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BRIEF REPORT

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Surfactant replacement therapy in acute respiratory distress syndrome from viral pneumonia

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Abstract A modified natural surfactant was administered to a patient with life-threatening adult respiratory distress syndrome caused by viral pneumonia. Subsequently, there was a marked improvement in gas exchange. In order to assess the mechanism for improved oxygenation, computed tomography of the lungs was done. Quantitative analysis of the scans taken before and after surfactant administration indicates that improvement in gas exchange was largely due to the expansion of underinflated and collapsed lung areas. Although this is a single case report, it provides insight into the possible beneficial effect of instilled surfactant in severe respiratory distress from viral pneumonia.

Key words ARDS · Pneumonia · Pulmonary surfactants · Surfactant replacement therapy

Introduction

The value of surfactant replacement therapy in the treatment of the acute respiratory distress syndrome (ARDS) in the adult remains unclear [1, 2]. Thus, there is a need for more information which can help us to better understand the response of the lungs to surfactant replacement therapy. In this case report we provide new information on the effect of instilled surfactant in a patient with life-threatening ARDS caused by viral pneumonia by using quantitative analysis of computed tomography (CT) of the lung.

Case report

A 3-year-old boy was admitted to our intensive care unit for weaning from ventilatory support. His diagnosis was croup and ARDS caused by pneumonia. We supported spontaneous breathing activity by biphasic positive airway pressure ventilation (BIPAP), because this mode allowed unrestrained spontaneous breathing in a continuous positive airway pressure system at different pressure levels [3]. On the following day, blood gas values were worse and chest X-ray showed deterioration, requiring increased ventilatory support. CT performed the next day showed atelectasis and underexpanded areas and infiltrates in the right and left lung (Table 1, day 3). To reduce ventilation-perfusion inequality, we decided to rotate the boy from the supine to the prone position every 12 h. Two days later CT showed no reduction in atelectasis or in underexpanded areas or infiltrates (Table 1, day 5).

On the following day our main concern was the deterioration in gas exchange which was regularly seen after a change in body position. In an attempt to prevent this complication, with the boy in the prone position, we instilled 3 g of a modified natural surfactant (Curosurf, Chiesi Farmaceutici, Parma, Italy, 200 mg/kg body weight, suspended in 37.5 ml saline) into the five lung lobes via fiberoptic bronchoscope. One hour after surfactant replacement therapy gas exchange and dynamic lung compliance were basically the same. However, when the boy was rotated to the supine position, a lasting improvement in gas exchange was found. CT showed a reduction in atelectasis and underexpanded areas, while infiltrates were still present (Table 1, day 6+5 h) (Fig. 1 a, b).

Nine days later parainfluenza 2 virus was confirmed as the agent causing the pneumonia. A 160-fold increase in antibody titer to this virus was found in the patient's serum. No bacterial growth was detected in either tracheal or bronchial aspirates.

Discussion

In this patient with life-threatening ARDS caused by viral pneumonia, impaired gas exchange was successfully re-

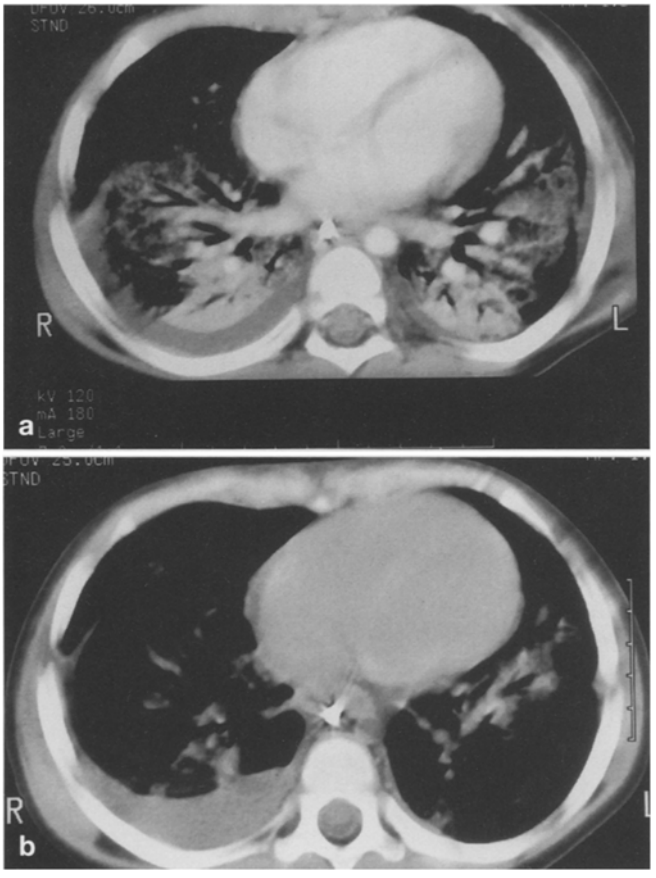

Fig. 1 Computed tomography of the lungs in a patient with acute respiratory distress syndrome of the adult (ARDS) caused by viral pneumonia before **a** and after **b** surfactant replacement therapy. Scan taken between carina and diaphragm level

stored by surfactant replacement therapy, as recently described (for a review, see Lachmann and Gommers [4]).

