

Sudden death in asthma

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ABSTRACT Two deaths after sudden severe asthma attacks in young people are reported from a clinic set up to identify and manage “at risk” patients. These deaths occurred despite frequent visits at which recommendations made by previous studies were implemented. The risk factors and management of such episodes have been reviewed. Precautions taken proved inadequate due to the severe, abrupt nature of the attacks, failure of the patients’ immediate treatment, and delay in reaching hospital. Consideration should be given to the self-administration of subcutaneous adrenaline or specific beta-agonists, the provision of a detailed medical card, and free access to the nearest hospital in such cases.

Until the present century asthma was considered a relatively mild illness with a normal or even long life expectancy. Laennec, Osler (Silverglade, 1971), and Trousseau (Earle, 1953) have all been quoted as holding this belief. The first death from asthma with necropsy findings to be recorded in the English literature was in 1945 (Thomson, 1945). During the 1960s there was concern and controversy over the association of increased asthma mortality with the rise in sales of pressurised aerosol bronchodilators (Inman and Adelstein, 1969). Since then considerable attention has been focused on asthma deaths. These deaths often occur in young people, are unexpected, but even so may be preventable. The fatal attack often develops rapidly, and death ensues before the administration of adequate treatment. Recommendations for the prevention of these episodes include: recognition of “at risk” patients, patient and doctor education, specialised units with a self-admission service, objective assessment of the severity of the attack, and appropriate drug treatment.

During the past 18 months we have attempted to implement the above recommendations but sadly report two cases of sudden death from asthma during this period, which we have been able to examine critically for the factors leading to their deaths.

Case histories

CASE 1

A 22-year-old female drama student had a long history of asthma and eczema dating from early

childhood. Throughout her life she had required continuous bronchodilator treatment and intermittent courses of corticosteroids. She was a tense and determined girl who always delayed seeking advice until her asthma attacks were very severe. She often refused corticosteroids due to the side effects of facial puffiness and weight gain.

In January 1977 after an upper respiratory tract infection she was admitted in severe acute asthma requiring intensive treatment (bronchodilators, steroids, antibiotics, oxygen, and physiotherapy). As a result of this admission and a past history of nocturnal and brittle asthma (Turner-Warwick, 1977) she was considered to be an at risk patient and therefore was followed closely as an outpatient with the aid of continuous, twice daily, peak expiratory flow rate (PEFR) recordings. Examples of her recordings (fig 1) show the wide diurnal variation in PEFR associated with sputum production and relative stabilisation with appropriate corticosteroids.

In July 1977 she was readmitted, unconscious in respiratory failure after a sudden, severe asthma attack. She responded promptly to immediate resuscitation measures (oxygen and intravenous aminophylline) and made a rapid recovery with intensive treatment. She did not require artificial ventilation.

After this admission, in an attempt to prevent her arriving in casualty moribund, she was instructed in the self-administration of subcutaneous injections of terbutaline for use during sudden severe asthma attacks that did not respond to pressurised aerosol bronchodilators.

