

THUNDERSTORM-ASSOCIATED BRONCHIAL ASTHMA: A FORGOTTEN BUT VERY PRESENT EPIDEMIC

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ترتبط نوبات الربو الحادة ببعض مسببات كملوثات الهواء والأحوال الجوية كالعواصف وتشير الدلائل إلى أن حالات الربو المرتبطة بالعواصف تشكل مجموعة خاصة من المصابين و أن أوبئة من هذا النوع تم رصدها ولكن ليس في المملكة العربية السعودية. السبب المباشر لهذه المراجعة لأدبيات هذا النوع من الربو كان الوباء الذي وقع في نوفمبر من العام 2002م بالمنطقة الشرقية بالمملكة العربية السعودية حيث تمت معالجة أغلب الحالات في مستشفى الملك فهد الجامعي بالخبر بفعالية وكذلك المستشفيات المجاورة. وترتبط ثلاثة عوامل بحالات الربو الناتجة عن العواصف : الملوثات البيولوجية أو الكيميائية أو الأحوال الجوية. وتضم الملوثات البيولوجية المحمولة بهواء العواصف التي تتسبب في إحداث حالات الربو. وتشمل الملوثات الكيميائية الغازات المنبعثة من المحميات الزراعية، المعادن الثقيلة، الأوزون، وثاني أكسيد النيتروجين، وثاني أكسيد الكبريت، والأدخنة المنبعثة من الكمان والذرات الدقيقة. وترتبط هذه العوامل بالربو من خلال تكون أحماض الكبريت والنيتروجين . وترتبط جوانح الربو غير الوبائي بالأمطار الغزيرة، وانخفاض درجات الحرارة والضغط، وضربات الصواعق وارتفاع معدلات الرطوبة ويمكن أن تحدث كلها أثناء العواصف مما يجعلها ترتبط بحدوث وباء الربو. عادة ما يكون المصابون بالربو المرتبط بالعواصف من البالغين في عمر الشباب والمصابين بالتحسس ولا يتناولون الكورتيزون المستنشق وغالباً ما تكون أولى إصابتهم أثناء هذا الوباء. كما يتعرض الأشخاص ذوي الحساسية لحبوب اللقاح الذين يتواجدون أثناء العاصفة لاستنشاق الهواء المحمل بحبوب اللقاح الذي يتسبب بإصابتهم بنوبة الربو. على الأطباء الإلمام بهذه الظاهرة و احتمالات ظهور جائحة الربو أثناء الأمطار الغزيرة. وعلى العاملين في وحدات الطوارئ والعناية المركزة توقع أعداد كبيرة من الحالات ومن ثم يحضرون أجهزة التنفس الصناعي والاحتياطات الأخرى للإنعاش القلبي الرئوي . وعلى الطاقم الصحي التعاطي مع هذه الظاهرة بأسلوب علمي في المستقبل و يتطلب هذا العمل كفريق.

مفاتيح الكلمات: الربو المرتبط بالعاصفة، الوباء

Acute episodes of bronchial asthma are associated with specific etiological factors such as air pollutants and meteorological conditions including thunderstorms. Evidence suggests that thunderstorm-associated asthma (TAA) may be a distinct subset of asthmatics, and, epidemics have been reported, but none from Saudi Arabia.

The trigger for this review was the TAA epidemic in November 2002, Eastern Saudi Arabia. The bulk of patients were seen in the King Fahd Hospital of the University, Al-Khobar. The steady influx of acute cases were managed effectively and involved all neighboring hospitals, without evoking any "Major Incident Plan".

Three groups of factors are implicated as causes of TAA: pollutants (aerobiologic or chemical) and meteorological conditions. Aerobiological pollutants include air-borne allergens: pollen and spores of molds. Their asthma-inducing effect is augmented during thunderstorms.

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Chemical pollutants include greenhouse gases, heavy metals, ozone, nitrogen dioxide, sulfur dioxide, fumes from engines and particulate matter. Their relation to rain-associated asthma is mediated by sulfuric and nitric acid.

Outbreaks of non-epidemic asthma are associated with high rainfall, drop in maximum air temperature and pressure, lightning strikes and increased humidity. Thunderstorm can cause all of these and it seems to be related to the onset of asthma epidemic.

