

A major outbreak of asthma associated with a thunderstorm: experience of accident and emergency departments and patients' characteristics

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Abstract

Objective—To investigate the time course of an epidemic of asthma after a thunderstorm, characteristics of patients affected, and the demand on emergency medical resources.

Design—Study of registers and records in accident and emergency departments and questionnaire to staff.

Setting—London area.

Subjects—All patients presenting at 12 accident and emergency departments with asthma or other airways disease.

Main outcome measures—Numbers of patients, clinical features, information on shortage of resources—equipment, drugs, and staff.

Results—The epidemic had a sudden onset on 24 June 1994; 640 patients with asthma or other airways disease attended during 30 hours from 1800 on 24 June, nearly 10 times the expected number. Over half (365) the patients were aged 21 to 40 years. 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 five to an intensive care unit). Several departments ran out of equipment or drugs, called in additional doctors, or both.

Conclusions—This study supports the view that this epidemic was larger than previously reported epidemics and the hypothesis that "thunderstorm associated asthma" is related to aeroallergens. Demands on resources were considerable; a larger proportion of patients needing intensive care would have caused greater problems.

Introduction

During the night of Friday 24 June 1994 accident and emergency departments in London experienced an epidemic of patients with asthma after a severe thunderstorm. Localised outbreaks of asthma after thunderstorms have been reported previously,¹⁻⁴ but this epidemic was striking because of the large number of patients affected and the wide area in which it occurred.⁵ We report the first systematic study of this episode of "thunderstorm associated asthma."

We aimed to delineate the time course of the epidemic, characterise the patients affected by the epidemic, and assess the demand on emergency medical resources. We carried out the study in 12 accident and emergency departments in London—three in North Thames (West), four in North Thames (East), two in South Thames (West), and three in South Thames (East). The 12 departments served an area with a population of about 2 735 000 and constituted about

one third of the accident and emergency departments in Greater London.

Method

We used registers to identify patients and inspected clinical records as necessary. We recorded the number of patients with asthma or other airways disease presenting daily between 27 May and 23 July 1994. We included all patients presenting with asthma, wheeze, or hay fever or allergy with wheeze or difficulty in breathing and all patients requesting an inhaler or asthma treatment. We included patients complaining of shortness of breath, difficulty in breathing, cough, or chest infection if the clinical record showed that their symptoms were due to airways disease. We defined the epidemic as being from 1800 on 24 June to 2400 on 25 June 1994. For each patient seen during the epidemic we completed a questionnaire about demography, clinical features, and outcome. We asked participating departments to complete a questionnaire about the demand on equipment, drugs, and staff during 24 to 25 June 1994.

The observed number of patients during the epidemic was compared with the estimated expected number of patients derived from six other Fridays and Saturdays in June and July. A Friedman two way analysis of variance showed no significant difference between the six control time periods ($P=0.616$). Wilcoxon's matched pairs signed ranks test was used to compare the number of patients attending during the epidemic with the number attending on one control period two weeks previously. (The number attending on the control Friday and Saturday, being a figure for 48 hours, was multiplied by 5/8 to give the number of patients expected to attend over a comparable 30 hour period.)

To investigate further the cause of the epidemic, we obtained information about aeroallergens from the Pollen Research Unit. Air samples were taken by a volumetric spore trap on the roof of the five storey building of the University of North London.

Results

During the epidemic 640 patients attended the accident and emergency departments, compared with an expected number of 66 ($P=0.003$). The number of patients attending increased suddenly after 2200 on 24 June; 373 presented within eight hours between 2200 on 24 June and 0559 on 25 June (fig 1). The increase in attendances that began on the night of 24 June had subsided by 26 June (fig 2). The records of about 57 patients could not be found.

The 640 patients (368 male, 272 female) seen during

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BMJ 1996;312:601-4

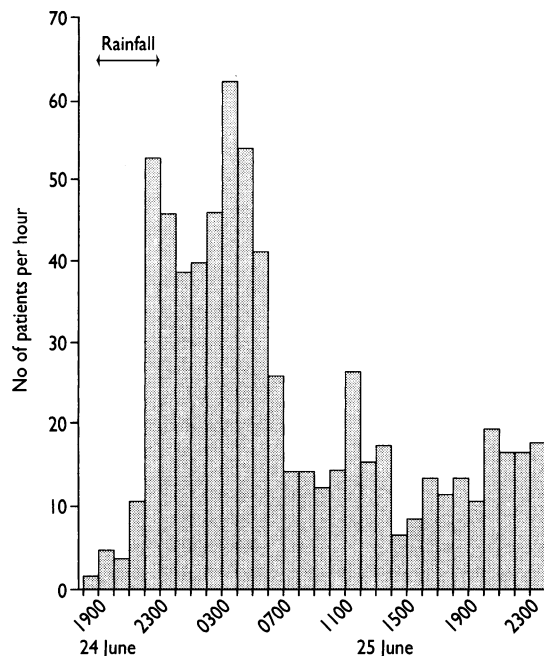


Fig 1—Number of patients presenting with asthma or other airways disease to 12 accident and emergency departments between 1800 on 24 June and 2400 on 25 June 1994 and approximate time of rainfall