In October 1977 she was admitted to a nearby

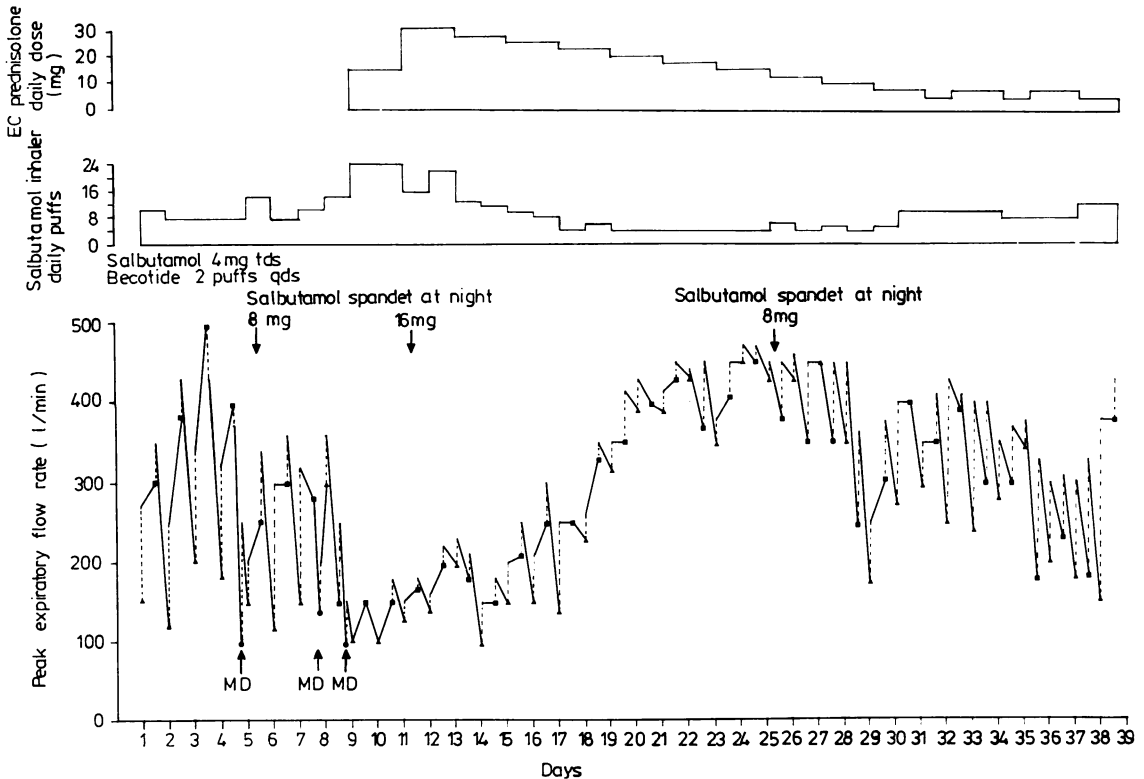


Fig 1 Patient 1. Examples of morning (▲) and evening (■) PEFR recordings. Reversibility of airways obstruction with an aerosol bronchodilator denoted (---). Nocturnal attacks (morning dip: MD) denoted (●).

hospital with cardiorespiratory arrest after a final sudden severe asthma attack. Regrettably her terbutaline injection had been left in another handbag at home and consequently was never used. Initial resuscitation was successful, although she remained in coma and required artificial ventilation. Some 18 hours later she died after a second cardiac arrest. At necropsy there was extensive mucus plugging and muscular hypertrophy of the smaller airways, the surfaces of which were denuded of epithelium and their basement membrane thickened.

CASE 2

A 25-year-old female hairdresser had a three year history of asthma. Her first attack was precipitated by an upper respiratory tract infection and necessitated hospital admission. After this admission her asthma remained relatively mild, requiring only intermittent use of a salbutamol inhaler. She did, however, have a history of nocturnal wheezing. After an upper respiratory tract infection in October 1976 she was admitted under

our care for the first time in severe acute asthma. With intensive therapy (bronchodilators, corticosteroids, antibiotics, oxygen, and physiotherapy) she made a rapid and complete recovery. On discharge her progress was closely followed with the aid of continuous PEFR recordings (fig 2). For the first two months after discharge her asthma was in remission requiring no treatment. From then on, however, she exhibited a moderately wide diurnal variation in PEFR associated with sputum production. Her early morning wheezing improved after the administration of a slow release bronchodilator (salbutamol spandet). Appropriate oral corticosteroid therapy, however, completely stabilised her asthma and sputum production ceased.

Again, this young woman had a tense and determined personality and was most unwilling to take corticosteroids, for similar reasons as the first patient. She was not, however, considered to be at risk from sudden severe asthma attacks.

