Commentary

Treatment of atelectasis: where is the evidence?

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See related research by Hendriks *et al.* in this issue [http://ccforum.com/content/9/4/R351]

Abstract

Lobar atelectasis is a common problem caused by a variety of mechanisms including resorption atelectasis due to airway obstruction, passive atelectasis from hypoventilation, compressive atelectsis from abdominal distension and adhesive atelectasis due to increased surface tension. However, evidence-based studies on the management of lobar atelectasis are lacking. Examination of air-bronchograms on a chest radiograph may be helpful to determine whether proximal or distal airway obstruction is involved. Chest physiotherapy, nebulised DNase and possibly fibreoptic bronchoscopy might be helpful in patients with mucous plugging of the airways. In passive and adhesive atelectasis, positive end-expiratory pressure might be a useful adjunct to treatment.

In this issue of *Critical Care*, Hendriks and colleagues [1] report on the use of nebulised or endotracheal DNase in paediatric patients with atelectasis. Pulmonary atelectasis is one of the most common abnormalities encountered on chest radiography and is caused by a variety of processes. These include: resorption atelectasis caused by resorption of alveolar air distal to obstructing lesions of the airways; adhesive atelectasis from increased surface tension and surfactant deficiency after ventilator-associated pneumonia; passive atelectasis caused by diaphragmatic dysfunction, or hypoventilation; compressive atelectasis due to space-occupying intrathoracic lesions or abdominal distension; and cicatrisation atelectasis due to pulmonary fibrosis [2]. Thus, when evaluating a patient with atelectasis, it is important to understand the mechanism, cause and functional significance of the atelectasis in that patient before possible treatment strategies can be developed, because no single therapy is likely to be successful in all forms of atelectasis.

On review of the literature it becomes apparent that there is a complete lack of evidence-based studies to guide the management of this common problem. Treatment modalities that have been described include chest physiotherapy [3], bronchodilators [3], fibreoptic bronchoscopy [4], DNase [1], positive end-expiratory pressure [5] and surfactant [6].

Chest physiotherapy is the traditional first-line therapy for atelectasis [4]; however, even for this basic therapy, evidence is lacking: there are only two published studies [7,8]. In 57 ventilated children, chest physiotherapy with saline lavage and simulated cough was successful in improving lung expansion in 84% of patients [7]. If physiotherapy fails, further examination of the chest radiograph to identify the level of air bronchogram may be helpful to identify whether airway obstruction is the cause and to determine whether proximal lobar or distal bronchi are involved [4]. Fibreoptic bronchoscopy to aspirate secretions has been used in the management of proximal airway obstruction, and has been found to resolve atelectasis successfully in 26 of 35 (74%) paediatric intensive care patients [9]. However. in a small randomised control trial, fibreoptic bronchoscopy did not improve the rate of resolution of volume loss in comparison with chest physiotherapy, and it may have adverse effects on intracranial pressure [8]. Nebulised bronchodilators are traditionally recommended for the management of atelectasis [3]. In patients with acute bronchoconstriction, a bronchodilator may increase airway diameter and hence improve secretion clearance, but there are no published studies evaluating its use in the management of atelectasis in asthmatic or non-asthmatic patients. In infants and children with bronchiolitis, nebulised adrenaline (epinephrine) to decrease airway mucosal oedema and hence increase airway diameter may be more beneficial than bronchodilators [10].

Nebulised or direct tracheal application of DNase reduces the viscoelastic properties of purulent airway secretions by breaking down the highly polymerised deoxyribonucleic acid [1]. Reducing the viscosity of the secretions makes them easier to clear, and DNase may thus reduce mucous plugging of airways and hence improve atelectasis. Again, there are no

DNase = deoxyribonuclease.

randomised control trials evaluating its use in the management of atelectasis, but there is one randomised trial of 75 infants with respiratory syncytial virus bronchiolitis, showing an improvement in chest radiograph scores in the patients given nebulised DNase [11]. For the management of atelectasis, there are five small published case series describing the successful use of DNase in one to five patients [1].

Hendriks and colleagues [1] now describe the use of DNase in the largest retrospective case series published so far, involving 25 children with persistent atelectasis despite physiotherapy and bronchodilators. In this study, 68% of patients improved after DNase administration. The lack of improvement in all the patients might have been partly due to the diverse aetiologies and predisposing factors present which included airway malacia, psychomotor retardation, neuromuscular disease, cardiovascular disease, bronchiectasis, and chronic lung disease. DNase is more likely to improve atelectasis due to mucous plugging of the airways, and some of these patients would have had other mechanisms of atelectasis such as passive and adhesive-induced atelectasis. Also of note is the observation that direct tracheal administration of DNase resulted in deterioration in three patients due to presumed rapid mucous mobilisation as a result of the higher delivered dose. This did not occur after nebulised administration of the DNase, which suggests that if direct tracheal administration is used, a small dose should be tried initially.

For atelectasis not due to mucous plugging of the airways, increased end-expiratory pressure has been used and resulted in complete resolution of lobar atelectasis in four patients [5] and re-expansion of atelectasis in experimental studies [12]. Atelectasis in 12 ventilated adults was associated with increased total protein, inflammatory markers and reduced surfactant in the bronchoalveolar lavage fluid, suggesting increased alveolar-capillary permeability, severe surfactant abnormalities, and signs of local inflammatory reaction [13]. This suggests a possible role for surfactant, and it has been used successfully to re-expand left lobar atelectasis in an adult with asthma [6]. It is curious that surfactant has not been used more extensively in the management of atelectasis; however, the traditional volumes used (4 ml/kg) are large, resulting in increased expense. Small volumes may be equally effective: 0.5 ml/kg fluorocarbon facilitates lung recruitment by reducing surface tension and ungluing adherent lung surfaces in saline-lavaged rabbits [14].

Overall, however, it is clear that there are very few published studies available to guide our management of lobar atelectasis, which is a common complication in critically ill patients; further studies are urgently needed. Hendriks and colleagues [1] are to be commended in attempting to fill this void.

Competing interests

The author(s) declare that they have no competing interests.

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