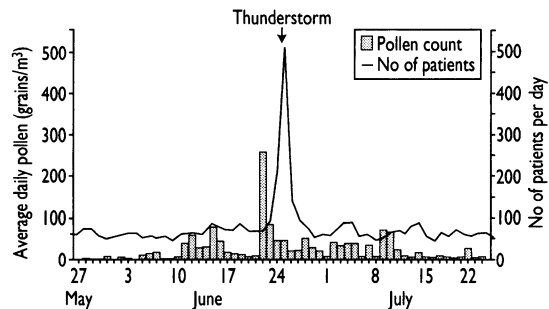


Fig 2—Number of patients presenting with asthma or other airways disease to 12 accident and emergency departments and daily average grass pollen count from 27 May to 23 July 1994

the epidemic had a mean age of 32 years; 365 were aged between 21 and 40 years, and 86 were aged under 16. In all, 403 patients had a history of hay fever, and for 283 this was the first recorded attack of asthma; a history of chronic obstructive airways disease was noted in 12 (table 1). Data about previous treatment in patients known to be asthmatic were available from seven out of 12 departments: of 209 such patients 40 were recorded as having a steroid inhaler and 103 a β_2 agonist. A pre-treatment peak flow was recorded in 499 of 600 patients aged over 8: the mean peak flow was 221 l/min. In all, 507 patients were treated with a β_2 agonist, and 224 given a steroid.

Altogether, 104 patients were admitted, 500 were discharged home, and 31 did not wait to be seen; data about five patients were unknown. Five patients were admitted to an intensive care unit, where none died; the mean duration of hospital stay for all those admitted was three days. Of the 531 patients not admitted, 15 returned to the same accident and emergency department for recurrence of symptoms within 48 hours. A prescription for oral steroids to be taken at home was documented for 197 of those discharged from an accident and emergency department, although some form of treatment was given to 379 of those discharged.

Eleven out of 12 departments supplied data about the resource implications of the epidemic. Five departments ran out of nebuliser face masks, one out of

mouthpieces for peak flow meters, four out of β_2 agonist nebulisers, six out of β_2 agonist inhalers, and eight out of prednisolone tablets. Five departments needed additional supplies of oxygen. Six departments called in additional medical staff (either an accident and emergency doctor from home or the on call medical team to see patients who had not been referred), and one brought in extra nurses.

Accident and emergency departments adopted various measures to deal with the unexpected pressure on resources. β_2 Agonist nebulised with oxygen from portable cylinders was given to patients who were sometimes two to a cubicle or seated in the corridor. Equipment and drugs were borrowed from wards; pharmacists were called in to restock drugs; and supplies of prednisolone were conserved, patients being given a single dose in the accident and emergency department and a prescription to be dispensed the following morning.

Figure 2 shows daily grass pollen counts in London during the eight week period. The daily average pollen count was exceptionally high for London (258 grains/m³) two days before the epidemic, and the concentration remained high until it rained on the evening of 24 June. Concentrations of allergenic fungi were not exceptionally high in the London area before, during, or immediately after the epidemic, although levels of damp air spores such as ascospores of *Phaeosporia nigra* and *Diatrypaceae* rose during and after the thunderstorm. The concentration of respirable particulate matter was unusually high all day on 24 June before the storm; it rose to a peak at 2000, coinciding with the evening peak pollen concentration. The peak concentration of the <2.9 μ fraction of particulate matter was 130 916/m³, more than 40 times previously high values during the 1994 grass pollen season (which did not exceed 3000/m³).

Discussion

SCALE OF EPIDEMIC

The results confirm a true epidemic, with almost 10 times the usual number of patients presenting during 30 hours and an excess of 574 patients attributable to the epidemic. The outbreak was not restricted to the London area,^{5,7} 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.⁷ Furthermore, not all affected patients attended hospital.⁸ It therefore seems that this epidemic was substantially larger than previously reported epidemics of thunderstorm associated asthma.⁵

Although potential sources of error are present in the study, we believe that they are unlikely to affect this conclusion. Firstly, the number of accident and emergency records not found was relatively small. Secondly, we believe that the criteria for identifying affected patients were sensitive and specific. In one study a presenting complaint of asthma was 80% sensitive and 98% specific for asthmatic patients in accident and emergency departments outside the epidemic,⁹ but this complaint was of low sensitivity during the epidemic. To ensure sensitivity we identified

