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LETTER TO THE EDITOR

A case of respiratory distress syndrome in a newborn immediately after induction of anesthesia

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To the Editor:

Respiratory distress syndrome (RDS) is a condition leading to hypoxemia and ventilatory failure soon after birth that is caused by the lack of sufficient surfactant due to immature pulmonary development. Surfactant is produced at approximately week 34 of pregnancy; therefore, infants born before 34 weeks of gestation are susceptible to RDS. Although surfactant is found in the lungs immediately after birth, its subsequent depletion can result in the onset of RDS approximately 2 h after birth [1]. Here, we report a case of RDS developing immediately after anesthesia induction in a newborn who underwent radical surgery for an omphalocele 3 h after birth.

The patient was a 3-h-old girl with a height of 44.5 cm and weight of 1,936 g. The fetus was diagnosed with an omphalocele and liver herniation at 22 weeks of gestation. The mother was delivered by emergency cesarean section due to threatened premature delivery. She was not administered steroids before cesarean section. The infant was delivered at 34 weeks 0 days of gestation. Apgar scores at 1 and 5 min after birth were 5 and 9, respectively. An omphalocele and liver herniation were observed in the abdomen; consequently, we elected to surgically place a silo over the omphalocele. The respiratory rate was 30 breaths/min, and oxygen saturation was 100 % under administration of oxygen at 1 L/min.

The infant had tachypnea in the absence of retracted breathing or grunting, and the oxygen saturation was 97 % under oxygen administration at 1.5 L/min prior to entering the operating room. The preoperative plain chest radiograph is shown in Fig. 1. For anesthesia, 0.02 mg of atropine, 6 μg of fentanyl, and 2 mg of rocuronium were administered intravenously. The oxygen saturation did not increase despite endotracheal intubation, and the airway pressure remained around 30 mmHg. We suspected RDS and therefore performed a second plain chest radiography (Fig. 1). The radiograph revealed a markedly reduced permeability of the lungs; the lungs also had a reticulonodular pattern, which is characteristic of RDS. The infant was diagnosed with Stage III RDS according to Bomsel's classification [2]. Upon diagnosis with RDS, 240 mg of surfactant [3] was promptly injected into the trachea. After 5 min, the oxygen saturation improved to 100 %. Intraoperative oxygen saturation did not decrease, and the infant was returned to the neonatal intensive care unit without extubation. The following day, plain chest radiography revealed improved permeability in the lung fields (Fig. 1).

The infant was 3 h of age when she entered the operating room and was susceptible to RDS. She had risk factors, such as premature birth, low birth weight, and delivery via cesarean section. However, we speculate that the development of RDS would be more a natural course in this case. Therefore, we do not consider that anesthesia induction played an important role in the development of RDS in this case, although the lung pathology immediately after anesthesia induction may be attributable to it. For newborns with risk factors for RDS who undergo procedures under anesthesia, RDS should be taken into

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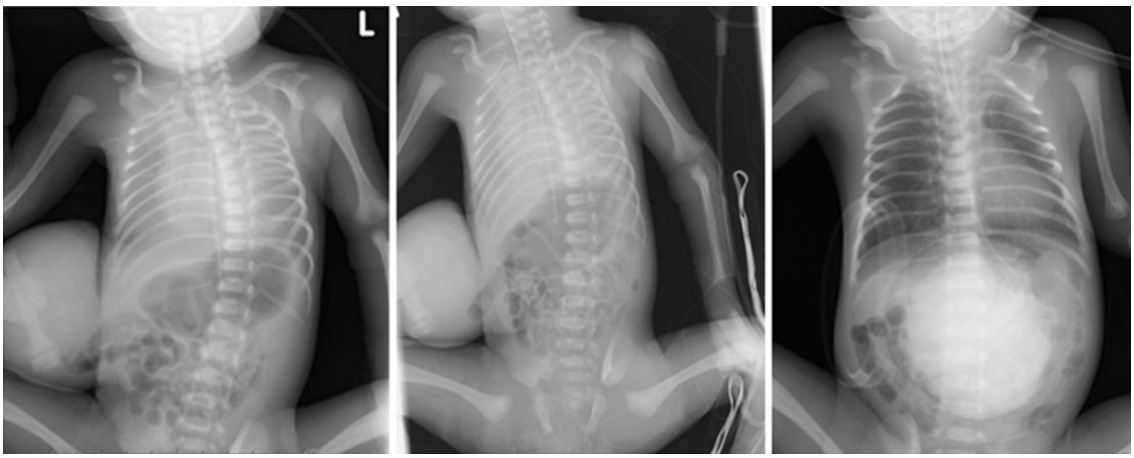

Fig. 1 Chest radiography before (*left*) and after induction of anesthesia (*middle*) and on the day following surgery (*right*). Two hours after birth there was a decreased permeability in the lung fields. After induction of anesthesia, the permeability of the lungs was markedly decreased. The lungs of the infant had a reticulonodular pattern, which is characteristic of RDS. The following day, a remarkable improvement in permeability could be seen in the lung fields on the radiographic scan

consideration in diagnosing hypoxemia, and surfactant must be prepared beforehand.

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