Acute respiratory arrest in status asthmaticus

A report of 2 cases

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Summary

Respiratory arrest is a rare but serious complication of status asthmaticus. Two such cases, which were closely associated with the use of intravenous steroids, are reported. Possible causes for the sudden deterioration of patients in status asthmaticus are discussed.

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There are a number of reasons why the condition of an asthma patient may deteriorate to the state where ventilation is required. The patient may have coexisting disease such as left ventricular failure or chronic obstructive airways disease or be acidotic, for a variety of reasons. Respiratory infection and non-compliance are relatively common precipitating events.[1,2] A wide variety of drugs interfere with effective therapy; ß-blockers, sedatives, morphine, tricyclic antidepressants and carbonic anhydrase inhibitors as well as prostaglandin synthetase inhibitors have all been associated with a poor response to bronchodilator therapy.[1,3,4] More recently an anaphylaxis-like reaction to steroids in asthmatics has been described.[5-7] Patients experience severe bronchospasm and urticaria. This has been demonstrated to be a dose-related phenomenon;[6] skin tests showed that the steroid and not the diluent was responsible. The rarity of this response may be judged from the fact that only 1 report[7] is listed in a recent publication on adverse drug reactions.[8]

This is a report on 2 patients who developed severe bronchospasm resulting in respiratory arrest immediately after receiving intravenous hydrocortisone.

Case reports

Case 1

A 48-year-old Coloured man had suffered from asthma all his life but had never been hospitalized or received long-term steroid therapy. On this occasion bronchospasm had been precipitated by an upper respiratory tract infection, and he presented with a 2-week history of low-grade bronchospasm which had not responded to treatment (which had included prednisone) by his general practitioner. On examination he was mildly distressed with a pulse rate of 108/min and pulsus paradoxous of 20 mmHg. He was tachypnoeic but not cyanosed and had moderately good air entry with a diffuse wheeze.

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He received nebulized hexoprenaline, followed by intravenous theophylline and later a bolus of hydrocortisone 400 mg. Immediately he received the steroid he became intensely distressed and cyanosed and experienced a respiratory arrest. The pulse rate dropped to 48/min, although the blood pressure remained normal. The patient was intubated and ventilated via an Ambu-bag and later by intermittent positive-pressure ventilation. In addition an infusion of salbutamol 10 mg/l was commenced, while the theophylline was continued. Spontaneous respiratory effort was noted within 10 minutes but ventilation was continued electively for a further 18 hours before extubating the patient.

His course was complicated by conjunctival haemorrhages and surgical emphysema for which bilateral chest drains were inserted. However, a chest radiograph taken shortly after the incident failed to reveal a pneumothorax.

Case 2

A 57-year-old Coloured man had been an asthmatic all his life. His asthma was normally well controlled on oral theophylline and a salbutamol inhaler. He presented with a 3-day history of low-grade bronchospasm which had not responded to medication, and despite the relatively mild nature of the bronchospasm it was decided to commence steroid treatment *ab initio*. The treatment and subsequent course of events were almost identical to those in case 1 except that on this occasion the patient received 0,5 ml 1: 1 000 adrenaline subcutaneously immediately the symptoms of bronchospasm occurred. In this case neither surgical emphysema nor conjunctival haemorrhage complicated the course. The patient showed signs of spontaneous respiration and was extubated at 4 hours. A chest radiograph failed to reveal unexpected lesions.

Discussion

These cases are not intended as examples of an anaphylactoid reaction to steroids, for there is nothing to support this except the chronological sequence of events. The object is rather to draw attention to this rare event and the importance of recognizing that an asthmatic whose condition does deteriorate rapidly while on steroids may in fact be adversely affected by the preparation. It is possible for patients to be exposed repeatedly to the hazards of intubation and ventilation because of this aberrant response.[6,7] Furthermore, it is not a typical anaphylactic reaction but a dose-related phenomenon, and it is possible for these patients to receive low-dose steroids or infusions without ill-effects.[6]

Although skin tests have been used in such cases, they are not an accurate index of bronchial sensitivity.[9] It is important therefore to challenge patients who have suffered an arrest with a bolus of steroid while intubated to document their response. Despite the rarity of the reaction, the large number of asthmatics makes it likely that the actual number of individuals sensitive to steroids may well be significant; they should be identified to prevent potentially severe complications.

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Progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome)

A case report

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Summary

A 60-year-old Black man with advanced clinical signs of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome) is described. He was initially thought to suffer from Parkinson's disease, but as the disease progressed supranuclear palsy was suspected. The clinical picture and pathological changes in this rare syndrome are discussed.

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Progressive supranuclear palsy is a progressive condition of unknown aetiology characterized clinically by supranuclear ophthalmoplegia of vertical gaze, extrapyramidal syndromes of parkinsonian type and axial dystonia, pseudobulbar palsy, dementia, ataxia and occasional pyramidal signs. The syndrome was established in 1964 by Steele as a separate nosological entity, although it was probably first described by Posey at the beginning of this century.[1]

Diagnosis is primarily clinical and pathological, although characteristic pneumencephalographic and computed tomographic findings have been described.[2] Characteristic autopsy findings include neurofibrillary tangles and gliosis associated with extensive loss of nuclei in the brainstem, diencephalon and cerebellum. There is no specific treatment, although L-dopa has been tried.[3]

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Case report

A 60-year-old Black man presented with a 3-year history of progressive weakness of horizontal and vertical gaze, associated with gait, speech and swallowing difficulties. He had had two previous admissions to hospital within a year with the same complaints and was diagnosed as having possible parkinsonism. His past history was non-contributory and his family history negative for parkinsonism and related neurological diseases. General examination revealed marked generalized muscle wasting. His blood pressure was 130/80 mmHg and his pulse rate 88/min; there was no postural hypotension. His skin colour was normal and there was no lymphadenopathy. The rest of the general examination was negative. He was fully orientated as to time, place and person. His use of language was normal. His short-term memory was poor, but his long-term memory was intact. His general knowledge was good and his affect normal and appropriate, but he had some difficulties in simple calculation. The first cranial nerves were intact. He had bilateral miotic pupils, and vertical gaze on voluntary effort and optokinetic or caloric induced nystagmus were absent, but doll's eye movement was intact both laterally and vertically (Fig. 1). Hearing was intact and the gag reflex was present. His speech was slow and mildly dysarthric and his tongue was spastic. There was no emotional lability. His head was held rigidly in an extended position and his facial expression was mask-like. Examination of the motor system revealed generalized wasting, and muscle tone was increased in all limbs. He had marked cogwheeling and bilateral mild resting tremor of both upper limbs. Power was normal and his knee reflexes brisk on both sides with negative Babinski responses. The sensory system was normal to all modalities. His gait was broad-based and slightly ataxic.

In summary, the patient presented with the following symptoms: supranuclear gaze palsy, symptoms resembling those in parkinsonism, abnormal head posture, pseudobulbar palsy, ataxia, pyramidal signs, and mild dementia which had developed insidiously over a period of 3 years. Laboratory tests, including analysis of the CSF, were negative. Computed tomography (CT) revealed only mild generalized cortical atrophy. The electroencephalogram was normal.