Patients in epidemics of TAA are usually young atopic adults not on prophylaxis steroid inhalers. The epidemic is usually their first known attack. These features are consistent with the hypothesis that TAA is related to both aero-allergens and weather effects. Subjects allergic to pollen who are in the path of thunderstorm can inhale air loaded with pollen allergen and so have acute asthmatic response. TAA runs a benign course

Doctors should be aware of this phenomenon and the potential outbreak of asthma during heavy rains. A & E departments and ICU should be alert for possible rush of asthmatic admissions and reinforce ventilators and requirements of cardio-pulmonary resuscitation. Scientific approach should be adopted to investigate such outbreaks in the future and must include meteorological, bio-aerosole pollutants and chemical pollutant assessment. Regional team work is mandatory.

Key Words: *Thunderstorm-associated asthma, epidemic.*

The prevalence of bronchial asthma is increasing and its etiology remains obscure, but acute episodes have been associated with specific etiologic factors. These include air pollutants such as ozone, nitrogen dioxide, sulfur dioxide and other chemicals, as well as particulate matter of respirable diameter (e.g. fog, organic dust, and aero-biological products). It is difficult to standardize "measures of exposure". Accordingly, researchers employ ecological designs to seek associations between, on one hand, "exposure" measured in terms of the environmental factors and, on the other hand, "outcome" measured as individuals' asthma attacks or their utilization of health care facilities. In spite of the volume of research done, the assertion that air pollution causes asthma remains controversial.¹

Some reports suggest a "point source" of environmental causative agents. An example is the Barcelona asthma epidemic which was linked to soya bean dust.² Other reports of asthma epidemics have implicated meteorological conditions such as thunderstorms and sudden changes in atmospheric temperature or pressure.³⁻⁵ Factors associated with non-epidemic asthma differ from those associated with the epidemic form. This suggests that patients attending Accidents and Emergencies (A & E) departments with reversible airway obstruction related to rain ("Thunderstorm-associated asthma"), may be a distinct sub-set of asthmatics who are more sensitive to environmental stimuli.⁶ Indeed, epidemics of thunderstorm-associated asthma

have been documented in the literature, but no Saudi experience has been reported.

Throughout Saturday, 2 November 2002, an epidemic of acute episodes of asthma after a heavy downpour of rain was observed by the A & E Department, King Fahd Hospital of the University (KFHU), Al-Khobar, Eastern Province. It has been observed that the number of asthmatics requiring emergency room services increases during and following rains or thunderstorms. The KFHU epidemic, however, was striking for two reasons: the number of patients affected was large, and, the geographic area involved was wide. Thus, on that day, other hospitals in the Eastern Province reported a similar pattern of cases.

None of the treating hospitals in the region evoked their "Major Incident Plan" although the number of additional patients in some of these hospitals was big enough to trigger a mass casualty response. The "Major Incident Plan" is also termed Mass Casualty Plan or Disaster Plan. This may be explained by at least two possibilities. First, there may have been a delay in appreciating and accepting that extraordinary measures were necessary because the influx of patients had resulted from a "medical incident" rather than a major accident. Secondly, most patients were not severely affected. The majority were treated and discharged home; only a few required admissions, and less than 1% needed to be in the Intensive Care Unit (ICU).

However, if a higher proportion of those admitted had needed to be in the ICU, there would have been serious logistical problems. This is because in major incidents, the hospital's capacity to handle patients requiring resuscitation and artificial ventilation is a limiting factor. Once a designated hospital becomes saturated, patients are diverted to supporting hospitals in the same region. Thus, should all hospitals in an area be affected simultaneously in asthma epidemics, even greater logistical and management problems would ensue.

POSSIBLE AETIOLOGY OF THUNDER-STORM-ASSOCIATED ASTHMA (TAA)

Three groups of factors have been suggested as causes of TAA, but it is not yet known which ones play the major role. The groups are (1) Aero-biological pollutants, (2) Chemical pollutants and (3) Meteorological conditions.

