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Non-invasive management of acute respiratory distress syndrome related to *Influenza A* (H1N1) virus pneumonia in a pregnant woman

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A 38-year-old pregnant woman was admitted at 31 weeks' gestation to the intensive care unit on 4 August 2009 for fever, chills, cough, vomiting and weakness of 1-week duration. The core temperature was 39.8°C, the respiratory rate 40 cycles/min, the oxygen saturation 92% on 10 l/min oxygen, the heart rate 135 beats/min and the arterial blood pressure 100/50 mmHg. Physical examination revealed a bloody expectoration and bilateral crackles. A chest X-ray showed extensive bilateral opacities. Partial pressure of oxygen (PaO_2) was 116 mmHg and carbon dioxide ($PaCO_2$) 32 mmHg on 10 l/min oxygen. *Influenza A* pneumonia was suspected, and oseltamivir (75 mg tid) was administered on 5 August in association with cefotaxim, spiramycin and linezolid. A nasopharyngeal swab specimen was positive for *Influenza A* (H1N1) virus, using real-time reverse transcription-polymerase chain reaction (rRT-PCR). Sputum and *Streptococcus pneumoniae* and *Legionella pneumophila* urinary antigen tests were negative. Severe acute respiratory distress syndrome (ARDS) rapidly developed, and non-invasive positive pressure ventilation (NIPPV) was administered continuously during the next 72 h through a facial mask, with a FiO_2 of 1, a 14 cmH_2O maximum positive airway pressure and a 5 cmH_2O positive expiratory pressure to maintain the pulse oximetry $\geq$94%. Arterial oxygenation improved slowly with PaO_2/FiO_2 ratio ranging from 98 to 184. There was no left heart dysfunction on echocardiogram. Fetal monitoring was satisfactory. After 3 days, NIPPV was administered intermittently. A repeated nasopharyngeal swab specimen was negative. Oseltamivir and antibiotics were stopped. On 15 August, the shortness of breath worsened, and intermittent NIPPV was administered again. A CT scan demonstrated bilateral ground-glass opacities (Fig. 1). A fiberoptic bronchoscopy with bronchoalveolar lavage (BAL) was performed. Bronchial mucosa was normal; BAL was macroscopically haemorrhagic with 170,000 cells per ml (neutrophils 77%, lymphocytes 11%, macrophages 10%, eosinophils 7%), a very low residual positivity for *Influenza A* (rRT-PCR) and no bacteria. Broad-spectrum antibiotics and oseltamivir were again administered together with intravenous methylprednisolone (2 mg/kg/day) on 17 August. As the clinical status did not improve, a multidetector CT angiography was performed showing a right upper lobe pulmonary embolism and fibrosis (Fig. 2). Finally, a cesarean delivery was performed, under spinal and peridural analgesia and high-flow oxygen. The infant was born in good health, with no influenza infection. The mother's clinical status improved slowly thereafter.

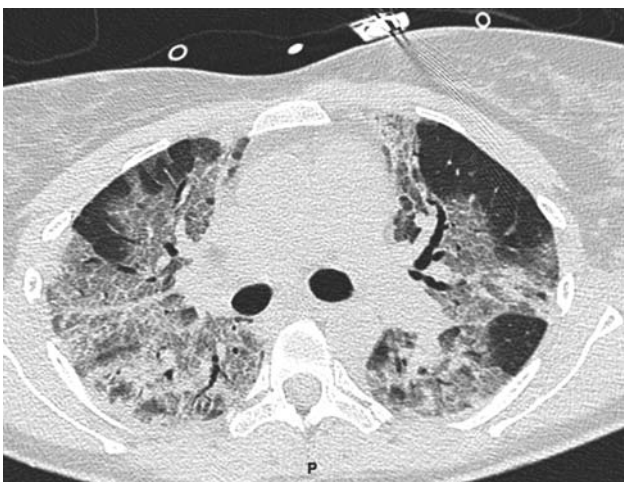

Fig. 1 Chest computed tomography performed on 16 August demonstrating bilateral ground-glass confluent opacities

A mortality rate reaching 60% has been reported in patients requiring mechanical ventilation (MV) for H1N1-related ARDS [1]. Six deaths in pregnant women with ARDS were recently reported to the US Centers for Disease Control and Prevention, five of whom had undergone a caesarean delivery [2]. Although NIPPV may be associated with a reduction of endotracheal intubation and mortality rates in ARDS [3], it has not been evaluated during pregnancy. Neither the optimal time for delivery nor the modality is clearly defined in the absence of obstetrical indications [4], although cesarean delivery appears more appropriate in case of

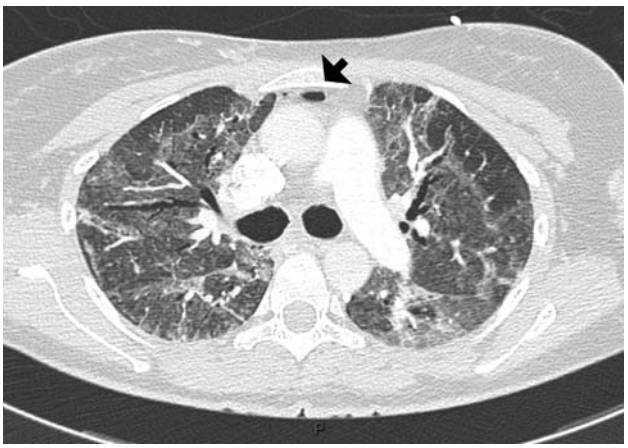

Fig. 2 Multidetector CT angiography performed on 21 August demonstrating parenchymal lesions suggestive of fibrosis development, with worsened bilateral ground-glass opacities, mild-to-moderate dilatation of the bronchi and air in both anterior mediastinum and cervical soft tissue (*arrow*)

respiratory failure. This observation highlights the multidisciplinary therapeutic approach to management ARDS during pregnancy and suggests that NIPPV may be useful in selected women with isolated acute respiratory failure. The role of steroids for H1N1-related ARDS remains to be determined [5].

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