Leading Article

Managing asthma in hospital: cause for concern

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Asthma is now the commonest chronic disease in this country, but remains underdiagnosed and undertreated in the community.1,2 The mortality for asthma is not being reduced and may even be increasing,3 in spite of greater understanding and increasing prescribed therapy. Despite the fact that asthma is a treatable condition, deaths certified as due to asthma continue to occur in hospital. Estimates of the proportion of asthma deaths occurring in hospital vary from 14% to as high as 37%.4,5 Recent surveys have highlighted the poor standards of care of asthma in hospitals, with inadequate monitoring and treatment of acute attacks and inadequate follow-up.6-8 A review of 35 deaths due to asthma in hospitals in the North-East Thames region revealed that management could be considered defective compared to matched control admissions.7 Assessors considered that there were important defects in management in 29/35 fatal cases and 14/35 controls, which included inadequate monitoring of blood gases or lung function, inadequate treatment with steroids or beta-agonists, inappropriate use of aminophylline, under-assessment of severity, and failure to institute artificial ventilation. It is particularly disturbing to see that these defects in management were also seen in 40% of the matched controls. A retrospective study from Birmingham and Manchester demonstrated the relative deficiencies in care of patients admitted under general physicians compared to respiratory physicians.8 Two prospective studies from Glasgow have further highlighted the discrepancies in the care of patients admitted with acute asthma, depending upon whether they were admitted under a respiratory specialist or under another physician.9,10 Although patients in the two groups were clinically comparable, in the non-specialist managed group fewer patients were treated with steroids, fewer had regular peak flow recordings made and fewer were given follow-up appointments. After discharge from hospital, more patients from the non-specialist group reported asthma symptoms. But perhaps the most striking difference was the finding that 20% of first admissions in the non-specialist group were re-admitted within the year compared to 2% of the group treated by respiratory specialists.

We are now in the age of audit and such discrepancies in care must be questioned. Management of acute asthma is ideally suited to audit, since the clinical condition is relatively well defined, the best way of treating acute asthma is generally agreed, and the outcome in terms of recovery and re-admission can be determined. In a recent informal survey of acute severe asthma admissions to Brompton Hospital we found a defect in management (inadequate assessment and inappropriate treatment) in many of the patients. We have acted on this deficiency by devising guidelines for management of acute severe asthma, which we hope will improve the standard of care in the future.

The management of most cases of acute severe asthma in hospital is relatively straightforward. On admission, the patient should be rapidly assessed and the heart-rate and degree of respiratory distress and the presence of cyanosis should be recorded. Pulsus paradoxus is present in some patients with severe asthma, but it does not correlate well with severity,10 and it is probably not worth measuring since this wastes time. Curiously, we found it to be the single measurement recorded in every patient in the Brompton Hospital audit. It is important to measure airflow obstruction objectively and peak respiratory flow (PEF) is more convenient and quicker than forced expiratory volume in one second (FEV1), and more suited to repeated measurements during the course of treatment. Arterial blood gases (radial artery puncture) should also be measured in all patients with a severe attack. A low PaO2 is expected but Paco2 should be low; a normal, rising or high Paco2 may indicate a potentially dangerous situation and impending respiratory failure.11 A chest X-ray is important in identifying complications such as pneumothorax, pneumonia or collapse, but should be deferred until after initial assessment and treatment. Blood tests and electrocardiography are not helpful, although plasma potassium should be checked at a later stage since hypokalaemia may develop from beta-agonist, theophylline or steroid therapy.

Management of acute asthma is relatively straightforward, yet undertreatment is a common
feature of all the hospital surveys. Hypoxaemia is present in all cases and is related to the severity of the acute attack. It should be treated immediately with high concentration oxygen (35% via a Ventimask or 40% via a Hudson mask); 24% and 28% masks are inappropriate as chronic hypcapnia is not a problem. A nebulized beta-2 agonist should be given immediately in an adequate dose, such as salbutamol 2.5–5 mg, or terbutaline 5–10 mg, and, because of a risk of increasing hypoxaemia, due to pulmonary vasodilatation and increased shunting, the nebulizer should be driven by oxygen. There is convincing evidence that nebulized beta-agonists are just as effective as intravenous beta-agonists or aminophylline, but have a lower risk of side effects. It is curious that this should be so, but presumably drugs absorbed from the upper respiratory tract are distributed to lower airways in the bronchial circulation. There is some evidence that the equivalent dose of beta-agonist, given by a metered dose inhaler such as 25–50 puffs terbutaline, into a large volume spacer device, produces similar bronchodilatation to nebulization, but patients may not find it as easy to use when acutely distressed and hyperventilating. Nebulized ipratropium bromide may give some additional bronchodilator effect when combined with beta-agonists, but should never precede beta-agonists because of the dangers of bronchoconstriction.

