Respiratory Medicine (1994) **88,** 387–389

Case Reports

The therapeutic use of ultrasonic nebulizers in acute asthma

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Introduction

Reversible airway obstruction is a characteristic feature of asthma (1). On a pathophysiological basis this is produced by bronchoconstriction, mucosal oedema and inspissated secretions (2). Modern drug therapy for asthma is directed towards controlling mucosal inflammation and hence reversing bronchoconstriction and mucosal oedema (3). However, during acute exacerbations, sputum plugging may contribute significantly to the pathophysiology of asthma and this may be more difficult to treat.

The possible role of intensive humidification to liquefy inspissated secretions, in conjunction with physiotherapy to mobilize these secretions, has received little recent attention in the management of asthma. We report four cases of acute asthma which failed to reverse fully until intensive humidification of their respiratory tract was achieved. We used an ultra-Neb 99 ultrasonic nebulizer (De Vilbiss Health Care U.K. Ltd, Heston, Middx, U.K.) adjusted to produce an aerosol output of 3 ml min^{-1}, particle size being 0·5–5 μm. Sterile 0·9% sodium chloride warmed to room temperature was used in all instances. In all cases humidification was combined with regular physiotherapy to clear the increased volume of secretions. All patients had been receiving regular physiotherapy as part of standard treatment prior to the introduction of intensive humidification.

Case Reports

CASE 1

A 39-year-old woman, a non-smoker with asthma since childhood, presented with an acute severe attack precipitated by an upper respiratory tract infection. On admission she was too breathless to talk, centrally cyanosed and mildly pyrexial (37·8°C). Her pulse was 120 beats/min, pulsus paradoxus was present (15 mmHg), peak expiratory flow rate (PEFR) was unrecordable and PaO_2 and $PaCO_2$ on 40% O_2 by face mask were 7·4 kPa and 3·7 kPa respectively. Physical and radiological examination revealed collapse of the left lower lobe. She was treated intensively with nebulized and intravenous bronchodilators, intravenous and oral glucocorticosteroids and intravenous antibiotics. She made good initial progress over the first few days, PEFR rising to 200 l min^{-1}, but thereafter failed to improve further. Chest X-ray confirmed persisting left lower lobe collapse (Plate 1). This did not respond to routine humidification or to physiotherapy. She was therefore treated with inhaled normal saline produced by an ultrasonic nebulizer. After a test dose to exclude aerosol-induced bronchoconstriction, she was encouraged to inhale the ultrasonically produced normal saline via a mouthpiece for as much of each day as was possible. During this time she received regular physiotherapy. Over the next 48 h she produced a large quantity of thick green sputum, her breathlessness improved and her PEFR rose to 340 l min^{-1} and her chest X-ray returned to normal. Investigations revealed no eosinophilia and an absence of precipitating antibodies for *Aspergillus fumigatus*.

CASE 2

A 21-year-old man with lifelong asthma was admitted with an infective exacerbation. He was apyrexial but producing a small amount of green sputum. There was no tachycardia or pulsus paradoxus, but he had diffuse loud expiratory wheezes and his PEFR was reduced from his normal value of 550–660 to 220 l min^{-1}. Arterial blood gases breathing room air revealed a PaO_2 of 5·2 kPa. Chest X-ray

Received 1 June 1992 and accepted in revised form 11 January 1993.

0954-6111/94/050387+03 $08.00/0

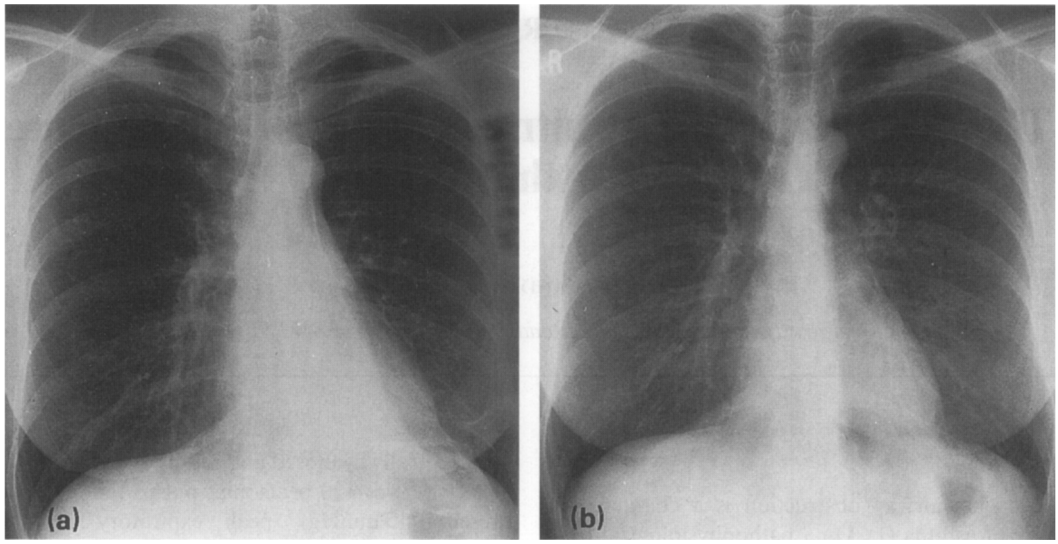

Fig. 1 Chest radiograph of patient 1 showing (a) left lower lobe collapse and (b) re-expansion of the left lower lobe after treatment with ultrasonically nebulized normal saline.