1. Aero-biological pollutants

A sudden rise in bio-aerosol concentrations has been observed during thunderstorms. Recent studies of the kinetics of the release of aero-allergens (air-borne allergens) and their effects on patients have confirmed the presence of high concentrations of pollen grains and mold spores in the surrounding atmosphere.

Pollen is a fine powdery substance produced by flowers. Insect and natural agents such as air, rain and storm transfer pollen from one plant to another for pollination. Pollen is responsible for most asthmatic attacks in flowering seasons. Produced in large quantities, pollen is of respirable size²⁻¹⁰ and, therefore, enters the airways. It is readily carried long distances by winds and therefore affects individuals miles away from source. The maximal concentration in the atmosphere occurs before sunrise and after sunset.

Molds are plant species which grow in damp areas with high humidity. They are microscopic, reproduced by the release of millions of spores into the air which then settle on organic matter and grow. Inhaled air-borne spores can cause asthmatic attacks.

Raised concentrations of bio-aerosol during thunderstorm is attributed to sudden onset of high winds which trigger the sudden release of spores and pollen into the atmosphere. This is probably responsible for asthma epidemics. Winds

associated with thunderstorms may have re-suspended residual pollen locally. It is conceivable that aero-allergens are carried, firstly, up in the rapid up-lift of air associated with connective storms,⁷ and secondly, horizontally with the storm. They are then re-deposited by a cold downdraft ahead of the rainstorm. Lastly, rainfall itself causes rapid changes of humidity or temperature or both. This may lead to a rapid rise in respirable allergens. Exposure to allergen can increase non-specific bronchial reactivity; the magnitude and duration are proportional to the last asthmatic response.^{8,9} If recent exposure to allergens increased bronchial responsiveness, acute airway narrowing might then be triggered by a variety of precipitants.

2. Chemical Pollutants

Air pollution, a side effect of global industrialization and urbanization, has become a fact in today's world. The earth is a closed system; nothing gets out. Today's greenhouse gases, heavy metals and other compounds, accumulate as they are produced in greater quantities than nature can absorb. It is like smoking in a closed room. Today's world harbors millions of people and machines. Gas, diesel engines and industrial pollutants result in a haze of smog hanging over most major cities around the world.

Ozone, nitrogen dioxide, sulfur dioxide and particulate matter are the main forms of air pollution of the out-doors. These oxidizing gases are formed as by-products of combustion and can cause airway inflammation in humans whether or not they have asthma. Since the level of ozone and pollutants increases and temperatures also increase, the phenomenon of global warming threatens to make this a continuing problem.

An average human consumes 12 kg of air per day to form carbon dioxide. It is about twelve times higher than the food we take. Hence, even small concentrations of pollutants in the air become more significant in comparison to similar levels present in food. Nearly 15 kg of air is consumed to burn one liter of fuel in automobiles. Carbon monoxide, lead and hydrocarbons are emitted in high quantities in petrol combustion. These can cause the loss of visual accuracy and mental alertness. Diesel combustion emits considerably higher amounts of nitrogen dioxide, particulate matter and sulfur dioxide. Pollutants

of diesel combustion are important trigger factors which aggravate asthma. A consensus on research studies indicate that air pollutants cannot induce asthma in a person who is not predisposed to it (except ozone effect), but they can trigger an acute attack in asthmatic patients, and can cause other lung diseases such as chronic bronchitis in healthy, non-asthmatic persons.

The relation of Chemical Pollutants to Rain-associated Asthma

Air pollution is a major cause of acidic deposition or acid rain as it is commonly known. It occurs when emissions of sulfur dioxide (SO₂) and nitrogen dioxide mix in the atmosphere with water, oxygen and oxidants to form mild solutions of sulfuric and nitric acid. Sunlight increases the rate of these reactions. These compounds then fall to the earth in the wet forms of rain, snow, fog or in dry forms as gas and particles. About half of the acidity in the atmosphere falls back to earth through these dry depositions which the wind blows into buildings, cars and homes. Prevailing winds transport these compounds, hundreds of miles across countries and national borders, resulting in a significant negative impact on health.

