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SMJ

Editorial

Acute Life-Threatening Asthma: Not a Time to Procrastinate!

K N Sin Fai Lam

In the words of Dr Thomas Petty[1], "the best treatment of status asthmaticus is to treat it 3 days before it occurs". The implication is that in the majority of cases, there is a progressive worsening of symptoms leading to the life-threatening attack, and pre-emptive measures taken early are able to abort or reduce the severity of the attack. On the other hand, other patients may have impaired perception of dyspnoea, unaware of worsening airflow obstruction and a minority may have the truly catastrophic type of asthma with sudden and unexpected bronchospasm. Whatever the tempo, acute life-threatening asthma can lead to ventilatory failure and death.

In this issue of the SMJ, Khadadah et al[2] describe their experience in the management of severe acute asthma in the intensive care unit of a tertiary medical centre. Over a 2 year period, they had experience of treating 30 patients in the ICU. The patients were critically ill, 11 of whom were in a coma and 26 had hypercapnia at presentation. The mortality of the 21 subjects who required mechanical ventilation was 14%. This figure was compared with those of 4 other studies in which the mortality of ventilated patients ranged from 0 to 21%. The authors found no complication due to barotrauma as a result of mechanical ventilation and the patients who died did so because they were already moribund at presentation.

The most significant aspect of risk stratification as to the likelihood of near-fatal asthma is the prior clinical course. Hence the patient is considered high-risk if there is a history of several hospitalisations over the past year, deterioration while receiving oral steroids, development of hypercapnia during an attack, marked diurnal variation in PEFR, a previous episode of near fatal asthma or a history of intubation and ventilation. Findings such as disturbance of consciousness, inability to speak, absent breath sounds and central cyanosis clearly indicate that the patient is in impending or frank respiratory failure. In other cases, indicators of severity include the upright position, marked sternocleidomastoid retraction, tachycardia exceeding 120/min, respiratory rate over 30/min, pulsus paradoxus and diaphoresis. Although objective measurement of the PEFR is a useful guide, if all the other signs clearly indicate life-threatening asthma, such a measurement

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should be deferred until after the clinical status has improved; the PEFR manoeuvre may lead to a dangerous reduction in airflow and even precipitate respiratory arrest. Although a normal or elevated $PaCO_2$ are signs of severe asthma, the presence of hypocapnia does not rule it out. Metabolic acidosis may even be more significant indicating very severe airflow obstruction.

Most patients presenting with an attack of asthma respond to treatment with oxygen, beta-agonist nebulisation, corticosteroids, supplemented if necessary with anticholinergics, or aminophylline. Other patients do not respond so well and it is imperative that continuous observation of the patient is maintained as long as definite signs of improvement are not apparent. Patients who do not improve after initial maximum treatment or who deteriorate should be transferred to the ICU electively for closer observation and preparation for elective intubation. A more liberal use of the ICU for patients with severe asthma has previously been advocated[3,4]. Procrastination may result in a respiratory arrest. The outcome is always worse after a respiratory arrest; Lee KH et al[5] reported only 50% survival from hypoxic brain damage after a respiratory arrest in spite of good intensive care treatment. It is not optimal to wait until the patient is nearly moribund from CO_2 narcosis to take control of the airway. If active airway intervention is inevitable, it is preferable to make this decision early rather than late. It is better to be pro-active rather than to procrastinate. Early endotracheal intubation and mechanical ventilation in the patient with life-threatening asthma may be prudent measures which save life and result in good clinical outcome. Absolute indications for intubation are cardiac and respiratory arrest or significant alteration in mental state. At other times, the decision is taken in the face of progressive deterioration and increasing exhaustion. We would like to see more patients who require ventilation be intubated electively rather than as a result of a cardiorespiratory arrest. Blood gas abnormalities per se are not an indication to intubate the patient. Many with hypercapnia and respiratory acidosis will respond to treatment with bronchodilators and do not require mechanical ventilation[5,6,8]. More important is the trend in clinical findings and in the arterial blood gases.

Procrastination in the initiation of mechanical ventilation when it is undoubtedly required is related to the fear of complications. Admittedly, consideration must always be given to the potential risks. Intubation in a dyspnoeic, anxious and restless patient should be done by the most experienced clinician available. A large endotracheal tube is selected as it helps suction and reduces airways resistance. There are advantages of both nasotracheal and oral intubation, and sedative choices in the preparation of the patient vary between individuals. Once endotracheal intubation has been carried out, the physician's priority is to take control with positive pressure ventilation. This will require the use of adequate sedation and usually paralysis with a muscle relaxant. Paralysis may often only be required in the initial stages of ventilation, and should be weaned off as soon as possible to minimise the risk of acute myopathy. Ventilation should initially be in the CMV mode. Ventilatory settings are chosen to avoid excessive lung inflation. Such a strategy decreases the risk of pneumothorax or systemic hypotension. Lung inflation is minimised by allowing an adequate time for exhalation (T_E). Expiratory time may be prolonged by decreasing minute ventilation (V_E) by either lowering respiratory rate RR or tidal volume (V_T) or minimising inspiratory time (T_I). Inspiratory time is reduced by increasing inspiratory flow rate and by using a square flow wave form. Tidal volumes of 8-10 mls/kg with a

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respiratory rate of 10-14 and inspiratory flow rates of 60 L/min or higher are often suitable. An acceptable peak airway pressure of < 50 mm H_20 is aimed for. *Controlled Hypoventilation* is a technique used to reduce the risk of barotrauma and hypotension. The aim is to ensure adequate oxygenation, avoid excessive dynamic hyperinflation (DHI) and allow a degree of hypoventilation. It is perfectly acceptable to allow the PCO_2 to rise in order to avoid DHI. One should not attempt to normalise the PCO_2 at the expense of DHI. Hypotension may be attributed to excessive DHI. A brief trial of apnoea (30-45 seconds) is diagnostic, as venous return increases and blood pressure rises during the period of apnoea. If this does not happen, other causes of hypotension such as tension pneumothorax, fluid depletion, excessive sedation or myocardial depression must be considered.

Several studies have clearly indicated that mechanical ventilation saves lives in life-threatening asthma[2,3,5-8]. With good ICU management, complications can be kept to a minimum, the period of required ventilation is short and there is usually no problem in weaning the patient off the ventilator. Let us remember, however, that at the end of the day, the treatment of acute life-threatening asthma does not just start 3 days before the attack, but much earlier, as most acute attacks are preventable with the proper use of regular prophylactic medication, the avoidance of trigger factors, the proper education of patients, the objective measurement of the PEFR, the appropriate implementation of a co-management plan between the patient and his doctor, and the accessibility to medical care in times of crisis. SMJ

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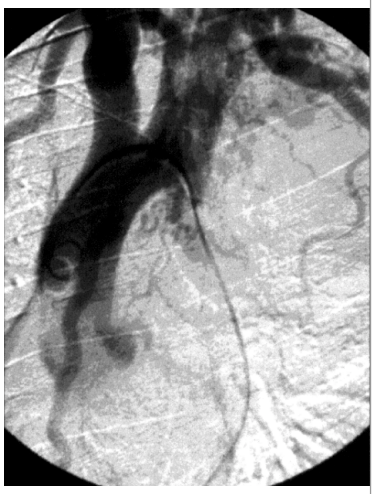

Cover Picture: An aortogram showing a tight stenosis of the thoracic aorta just distal to the left subclavian artery with very large collateral vessels.
(Refer to pages 235-238)