Table 1—Clinical features of 640 patients presenting to the accident and emergency departments during asthma epidemic. Values are numbers of patients

	Yes	No	Unknown
Asthma	294	283	63
Hay fever	403	78	159
Chronic obstructive airways disease	12	556	72

patients with complaints such as shortness of breath, and to maintain specificity we checked patients' records. The number of patients with chronic obstructive airways disease was less than 2% of the total 640. Thirdly, calculating the expected number of attendances due to asthma for the 30 hours of the epidemic by multiplying the 48 hour figure for a control Friday and Saturday by 5/8 could result in overestimating the difference between epidemic and control if there is diurnal variation in attendances due to asthma in accident and emergency departments. We believe that this is not significant in practice, given the magnitude of the difference between the two weekends. For 11 departments we were able to determine directly the attendances during the control 30 hours (1800 on Friday to 2400 on Saturday); there were nine to 10 times more attendances during the epidemic than during the control hours (602 v 64; P=0.004). If the 640 patients attending the 12 departments during the 30 hour epidemic are compared with attendances during the whole 48 hours of the control Friday and Saturday there were still more than six times as many (640 v 105; P=0.003).

PATIENTS' CHARACTERISTICS, AEROALLERGENS, AND WEATHER

Patients during the epidemic were characteristically young atopic adults; most of those with a history of asthma probably did not have steroid inhaler prophylaxis. A high prevalence of atopy is a feature of similar outbreaks^{2-4,10} and is consistent with the hypothesis that thunderstorm associated asthma is related to aeroallergens and the effect of weather.¹¹ Many patients complained that they had been troubled by hay fever symptoms on the days before the thunderstorm, an observation recorded in the Birmingham outbreak.¹⁰ We have data that suggest that grass pollen was the most significant aeroallergen in the period before the storm. The daily average grass pollen count on 22 June was the highest recorded in London for six years. Pollen released during the days before the thunderstorm would have been deposited on surfaces in the city and could have been resuspended by gusting winds. The relevance of the high concentration of respirable particulate matter is uncertain, but the high concentration of pollen and small particulate matter may have had a synergistic effect. From our data it seems unlikely that fungal spores were a factor in the epidemic; a rise in the concentration of certain ascospores after the storm was noted elsewhere.⁶

The abrupt onset of the epidemic is consistent with an effect of the thunderstorm, which travelled from south west to north east across London. A sudden fall in temperature and a pronounced increase in wind speed preceded the rain, which began to fall over London between 1900 and 2000, was most intense between 2100 and 2200, and ceased between 2200 and 2300 (fig 1). The key feature of this type of storm (mesoscale convective system) is the "outflow region" preceding the advancing rainstorm, characterised by high wind speeds and powerful vertical air currents (meteorological data from Professor Collier of the Meteorological Office, now of the University of Salford, personal communication). Asthma may have been triggered by a dramatic rise in aeroallergens resulting from one or more effects. Firstly, gusting winds associated with the thunderstorm outflow may have resuspended residual pollen locally. Secondly, albeit less likely, aeroallergens may have been carried up in the rapid uplift of air associated with convective storms,¹² carried horizontally with the storm, and then redeposited by a cold downdraft ahead of the rainstorm (Professor Collier, personal communication). Thirdly, rainfall itself or rapid changes of humidity, or both, may lead to a rapid rise in respirable allergens, which

Key messages

- Under certain conditions an epidemic of asthma may be triggered by a thunderstorm
- People affected by thunderstorm associated asthma are characteristically young atopic adults who either are not known to have previously had asthma or are asthmatic but probably do not have steroid inhaler prophylaxis
- Thunderstorm associated asthma seems to have had a benign course in most patients in this epidemic, but many patients presenting to accident and emergency departments should not induce complacency in the treatment of acute asthma
- This kind of "major incident" puts a strain on emergency medical services in a way not usually anticipated because all hospitals in an area may be affected

might have precipitated bronchial narrowing in susceptible individuals.¹³⁻¹⁵ These mechanisms are being investigated further.

A history of hay fever may also have rendered the patients in this epidemic vulnerable to a variety of non-allergic stimuli. Exposure to allergen may cause an increase in non-specific bronchial reactivity, the magnitude and duration of which is proportional to the late asthmatic response.^{16,17} If recent exposure to allergens had increased bronchial responsiveness, acute airway narrowing might then have been triggered by a variety of precipitants—the fall in temperature and the increase in wind speed and electrically charged particles, as well as in inhaled particles and allergens carried in the downdraft of the storm. Almost half of the patients were not known to have previously had asthma, and our results suggest that most of those with a history of asthma did not have a steroid inhaler; these groups may therefore have been particularly vulnerable to the effect of the thunderstorm.

