CLINICAL NOTE

SUDDEN DEATH IN ASTHMATICS NOT IN SPASM

W. K. AITCHISON, M.B., B.S., D.Obst.R.C.O.G., and N. CROSS, M.B., Ch.B.
Gateshead-on-Tyne

IN THE COURSE OF TWELVE MONTHS, in a small industrial practice, four asthmatic patients, two male, two female, ages ranging from 14–45 years, died suddenly and unexpectedly from a cause or causes other than status asthmaticus. Two died in hospital and two at home. All four had been under close constant supervision, and had been treated in an orthodox manner. There are marked similarities in their histories, and the mode of death as far as can be ascertained was identical as will be seen in the following:

Case 1. Female, aged 44 at death, duration of asthma 24 years with no record of any precipitating factors. The disease presumably ran a benign course for 19 years—there are no detailed records.

Chest x-rays were normal repeatedly. Treatment consisted of routine antispasmodics with antibiotics for chest infection shown by purulent sputum. Steroids were introduced four years before death, and used intermittently in short courses. Following psychiatric assessment, tranquillizers and antidepressives were used for a few months, but with no detectable improvement. These were stopped two months before death.

Attacks gradually increased in frequency, severity and duration. One month prior to death she was admitted to hospital in status asthmaticus. An IVP, performed following detection of a concurrent urinary infection, was normal. In the evening before death she was given IV aminophylline at surgery, and started a course of broad spectrum antibiotics. She was able to return home alone and that night slept well. The following morning she awoke feeling well, but suddenly became distressed and died almost instantaneously without bronchospasm.

Family history. One spinster sister and two bachelor brothers living together with mother. One married sister lived away from home. Patient appeared to be the family favourite, but the domineering mother refused her permission to marry at age 43 in spite of the readiness of a home for her. One brother was a high grade mental defective who drank heavily, and the other had a low IQ. The family lived in a small dingy upstairs flat in slum property.

Case 2. Male, aged 14 at death. Duration of asthma, six years. Possible associated or precipitating factors were whooping cough, asian flu, alopecia areata. Sensitivity tests for animal and house dusts and pollens were positive but he was not desensitized.

Prior to death he was in hospital on a long-term basis, and then sent to a special residential school. Return home invariably precipitated attacks. While in hospital steroids were introduced and gradually withdrawn. They were similarly used at school on a few occasions.

Before his last admission, although not officially on steroids, he had apparently

taken an occasional tablet himself. On the day of death he had slight intermittent wheezing only, but in the presence of nurses he suddenly developed acute cardiac arrest, and attempts to revive him with artificial respiration and IV cardophylline, coramine and (codelsol) were unavailing.

Family history. He was the third of five children, one of whom had a flail hip. He was always regarded as ' soft ', was nervous and easily upset. At age three he had an eye injury partially dislocating the lens causing amblyopia, but this did not displace his crippled brother from his privileged position in the family. Living conditions in a small council house were poor and overcrowded.

Case 3. Male aged 45 at death. Duration of asthma 14 years preceded by pneumonia. Progress of respiratory disease was initially slow, but accelerated following separation and divorce from his first wife three years before death.

X-rays until this time were normal. Treatment had consisted of antispasmodics and concurrent antibiotics for infection, but steroids were added in hospital and continued at a low maintenance dose with one short break. Severe spasm was treated by full dose of steroids in hospital. Bronchoscopy and bronchography failed to demonstrate a neoplasm suspected because of repeated reports of atypical cells in the sputum. During one severe attack in hospital his PCO_2 was 55.4 mm. Hg. but fell to 32.9 mm. Hg. after two days. Expirograms were very flat. Electrolytes were always normal.

Hospital admissions became more frequent, and after the last one he developed an injection abscess which a few weeks later required surgical incision under general anaesthetic. He was seen at home in the morning and was free from spasm but still taking a broad spectrum penicillin, antispasmodics and a low dose of prednisone. In the afternoon he became distressed and suddenly collapsed and died.

Past history. In early adult life he had recurrent bronchitis and tonsillitis. He also had a 17-year history of recurrent eczema of both hands.

Family history. Three siblings. He was separated from his wife for two years before divorce, with no family. Later he married a timid woman but continued living with his mother who had a strong personality. Living conditions were satisfactory in a private semi-detached house.

Case 4. Female, aged 38 at death. Duration of asthma 14 years preceded by pneumonia. Initially attacks of wheezy bronchitis occurred annually, but the frequency increased until freedom from bronchospasm could be measured in days. During the last year of life she developed rheumatoid arthritis and the diagnosis was confirmed by a raised ESR and positive Rose Waaler test.

Other investigations including chest x-ray, blood chemistry and ECG's were repeatedly normal. Treatment consisted of antispasmodics and antihistaminics although no allergic factor was ever demonstrated. Almost every known antispasmodic, both oral and parenteral, was eventually tried prior to the introduction of steroids in hospital. Any chest infection was treated with broad spectrum antibiotics, although sputum cultures revealed on a number of occasions only *Streptococcus viridans.*

On her last admission to hospital severe recurrent spasm gradually subsided with full dosage of steroids and antispasmodics. Gradual withdrawal of prednisone was started, but during the night she complained of severe pleural pain for which she was given pethidine and crystamycin. The following morning she was pyrexial, became suddenly distressed and died. Post mortem revealed an early right lower lobe pneumonia; the adrenals appeared normal.

Family history. She lived in a pleasant semi-detached council house with husband and father who also suffered from wheezy bronchitis. His attacks

have been noticeable by their absence since his daughter's death. Psychiatric assessment failed to reveal any emotional factor in causing her asthma, although both her father and husband were not very sympathetic. She had no family.

Discussion

We have presented four cases of sudden death—all of whom had a terminal infection. None of these patients died in status asthmaticus. Walton *et al.*(1951) reporting on 13 cases of sudden death in asthmaticus (not on steroids) stressed: (1) widespread bronchial obstruction due to mucus plugging, (2) the danger of certain sedatives. Autopsy carried out in one case did not show mucus plugging. In none of the cases was sedatives given prior to death. Jarvinen (1952) stresses the exercise of caution when giving cortisone to an asthmatic patient with pulmonary emphysema/cor pulmonale.

In only one of the presented cases was emphysema/cor pulmonale demonstrable clinically or radiologically. None of the patients had at any time shown signs of cortisone overdose, or alteration in blood pressure.

These cases stress the need for caution in commencing steroid therapy in asthmatics irrespective of the presence of emphysema or cor pulmonalle and the need to increase the dosage of steroids above maintenance leve, in the presence of even minor infection.

Summary

This paper presents the case histories of four asthmatics who had been treated similarly with antispasmodics, antibiotics and steroids, and who had in common an emotionally unstable home life, a terminal infection and sudden death not due to status asthmaticus.

REFERENCES

Walton, H. A., Penner, D. W., and Wilt, J. C. (1951). *Canad. M. A. J.*,**64**, 95.
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