In October 1977, after nine months' follow-up, she suffered a sudden severe asthma attack while at work (about seven miles from our hospital),

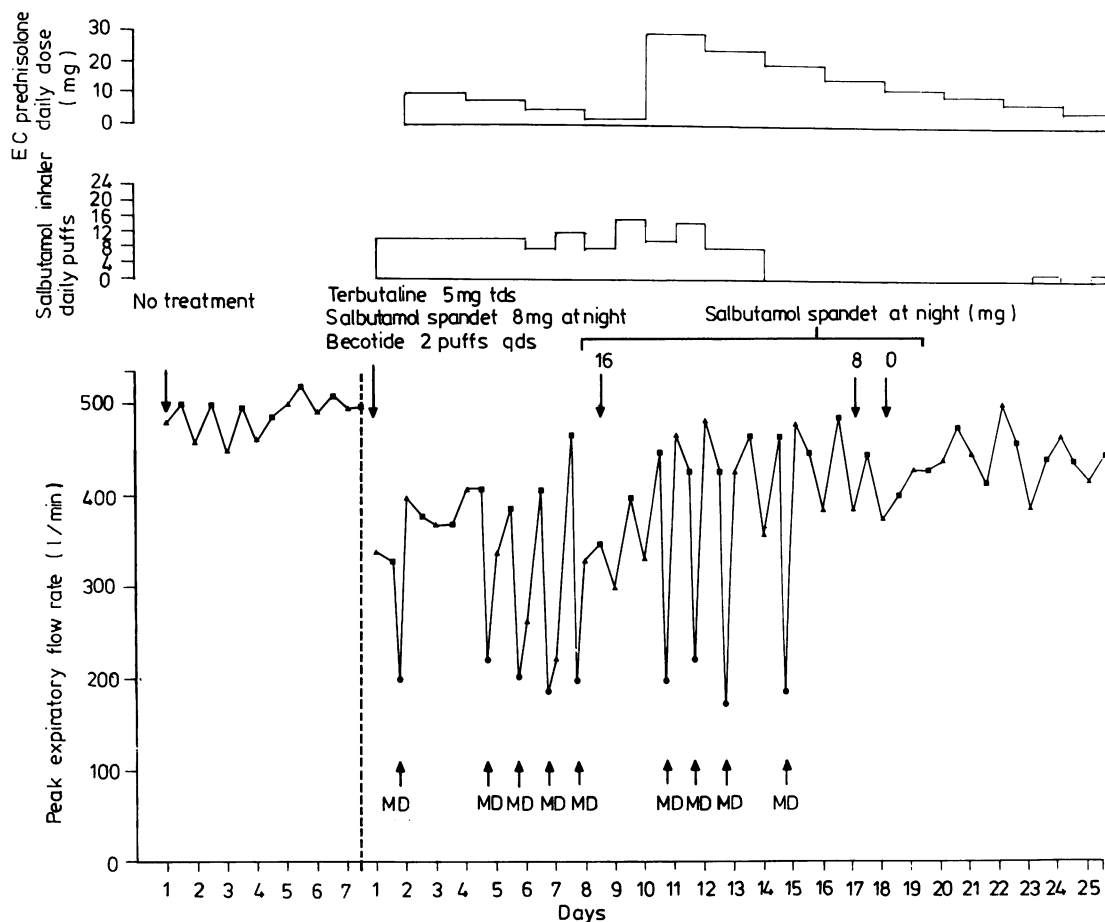


Fig 2 Patient 2. Examples of morning (\blacktriangle) and evening (\blacksquare) PEFR recordings. Nocturnal attacks (morning dip: MD) denoted (\bullet). First seven days are taken from a period of remission.

which did not respond to her salbutamol inhaler. She was rushed here by taxi, but on arrival was in cardiorespiratory arrest. The most striking event during resuscitation was the almost immediate response to parenteral adrenaline, with respect to the relief of airways obstruction and the restoration of effective cardiovascular function. Despite this rapid response she remained comatose and finally died one week later. Serial EEG recordings throughout this period were compatible with brain death. Again the classic pathological features of asthma were present in the lungs at necropsy.