showed left lower lobe collapse. In the past he had required bronchoscopy to clear inspissated secretions producing right upper lobe collapse. There was no eosinophilia and *Aspergillus fumigatus* precipitins were absent. Despite intravenous antibiotics, maximal oral and parenteral treatment of his asthma, and physiotherapy he failed to fully recover having made initial progress over the first 2 days. His PEFR remained at 380–450 l min^{-1}. After a test dose of ultrasonically nebulized normal saline to exclude aerosol-induced bronchoconstriction, he was treated with this for several hours each day. Regular chest physiotherapy was continued. Over the next 48 h he expectorated a large quantity of sputum and his PEFR rose to over 600 l min^{-1}. His left lower lobe collapse resolved radiologically.

CASE 3

An 80-year-old non-smoker with a 30-year history of asthma was admitted with a 3-week history of worsening breathlessness. She was mildly pyrexial, had a tachycardia of 110 beats/min, 20 mmHg of pulsus paradoxus and loud expiratory wheezes. Her PEFR was 60 l min^{-1}, and her chest X-ray did not reveal any focal lesion. Her PaO_2 and $PaCO_2$ on 40% inhaled oxygen were 14 kPa and 8·5 kPa respectively. She was treated with broad spectrum intravenous antibiotics, maximal oral and parenteral bronchodilators and glucocorticosteroids and physiotherapy. Despite these measures, she remained breathless and wheezy. She subsequently complained of difficulty expectorating her sputum and was treated with ultrasonically nebulized normal saline. This resulted in moderate sputum production and considerable improvement over the next 72 h, her PEFR rising to 200 l min^{-1}.

CASE 4

A 59-year-old man with longstanding atopic asthma, was admitted with an infective exacerbation which had failed to respond to oral antibiotics and glucocorticosteroids as an outpatient. He was dyspnoeic at rest, apyrexial, his pulse was 100 min^{-1} there was no pulsus paradoxus, loud inspiratory and expiratory wheezes were present and his PEFR was 100 l min^{-1} (best 360 l min^{-1}). Arterial blood gases breathing room air showed a PaO_2 of 9·7 kPa and a $PaCO_2$ of 4·4 kPa. There was no focal lesion on the chest X-ray. His antibiotic was changed, the dose of oral prednisolone was increased, and he was treated with nebulized bronchodilators and received physiotherapy. Despite an initial response to these measures, he failed to improve further. He was therefore treated with ultrasonically nebulized normal saline. After 24 h he was able to expectorate several plugs of sputum and thereafter a considerable volume of infected sputum. His clinical status consequently improved, his PEFR rising to 325 l min^{-1}, and he made a full recovery.

Discussion

In these four cases full resolution of the acute asthmatic attack was not achieved until the introduc-

tion of therapy aimed at liquefying and mobilizing inspissated secretions. In two cases (1 and 2) the site of sputum retention was most significant in the left lower lobe, whereas in the other two instances there was no evidence of a localized effect, and its was presumed that there was widespread and diffuse peripheral involvement. Nevertheless, the failure of these last two cases (3 and 4) to fully respond until the introduction of humidification and physiotherapy, and the occurrence of resolution *pari passu* with the production of a large volume of sputum, is putative evidence that there was indeed widespread obstruction of peripheral airways by inspissated secretions.

Certain aspects of the use of ultrasonically nebulized solutions for humidification deserve emphasis. We were careful to use only normal saline in view of the proven ability of hypo and hypertonic solutions to elicit bronchoconstriction in asthmatic subjects (4) and we did not use cooled solutions since ultrasonic nebulizers produce mist at ambient temperature and cold solutions might have provoked further airways narrowing. In addition, subjects were carefully observed during the initiation of therapy to ensure that it did not produce any deterioration in their condition and humidification was always used in conjunction with physiotherapy to mobilize the liquefied secretions, in view of the possibility that the increased volume of secretions might otherwise accumulate and increase airways obstruction (5). Finally, since at high volume outputs (3–6 ml min^{-1}) it is possible that the dense aerosol generated by ultrasonic devices might increase the work of breathing, we used an output of <3 ml min^{-1}.

This study has not compared humidification by ultrasonic nebulizer with humidification by jet nebulizer which may be just as effective. The only difference between the two is the 2–10 fold greater output delivered by ultrasonic devices, due largely to the larger particle size produced by them (5–7 μm volume median aerodynamic diameter compared with 2–3 μm). This may affect the site of particle deposition.

The increased sputum clearance produced by humidification is probably due to a number of factors. Humidification may alter the depth and viscosity of the periciliary layer to more optimum values (6) and it may increase the efficiency of the forced expiration technique (FET) used in physiotherapy, and of the cough mechanism. It may also improve the facility with which sputum can be 'sheared' from the airway wall by the FET or by cough.

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