictions of a log-linear autoregressive model (11).

However, although thunderstorms and high grass pollen levels preceded asthma epidemics, most thunderstorms, even those coincident with high grass pollen levels in atmosphere, were not followed by asthma epidemics. The authors concluded that using predictions of thunderstorms during periods of high pollen counts as an early-warning system of asthma epidemics would produce too many false alarms to be accepted (12) A more systematic investigation of the relationship between an abrupt increase in the number of asthma attacks and the occurrence of thunderstorms has been provided by Newson et al. (11). Daily numbers of sferic densities, a marker of lightning flashes during thunderstorms, were obtained for each area, and their connection with the number of asthma admissions was evaluated by means of log-linear autoregressive models. Although typical thunderstorms days were not associated with large asthma epidemics, the presence of very large sferic densities was associated with a moderate enhancement in hospital admissions for asthma crisis. High level of sferic densities were associated with a relative risk of approximately 25% in both the 0–14 years and 15 years and over age groups, whereas the excess risk associated with moderate sferic densities was smaller, but statistically significant (35). On the basis of the Melbourne hypothesis, the authors were able to obtain daily grass pollen counts in five regional health authorities and found that high pollen counts for the previous 5 days were associated with amplification of the excess risk associated with thunderstorms.

Unfortunately, the data of Newson et al. (11) do not support the possibility of predicting asthma outbreaks using meteorological data and pollen counts.

Finally, no moulds or viruses were involved in the epidemics.

Conclusions

There is evidence that thunderstorms trigger epidemics of exacerbations of asthma during the pollen season by washing down pollen grains and concentrating them in a band of air at ground level. Pollen grains may, after contact with rain or humidity, release part of their cytoplasmic content, including respirable, allergen-carrying paucimicronic particles.

Cytoplasmic granules of small size are released from the pollen grains by contact with rainwater. As a consequence, at the onset of a thunderstorm, there is a high respirable allergen load in the air and these conditions might expose susceptible subjects to a high increase of pollen allergens in the atmosphere.

Thunderstorm-associated asthma is a dramatic example of the allergenic potential of pollen antigens. Pollen-allergic patients who encounter the allergenic cloud would be more susceptible to undergo an asthma attack.

Subjects allergic to pollen, who are in the path of the thunderstorm outflow, are likely to inhale airborne pollen allergens and to experience an airway asthmatic response.

The characteristics of described epidemics of thunderstorm-associated asthma can be summarized as follows:

- There is a link between asthma epidemics and thunderstorm.
- The epidemics related to thunderstorm are limited to seasons when there are high atmospheric concentrations of airborne allergenic pollens.
- There is a close temporal association between the start of the thunderstorm and the onset of epidemics.
- There are not high levels of gaseous and particulate components of air pollution.

- Subjects with pollen allergy, who stay indoors with window closed during thunderstorm, are not involved.
- There is a major risk for the subjects who are not under antiasthma correct treatment, but subjects with allergic rhinitis and without previous asthma can experience severe bronchoconstriction.

In the light of the above, subjects affected by pollen allergy should be alert to the danger of being outdoors during a thunderstorm in the pollen season, as such events may be an important cause of severe exacerbations of asthma

In other words, when asthmatic patients realize that a thunderstorm is approaching the best thing for them to do is to stay indoors, with windows closed.

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