3. Meteorological Conditions

Climate variation has only marginal effect on normal persons. However, for asthmatic patients who are prone to dust, humidity, high temperature and changes in atmospheric pressure, bivariate and multivariate analyses have shown significant association between lightning strikes and asthma presentations. Celenze et al, using multivariate analysis of environmental factors, showed that between one and nine lightning strikes a day was associated with increased asthma presentations by a factor of 2.21 compared with when there was no lightning (P<0.05).

Bivariate and multivariate analyses of other meteorological variables suggest that there are significant associations of increased cases of non-epidemic asthma with the following four variables: high rainfall, drop in maximum air temperature, fall in air pressure and increased humidity. A sudden thunderstorm causes the greatest sudden fall in temperature and air pressure as well as the most pronounced increase in humidity, rainfall, and lightning strikes. Hence,

it seems to be related to the onset of asthma epidemic.

WHO IS AT RISK OF TAA?

During the epidemic of thunderstorm-associated asthma (TAA), characteristically, the patients were young atopic adults. A high prevalence of atopy was also a feature of similar outbreaks.¹⁰ In a significant number of the patients, the episode was the first known attack of asthma. Most gave a history of asthma, but were probably not on prophylaxis with steroid inhalers. A history of hay fever and allergy to regrass were found to be strong predictors for asthma exacerbation during thunderstorms.¹¹ These are consistent with the hypothesis that TAA is related to aero-allergens as well as the effect of weather. Subjects allergic to pollen who are in the path of thunderstorm are likely to inhale air which is heavily loaded with pollen allergen and consequently experience an acute airway asthmatic response.

TAA seems to have had a benign course in most patients in the epidemic alluded to, as they did not require hospital admission. However, there should be no complacency in the treatment of many patients presenting to A & E departments with acute asthma.

RECOMMENDATIONS

1. Doctors should be aware of this phenomenon, which may not however, merit the "asthma weather alerts." Adding fuel to the flames of the tabloid press is counter-productive to developing a wider understanding of asthma and its prevention.
2. Hospital accident and emergency departments in the area should be aware of the potential outbreak of asthma during heavy rains, and take measures to set up departments with the necessary equipment.
3. Physicians in Intensive Care Units should be on high alert for possible rush of admissions and demand for ventilators, and cardio-pulmonary resuscitation.
4. A systematic, organized scientific approach and collaborative effort should be adopted in the future to investigate factors relating to such outbreaks involving meteorological, bio-aerosole pollutants and chemical pollutants.

REFERENCES

1. Barnes P. Air pollution and asthma. *Postgraduate Med* 1994;70:319-24.
2. Anto JM, Sunyer J. A point source asthma outbreak, *Lancet* 1986; ii:900-3.
3. Morrison I. It happened one night. *Med J Aust* 1960; 47:850-2.
4. Logan WPD. Mortality in the London fog incident, 1952, *Lancet* 1953; i:336-8.
5. Anto JM, Sanyer J, Rodriguez-Roisin R, Suarez-cervera M, Vazquez L. Community outbreaks of asthma associated with inhalation of Soybean dust. *N Engl J Med* 1989;320:1097-102.
6. Antonio Celenza, Forthergill J, Kupek E, Shaw RJ. Thunderstorm associated asthma: a detailed analysis of environmental factors. *BMJ* 1996; 312:604-7.
7. Norris-Hill J, Emberlin J. The incidence of increased pollen concentrations during rainfall in the air of London. *Aerobiologia* 1993; 9:27-32.
8. Cartier A, Thomso NC, Frith PA, Roberts R, Hargreave FE. Allergen-induced increase in bronchial responsiveness to histamine: relationship to the late asthmatic response and change in airway caliber. *J Allergy Clin Immunol* 1982; 70:170-7.
9. Cockroft DW. Mechanism of perennial allergic asthma. *Lancet* 1983; ii:253-6.
10. Packe GE, Ayres JG. Aeroallergen skin sensitivity in patient with sever asthma during a thunderstorm. *Lancet* 1986; i: 850-1.
11. Girgis ST, Mrks GB, Downs SH. Thunderstorm associated asthma in an inland town in South-eastern Australia who is at risk? *Eur Respir J* 2000; 16:3-8.