

radiography revealed infiltrates in both lung fields. The diagnosis of severe hypoxic respiratory failure due to an overwhelming pneumonia (influenza type A) with acute respiratory distress syndrome (ARDS) was established. A nasal swab was positive for influenza type A. Treatment with oseltamivir was not started since symptoms had been present for more than 48 hours. Ventilatory support was started with high flow nasal therapy (Optiflow[®]) with an inspired oxygen fraction (FiO₂) of 50%. Cardiocotography (CTG) showed a normal fetal heart rate and prenatal ultrasound was reassuring. During the first hours of hospitalization, FiO₂ had to be augmented to obtain acceptable arterial oxygen saturations. Eventually, the patient had to be intubated to allow invasive mechanical ventilation. Arterial blood gas analysis before intubation showed: pH: 7.25, PaCO₂: 46.0 mmHg, PaO₂: 74.2 mmHg, bicarbonate: 20.1 mmol/L, and base excess: 6.9 mmol/L (FiO₂ = 100%). After rapid sequence induction, lung protective ventilation was pursued using a tidal volume of 6 ml/kg ideal body weight, a respiratory rate of 26/min, a FiO₂ of 95%, a positive end-expiratory pressure of 15 cmH₂O and a plateau pressure of 33 cmH₂O. Lung protective ventilation and recruitment manoeuvres failed to improve gas exchange. Therefore, the patient was intermittently ventilated in prone position. Despite these measures and initial improvement, the patient progressed to global respiratory failure on day 5. Peak systolic velocity of the fetal middle cerebral artery measured by ultrasound was above 1.05 Multiples of Median, which could be suggestive for fetal anemia, but no signs of fetal-maternal transfusion were found. Dexamethasone had already been started on day 1 to accelerate fetal lung development in anticipation of possible preterm delivery. Because of further maternal respiratory deterioration and signs of fetal distress, a multidisciplinary team agreed to perform an emergency caesarean section before the initiation of veno-venous extracorporeal membrane oxygenation (VV-ECMO) on gestational age of 27w4d, delivering a boy with a birth weight of 1220 g, Apgar 2/5/5, and arterial pH 7.35. Despite delivery, respiratory function further deteriorated, so that the patient was again intermittently ventilated in prone position and ultimately an additional jugular ECMO cannula was inserted on day 11. On day 17, the patient was successfully weaned from ECMO, and 3 days later she was weaned from the ventilator to high flow nasal oxygen. Despite two episodes of epilepsy provoked by several cerebral microbleeds, the patient had a perfect neurological evolution four months later and the baby did very well.

3. Discussion

About 0.1–0.2% of all pregnancies are complicated by respiratory failure,¹ evoked by either pregnancy-related complications or pre-existing conditions. Since maternal hypoxemia and hypercapnia may cause detrimental fetal complications, extensive knowledge of therapeutic options is essential. Several physiologic changes during pregnancy complicate the management of the pregnant patient in respiratory failure. Functional residual capacity (FRC) is reduced by 10–25%. Oxygen consumption is increased during pregnancy, leading to rapid development of hypoxia due to hypoventilation, apnoea or impaired gas exchange.¹ Downregulation of the maternal immune system is necessary to tolerate paternally derived fetal antigens, but leads to increased susceptibility to infections.

ARDS is one of the leading causes of non-obstetric mortality during pregnancy.¹ Physiologic changes in maternal circulation including an increase in circulating plasma volume, capillary leak, and a reduced serum albumin, facilitate the development of ARDS.^{1,2} ARDS-related mortality and morbidity were found to be comparable to that of non-pregnant women. This has been attributed to the young age and lack of comorbidity in the majority of pregnant patients.^{1,2} ARDS is associated

with a high rate of fetal loss.²

Endotracheal intubation can be challenging due to hyperaemia and oedema of the upper airway, increased breast volume and the need for crush induction due to impaired gastric emptying with a high risk of aspiration.¹ During mechanical ventilation, hyperventilation and alkalosis should be avoided to prevent uterine vasoconstriction, as well as hypoventilation and hypercapnia in order to avoid fetal respiratory acidosis. Additionally, maternal PaO₂ should be kept greater than 70 mmHg to maintain fetal normoxia.¹

When conventional lung-protective mechanical ventilation strategies fail, prone positioning may be considered. However, prone positioning has obvious limitations in late pregnancy. An alternative approach includes the use of ECMO.^{3–5} VV-ECMO is used to improve oxygenation and/or treating hypercapnia when impaired gas exchange cannot be corrected with conventional therapy. By resting the lungs, VV-ECMO is a powerful tool to avoid ventilator-induced and oxygen-induced lung injury.^{3,5} According to Vaquer et al., the use of prone-ventilation before initiation of ECMO is associated with superior outcomes.⁵ Nevertheless, prolonged mechanical ventilation and delayed initiation of ECMO has deleterious effects on outcome. ECMO should therefore be initiated before irreversible lung injury has been established.⁵

A recent systematic review identified severe H1N1-influenza complicated with ARDS as the most common indication for ECMO during pregnancy.² In this review, the use of ECMO was remarkably safe for mother and fetus comparable to non-pregnant patients. Maternal and fetal survival rates were 77,8% and 65,1%, respectively. Hence, pregnancy is not a contraindication to ECMO.^{2,4} If maternal oxygenation remains impaired despite maximum support, delivery of the fetus should be considered.¹ Caesarean section can be performed before or during therapy with ECMO.⁴

4. Conclusion

Care of a pregnant patient in acute respiratory distress requires a multidisciplinary team approach. The altered physiology in pregnancy predisposes mother and child to the rapid development of hypoxia and respiratory failure, possibly leading to preterm labor or intra-uterine fetal demise. Therefore, intubation and mechanical ventilation should be considered early on. When conventional therapy fails, prompt initiation of VV-ECMO to improve oxygenation is safe and may be life-saving for both mother and child.

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