Intravenous aminophylline should only be used in the rare patients who do not respond to adequate doses of nebulized beta-agonists. There is unlikely to be any advantage of adding theophylline to beta-agonists, but there is the disadvantage of side effects. Yet intravenous aminophylline continues to be used in the management of asthma in casualty and often at potentially toxic doses, and is particularly dangerous in patients already receiving an oral theophylline preparation. If aminophylline is to be used it is better to introduce a steady infusion and to omit the loading dose. The infusion rate can then be monitored in relation to the plasma theophylline concentration. Every survey of asthma deaths has highlighted the under-use of systemic steroids. Corticosteroids are important in speeding the resolution of the acute exacerbation and should be given to every patient in a high dose. Oral prednisolone is rapidly and well absorbed and should be given in a large dose (40 mg orally) as soon as possible. There is no advantage to using intravenous hydrocortisone or methylprednisolone, which should be given only when patients have impaired consciousness, are vomiting or are unable to swallow. Sedatives are absolutely contraindicated and antibiotics are indicated only by evidence of bacterial infection (which is rare in acute asthma). In a very severe attack a patient may be unconscious or confused. Other patients may be exhausted, with deterioration, despite appropriate emergency treatment. Other patients may have a high or rising PaCO₂, indicating ventilatory failure. These patients will require intermittent positive pressure ventilation and must be admitted to the intensive care unit. Surveys of asthma death in hospital have indicated that delay in instituting ventilation is common.

This regime of treatment is easy to follow and simple guidelines to treatment should be available in every casualty department. Although the immediate management of acute attack in hospital is poor, the subsequent management in hospital is even worse. Nebulization of beta-agonist should be repeated after 30 minutes if no improvement is seen after the first nebulization and, if improving, should be repeated 4 hourly. Peak flow should be measured after 1 hour then 4 hourly before and after each nebulization. Patients can usually be discharged from hospital within a week when lung function has returned to the best values recorded when well. Patients should not be discharged home until large diurnal swings of PEF have diminished as there is a high risk of sudden death during this period of instability. Nebulized bronchodilators should be changed to standard inhalers 48 hours before discharge and inhaler technique should be carefully checked and, if necessary, improved. All patients should be discharged on oral steroids (prednisolone 20–40 mg daily) for 1–3 weeks in a tapering dose and all patients should be given inhaled steroids (200–1000 μg beclomethasone or budesonide twice daily depending on the severity of the asthma prior to the attack). It is likely that every patient who is admitted with an acute exacerbation of asthma will require the long-term use of inhaled steroids. Patients should also take inhaled beta-agonists as required. A few patients may need slow-release theophylline as an additional bronchodilator; plasma concentrations should be checked before discharge.

It is important that arrangements are made for follow-up assessment. The patient should always see their general practitioner within one week of discharge and should carry a note of all medications. All patients should also be seen by a respiratory specialist within a month, even when admitted under a general physician. Admission to hospital with acute asthma can be regarded as a treatment failure and a change in chronic therapy may be needed to reduce the risk of further asthma exacerbations.

The value of self monitoring of peak flow is now being recognized, particularly in patients with unstable and unpredictable asthma. All patients admitted to hospital with an acute attack should be given a peak flow gauge (which are now prescribable) to record PEF at least twice daily until seen in outpatients. PEF recordings are particularly
valuable in patients with unstable or brittle asthma, but are also useful in other patients as a guide to preventive therapy. A written 'action plan' should be drawn up for patients admitted with acute attacks indicating what steps should be taken when asthma starts to deteriorate based on measurements of PEF. Thus, if PEF is less than 75% best value, patients should double the dose of inhaled steroids, if less than 50% best they should start a short course of oral steroids, if less than 25% best they should call a doctor or arrange readmission to hospital directly (using an agreed self-admission procedure). The introduction of such a plan has been found to significantly improve symptom control and improve lung function.

All the evidence suggests that asthma is poorly treated in hospital, particularly under the care of non-respiratory specialists. The introduction of audit procedures should highlight these deficiencies in each hospital and agreed guidelines have now been introduced. The treatment of acute severe asthma is not a controversial issue and most specialists will be in agreement with the treatment plan outlined above. Adoption of such an approach is likely to markedly reduce the morbidity of asthma, and hopefully also prevent deaths. This procedure will also save money in that readmissions to hospital are likely to be substantially reduced and less time will be spent off work. Ideally every patient who attends hospital with an asthma attack should be seen by a respiratory specialist. This is likely to place increased demands on over-burdened respiratory services, in view of the large number of patients involved, and argues very strongly for an increase in respiratory specialists in the future.

References