A 58-year old man presented to the hospital with right side upper and middle lobe pneumonia. Despite active therapy his condition progressively deteriorated. He developed acute anuric renal failure, acute pancreatitis and coagulopathy.

Four days later he was transferred to the Intensive Care. His condition deteriorated further with the development of respiratory distress, hypoxemia, mixed decompenesated severe acidosis. He remained normotensive at all stages. Chest CT scan revealed extensive right upper and middle lobe parenchymal consolidation with patent right main bronchi, normal picture of the left lung (Fig. 1). Sg catheter was inserted. Measurements of haemodynamic parameters suggested septic picture with high cardiac output and low systemic vascular resistance. Transoesophageal echocardiography suggested hypervolemia, adequate myocardial function and no valvular lesions.

Patient was intubated with orotracheal tube. BIPAP-SIMV ventilation was initiated (Dräger Evita 2 ventilator) with initial FiO₂ requirements of 0.5 and PEEP of 10 mbar, I:E ratio 1:1. His Pinsp. was maintained below 27/28 mbar allowing 800 - 950 mL VT to be delivered. CVVHDF was initiated. Despite continuous massive antibiotic therapy his right lung did not improve. Respiratory failure progressed with FiO₂ requirements increased over 0.65. No pathogen was isolated at this stage.

Two days later orotracheal tube was exchanged for the left- sided double lumen endobronchial tube. Unsynchronised differential lung ventilation was initiated (Dräger Evita 2 ventilator for the right lung and Dräger Evita 2 dura ventilator for the left lung). The assessment of lung mechanics revealed significant differences between lungs.

Right lung compliance was approximately 35 mL/mbar. Left lung compliance was measured appr. 85 mL/mbar. Ventilation was started as following: for the right lung: SIMV-BIPAP, I:E - 2:1, RR - 5/min, FiO₂ - 0.65, PEEP 15 mbar, PInsp. 30 mbar; for the left lung: SIMVAutoFlow, I:E - 1:1, RR 10/min, FiO₂ - 0.65, PEEP 5 mbar, VT = 0.5 L.
This allowed the delivery of 300 - 350 mL VT into the right lung and 500 - 550 mL VT into the left lung. During the next 8 hours it became possible to maintain adequate blood gases weaning FiO₂ down to 0.45, Pinsp. down to 24 mbar for the right lung; FiO₂ down to 0.45 and Pinsp. down to 22 mbar for the left lung. During the following 24 hours right side ventilatory parameters were not changed but compliance progressively increased and as a result tidal volumes raised. Left side Pinsp. was further decreased to 18 mbar. During the following 24 hours FiO₂ for the right lung was further decreased to 0.4. 72 hours after the initiation of the differential lung ventilation the compliance on both sides became virtually equal at approx. 80 mL/mbar. Chest CT scan was repeated (Fig. 2) and showed significantly diminished consolidation in the right lung. Left lung remained normal. Double-lumen endobronchial tube was again exchanged for the single lumen endotracheal tube. SIMV BIPAP ventilation was continued. However FiO₂ had to be increased to 0.5 following the change in ventilation.

Candida albicans and methicillin resistant staphylococcus aureus were isolated from the bronchial lavage and were treated. Patient consequently had another 5 days of ventilatory support after which his lungs fully recovered and he was successfully extubated. He remained in ICU for 2 more weeks. Sepsis and pancreatitis settled. He developed severe gastrointestinal bleeding (probably from the pancreatic duct) which also was eventually stopped. After 23 days in intensive care renal failure remained to be the only problem. CVVHDF was terminated and patient was transferred to the chronic dialysis unit.

This case illustrate the importance of a special approach to the intensive care patients with unilateral severe lung disease. The mechanics and physiology of the diseased lung is very different from the healthy lung. Multiple studies indicated that inappropriate ventilatory strategy creates further severe damage to the initially unaffected zones of the lung. It is therefore logical to use different regimes of ventilation for each lung.
In this particular case we used a BIPAP-SIMV mode for the affected lung. This allowed to have low frequency ventilation for the constantly expanded lung with the peak airways pressure below the damaging level. As a result of such strategy it was possible to avoid any further volutrauma to the sick lung and to create healing rest conditions. The unaffected lung was ventilated in SIMV AutoFlow mode with normal frequency and low PEEP. This allowed to provide optimal oxygenation of the body without damaging ventilatory effect to the unaffected lung. Such combination produced fast and significant improvement in pulmonary gas exchange and in total body oxygen delivery without the expense of ventilatory complications. In this case, initiation of differential lung ventilation provided a significant and rapid improvement in clinical and radiological findings in the affected lung’s tissue.

Differential lung ventilation certainly carries some problems and potential complications. The most frequent problem is misplacement of the double-lumen endobronchial tube, especially during the rotation of the patient. However in severe unilateral lung disease benefits of this modality can outweigh the potential problems. It requires careful mode selection with constant reassessment of the Ventilatory modes, such as BIPAP SIMV and AutoFlow became treating rather “time buying” strategies for the injured lungs.

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