Discussion

Similar features are present in both cases. Firstly, continuous recording showed a wide diurnal vari-

ation in PEFR associated with nocturnal wheezing and sputum production, which resolved with appropriate corticosteroids. Neither patient was taking corticosteroids at the time of the abrupt fatal attack. Secondly, parenteral, but not aerosol, administration of bronchodilators rapidly partially relieved the airways obstruction during the acute episode. Finally, the distances travelled and time taken to seek hospital treatment were such that both patients arrived with cardiorespiratory arrest.

A long history of asthma (Macdonald *et al*, 1976a), under-use of corticosteroids (Cochrane and Clark, 1975), and previous admissions with severe acute attacks (Macdonald *et al*, 1976b) are factors associated with death from asthma. Recently it has been shown that a wide diurnal variation in PEFR during the early days of recovery from

severe acute asthma was associated with an increased risk of sudden severe recurrence (Hetzel *et al*, 1977). Our patients showed this pattern well before the fatal attack, indicating airway instability and the need for more intensive therapy. In our experience the charting of twice daily PEFR on a diary card at home and subsequent modification of therapy has been a useful "bio-feedback" procedure in managing asthmatic patients. In this manner the individual's asthma pattern may be easily determined and possible at risk patients identified. Patients showing a wide diurnal variation in PEFR are probably at risk from abrupt severe asthma attacks both during recovery from an acute episode and, as in these two cases, well before the acute episode. A wide diurnal variation in PEFR may well be a better guide to the recognition of the susceptible patient than the factors previously mentioned.

Airway obstruction in asthma comprises smooth muscle contraction, mucosal oedema, and mucus plugging (Dunnill, 1971). The cases described suggest that mucosal oedema and mucus plugging were the key underlying disturbances leading to death and that acute smooth muscle contraction was the final event. Modern treatment of acute asthma centres on the reversal of smooth muscle contraction and often little attention is given to the underlying mechanism and treatment of mucosal oedema and mucus plugging. Corticosteroids reduce both mucosal oedema and lung secretions, and although their use in asthma has increased recent reports indicate that they are still under-prescribed (Cochrane and Clarke, 1975; Macdonald *et al*, 1976a; *British Medical Journal*, 1978; Seaton, 1978). Regrettably both patients in this report frequently refused to take steroids for cosmetic reasons.

Immediate and appropriate treatment is essential to survival during a sudden severe asthma episode. At the time of the final admission both patients immediately responded to parenteral adrenaline. Adrenaline is now little used in treating acute asthma (*British Medical Journal*, 1978), although it remains an effective first-line drug for the hospital management of the young acute asthmatic (Harris, 1975; Hutchinson, 1975). There is no convincing evidence in published reports that the administration of adrenaline is contra-indicated in young otherwise healthy people. Subcutaneous injections of terbutaline have been available since 1971 and of salbutamol since 1975. The effectiveness of these more selective beta-agonists has not been compared with that of adrenaline, and experience in their use is limited. Patients may easily be instructed in the self-

administration of subcutaneous injections and in this situation such a procedure may be life saving.

A self-admission service to a specialised asthma unit is effective in preventing fatal asthma (Crompton and Grant, 1975). Such a service has been in operation at our hospital for the past 18 months. The distances travelled and time taken by the two patients to reach hospital resulted in delay in their receiving appropriate emergency treatment. Their deaths might have been avoided if they had sought advice at the nearest casualty department. In future at risk patients should perhaps be issued with a medical bracelet and identity card displaying basic medical details and instructed to travel to the nearest casualty department in the event of an emergency rather than to try to reach their usual hospital.

About 1500 patients die from asthma each year in the UK (*British Medical Journal*, 1978). Many are young and in most death could have been prevented. We believe that prevention is possible, provided that, firstly, the at risk patient may be identified; secondly, outpatient treatment in such patients is adjusted with the aid of continuous PEFR recording, and thirdly, appropriate treatment is rapidly administered during an acute severe episode, initially by the patients themselves.

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