EMERGENCY MEDICAL RESOURCES

None of the 12 hospitals invoked their major incident plan, although the number of additional patients in some accident and emergency departments was as great as might be expected from an accident causing mass casualties. There may have been a delay in appreciating that extraordinary measures were appropriate because the influx of patients resulted from a "medical incident" rather than a major accident. In the event most patients were not severely affected, with only 16% needing admission and less than 1% needing intensive care. This has not always been the case in epidemics of asthma.¹⁸ A smaller total number of patients with a higher proportion needing intensive care would have posed a greater problem. In a major incident a hospital's capacity to deal with patients needing resuscitation, particularly artificial ventilation, may be the limiting factor. Once a designated hospital is saturated, patients are diverted to supporting hospitals; but this option would not be available in a large epidemic of asthma if all hospitals in an area were affected.

We thank Heather Cross and Maureen O'Sullivan for help with computing.

Funding: No special funding.

Conflict of interest: None.

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(Accepted 16 January 1996)

Thunderstorm associated asthma: a detailed analysis of environmental factors

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See editorial and p 601

Abstract

Objectives—To seek associations between meteorological factors, concentrations of air pollutants or pollen, and an asthma epidemic which occurred in London on 24 and 25 June 1994 after a thunderstorm.

Design—Retrospective study of patients' accident and emergency department records, with bivariate and multivariate analysis of environmental factors and data collection for the two months surrounding the epidemic.

Setting—The accident and emergency department of St Mary's Hospital in west central London.

Subjects—148 patients presenting with asthma between 1 June and 31 July 1994, of whom 40 presented in the 24 hours after the storm.

Results—The asthma epidemic was significantly associated with a drop in air temperature six hours previously and a high grass pollen concentration nine hours previously. Non-epidemic asthma was significantly associated with lightning strikes, increase in humidity or sulphur dioxide concentration, a drop in temperature or high rainfall the previous day, and a decrease in maximum air pressure or changes in grass pollen counts over the previous two days.

Conclusions—New episodes of asthma during the epidemic on 24 and 25 June 1994 were associated with a fall in air temperature and a rise in grass pollen concentration. Non-epidemic asthma was significantly associated with a greater number of environmental changes. This may indicate that the patients with thunderstorm associated asthma were a separate population, sensitive to different environmental stimuli.

Introduction

On 24 and 25 June 1994 an acute outbreak of asthma occurred in southern England associated with a thunderstorm.^{1,2} The abrupt rise in the number of patients presenting with asthma to the accident and emergency department of St Mary's Hospital in west central London provided an opportunity to assess whether there had been any precipitating environmental factors. Data on meteorological changes and local concentrations of air pollutants and grass pollen were collated for the two months surrounding the epidemic to see whether these factors were temporally

associated with cases of non-epidemic asthma presenting during the study period or with the asthma epidemic itself.

Patients and methods

We analysed the records of patients who had presented with asthma to the accident and emergency department between 1 June and 31 July 1994. Patients aged 16 or over who were assessed by a doctor and given a diagnosis of asthma were included. Patients who refused proper assessment or in whom the diagnosis was unclear were excluded. Only one patient reattended with asthma during the study period, and this was before the epidemic.

Three hourly measurements of rainfall, ambient temperature, barometric pressure, and humidity were obtained from the Meteorological Office. Measurements were taken at the London Weather Centre, Holborn, roughly 4 km from St Mary's Hospital. Hourly measurements of ground lightning strikes were obtained from EA Technologies, Capenhurst, Chester, which used vector analysis of current detected by local surface electrodes. Vector analysis covered a 10 km radius centred on St Mary's Hospital. Daily hourly maximum concentrations of the air pollutants nitrogen dioxide, sulphur dioxide, and ozone were obtained from Westminster Council. Hourly measurements of these variables were obtained for 24 and 25 June. Measurements were taken from Marylebone Road, roughly 1.5 km from St Mary's Hospital. Daily grass pollen counts with two hourly measurements for 24 and 25 June were obtained from the Pollen Research Unit, University of North London. Measurements were taken from the University of North London building in Holloway, roughly 6 km from St Mary's Hospital. Fungal spore concentrations were not available.

STATISTICAL ANALYSIS

Over the two month study period daily measurements were obtained either as totals for the 24 hours (asthma presentations, lightning strikes, rainfall), as maximum values for the 24 hours (air pollutant and grass pollen concentrations), or as the maximum changes that occurred during any three hour period during the day (temperature, pressure, humidity). More detailed three hourly environmental data were obtained for the period surrounding the thunderstorm

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BMJ 1996;312:604-7