Quantitative analysis of CT performed 5 h after the administration of surfactant showed a dramatic reduction in noninflated lung area between the carina and the diaphragm. This reduction occurred in favour of poorly and normally inflated lung areas when compared to CT the day before. Similar results with even more pronounced reductions in non-inflated lung areas were found with CT between lung apex and carina (55 to 21%), at carina level (57 to 21%), and right above the diaphragm (55 to 15%). This finding, in conjunction with the improvement in gas exchange at constant ventilator settings, indicates that surfactant administration helped to restore impaired gas exchange in our patient by the recruitment of atelectatic and underexpanded alveoli.

Improvement in lung volume within 2 h of surfactant administration has been reported for preterm infants [5]. In that study, improvement in lung volume was attributed to increased alveolar distention rather than to recruitment of functional alveoli. Overinflation can be assessed with CT, showing an increase in lung area with a CT density between −900 and −1000 Hounsfield units [6]. We did not detect such low CT densities in our patient after surfactant administration. Thus, it is unlikely that alveolar overdistention was the cause of the improvement in gas exchange in our patient.

In contrast to gas exchange, dynamic lung compliance did not improve within 5 h. This has been described previously in adults with ARDS [1]. A likely explanation is that surfactant not only aids in recruitment of alveoli but also moves them up to the plateau of the pressure/volume curve at constant ventilator settings. As a result, the net dynamic lung compliance change could remain zero [7].

ARDS lungs typically show areas of increased density in CT sections [8]. Recruitment of some of these areas can be achieved by body positioning [9]. Therefore, we administered pulmonary surfactant before rotation, assuming that it would spread into newly recruited areas, contribute to the formation of a stable monolayer, and thereby help to prevent the recollapse of these structures that is regularly seen after change in body position [9]. When the patient was rotated 1 h after surfactant administration, a lasting improvement in gas exchange was found. This was never observed in the days before. It is therefore possible that in this patient conditioning of the lungs for surfactant replacement therapy by changes in body position further contributed to his clinical improvement.

The results from this pediatric patient with ARDS caused by viral pneumonia suggest that a bolus dose of 200 mg/kg body weight of a modified natural surfactant is capable of providing sustained improvement in gas exchange by recruitment of atelectatic and underexpanded lung areas. It is also possible that in ARDS a large percentage of noninflated or poorly inflated areas might be more accessible to surfactant delivery by using different body positions to maximize the effect of surfactant replacement therapy.

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Table 1 Ventilator settings and quantitative analysis of lung inflation state from CT scans before and after surfactant replacement therapy (*BIPAP* biphasic positive airway pressure, FIO_2 fractional inspired oxygen, PaO_2 arterial partial pressure of oxygen, C_{dyn} dynamic lung compliance [expiratory tidal volume/($pressure_{high}$ – $pressure_{low}$)])

	Day after admission to ICU						
	3	5	6 (surfactant replacement)				7
			−4 h[c]	+1 h	+3 h	+5 h	+23 h
BIPAP[a]							
$Pressure_{high}$ (mbar)	22	28	29	29			25
$Pressure_{low}$ (mbar)	10	8	10	11			11
Pressure $time_{high}$ (s)	1.5	1.4	1.4	1.4			1.4
Pressure $time_{low}$ (s)	0.5	0.5	0.5	0.5			0.5
FIO_2	0.80	0.80	0.73	0.73	0.60	0.50	0.40
PaO_2/FIO_2	115	124	141	137	200	204	210
C_{dyn} (ml/mbar)	9.6	5.9	6.5	6.7	6.7	6.7	8.9
CT (% total lung area)[b]							
Noninflated	54	53				28	
Poorly inflated	41	44				67	
Normally inflated	5	3				5	
Body position	prone	supine	prone		supine		prone
Body temperature (°C)	41	39	38	38	41	40	37
White blood cell count (g/l)	6800	8300	8100		7100		9900

[a] Ventilatory mode similar to pressure-controlled, time-cycled mode. Pressure levels ($pressure_{high}$ and $pressure_{low}$) and phase duration (pressure $time_{high}$ and pressure $time_{low}$) are adjusted independently. Switching between the two pressure levels determines the contribution to mechanical ventilation

[b] Analysis is shown only for scans taken between carina and diaphragm level. Throughout imaging procedure the patient was apnoic (airway pressure maintained constant; pressure level set equal to mean airway pressure during BIPAP ventilation). Normally inflated lung area is defined as the number of CT pixels per image, which range in density between −1000 and −500 Hounsfield units (H); poorly inflated lung area: pixels range between −500 and −100 H; noninflated lung area: pixels between −100 and +100 H. For comparison: CT density of air; −1000 H; water, 0 H; and bone + 1000 H. For further details of analysis see Gattinoni et al. [6]

[c] −4 h: 4 h before surfactant replacement therapy was started; +1 h to +23 h: after surfactant replacement therapy was finished

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