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Case Report

Severe surfactant-responsive respiratory distress syndrome in an early-term infant following pre-labor caesarean section: a case report

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ABSTRACT

Respiratory distress in infants of 37-38 weeks' gestational age (GA) is usually due to transient tachypnea of the newborn (TTN). However, primary surfactant-deficient respiratory distress syndrome (RDS) also occurs at this gestation, especially following pre-labor caesarean section, and can be severe. A 2990 g male infant was born at 37 weeks by emergency caesarean section for maternal scar tenderness without labor and without antenatal steroids. He had respiratory distress at birth (Silverman-Andersen score 7/10) and required continuous positive airway pressure (CPAP) and fraction of inspired oxygen (FiO_2) 0.90 and nasal intermittent positive-pressure ventilation (NIPPV). Point-of-care lung ultrasound (LUS) within 30 minutes of NICU admission demonstrated coalescent B-lines and bilateral posterior subpleural consolidations with static air bronchograms (score 12/18 on six zones; 28/36 on 12 zones), strongly favoring RDS. The decision to give surfactant was based on the clinical findings and LUS; subsequent chest radiography confirmed a white-out appearance. At 2 hours of life poractant alfa was given at 160 mg/kg with a planned intubation-surfactant-extubation (INSURE) approach and a subsequent fall in FiO_2 to 0.25. Extubation at 20 min was unsuccessful. After six hours of ventilation, he was extubated to NIPPV and weaned to room air by day 4. Repeat lung ultrasound after 24 hours demonstrated A-lines. Echocardiography ruled out pulmonary hypertension; sepsis screen and blood culture were negative. Early-term pre-labor caesarean delivery can be complicated by surfactant-responsive severe RDS rather than TTN. Bedside lung ultrasound favored RDS over TTN within minutes and guided the surfactant decision. A rapid fall in oxygen requirement after surfactant did not mean readiness for extubation.

Keywords: INSURE, Early-term infant, Point-of-care lung ultrasound, Respiratory distress syndrome, Surfactant, Caesarean section

INTRODUCTION

Respiratory distress syndrome (RDS) is mainly a disease of the preterm lung. The absence of adequate surfactant leads to increased surface tension in the alveoli, causing the alveoli to collapse at the end of every breath, stiffening the lungs, and forcing the baby to work harder and thus requiring more oxygen.[1] This lack of surfactant naturally decreases as gestational weeks increase. In a United States (US) cohort of 233,844 deliveries, 10.5% of infants delivered at 34 weeks had RDS compared with only 0.3% at 38 weeks; even at 37 weeks, the odds of RDS were three times higher than at 39-40 weeks.[2] National US data indicate that infants with GA 37-38 weeks and those born at 39 weeks or later each comprised 15.3% of all RDS.[3] Secondary surfactant deficiency can also arise from inactivation of surfactant after lung injury (infection or meconium aspiration). This neonatal acute respiratory distress syndrome (NARDS) has been reported in about 1.5% of neonatal intensive care unit

(NICU) admissions in international and Chinese multicentre cohort studies.[4,5] However, at term gestation, RDS is uncommon enough that it is often not the first diagnosis considered.

Immature lungs also occur at early term (37+0 to 38+6 weeks gestational age (GA)).[6,7] RDS was one of the main causes in a large cohort of neonates born at ≥34 weeks receiving mechanical ventilation, and the risk decreased with each completed week of gestation.[8] Elective caesarean delivery before the onset of labor adds to the risk; respiratory morbidity after pre-labor caesarean delivery at 37 weeks is several times higher than after vaginal birth or after caesarean at 39 weeks.[9-11] A good deal of this excess is explained by retained lung fluid. These babies miss the catecholamine surge needed for the activation of epithelial sodium channels that come with labor, so many are labeled as transient tachypnea of the newborn (TTN).[12] Giving antenatal corticosteroids before elective term caesarean section may reduce these admissions for respiratory distress.[13] Term infants with TTN also have low counts of lamellar bodies in gastric aspirates, suggesting that relative surfactant deficiency, in addition to retained lung fluid, may contribute to more prolonged illness in some of these infants.[14]

The issue at the bedside involves the challenge of distinguishing TTN, RDS, congenital pneumonia, and persistent pulmonary hypertension of the newborn (PPHN) in the initial hours of life. These conditions can be differentiated by LUS, as they have differing patterns, with a semi-quantitative LUS score correlating with oxygenation and predicting surfactant need.[15-20]

We report a case of a pre-labor lower segment caesarean section (LSCS) delivery of an early-term infant not exposed to antenatal steroids who developed severe RDS and rapidly responded to a single dose of surfactant. Point-of-care LUS performed within 30 minutes of admission was highly suggestive of RDS and guided the decision to give surfactant, with subsequent chest radiography confirming the diagnosis.

CASE REPORT

Maternal and birth history

The mother was G2P1L1 (second pregnancy; one previous delivery, by LSCS; one living child), with blood group B positive. Gestational age was assessed as 37 weeks by last menstrual period. Her antenatal ultrasound reports and blood investigations were unremarkable. She gave no history of diabetes in pregnancy and her oral glucose tolerance test (OGTT) was normal. She had not received antenatal corticosteroids. An emergency LSCS was done for tenderness over the previous caesarean scar, before labor had begun.

A male infant was born weighing 2990 g (appropriate for gestational age) and cried at birth. Apgar scores were 7 and 9 at 1 and 5 minutes, respectively. Within minutes he developed tachypnea, grunting and severe chest retractions, with a Silverman-Andersen score of 7/10 and was admitted to the neonatal intensive care unit (NICU).[21]

Initial respiratory course

Nasal continuous positive airway pressure (CPAP) was started at a positive end-expiratory pressure (PEEP) of 6 cm H_2O, but he needed an FiO_2 of 0.90 to keep his saturations in the target range (91-95%). Owing to increased work of breathing, he was moved to NIPPV. Despite this, his oxygen requirement remained high.

Imaging and other investigations

Point-of-care lung ultrasound (LUS), performed at the bedside by the treating neonatologist within 30 minutes of NICU admission, showed coalescent B-lines over both lungs and bilateral posterior subpleural consolidations with static air bronchograms (Figure 1A and 1B). We first used the six-zone protocol described by Brat et al (upper anterior, lower anterior, and lateral zones on each side), which we use routinely to guide surfactant decisions, giving a score of 12 out of 18.[17] Because the six-zone protocol does not examine the posterior (dependent) lung of a supine infant, we extended the scan to the 12-area protocol (anterior, lateral and posterior regions on each side, each divided into upper and lower areas).[22] The same 0-3 aeration grading was applied to every area: 0, A-lines only; 1, three or more well-spaced B-lines; 2, crowded or coalescent B-lines with or without consolidation limited to the subpleural space; 3, extended consolidation.[17] The extended score was 28 out of 36. Together with the clinical picture, these findings fitted RDS and the decision to give surfactant was made on this basis.[15,20] A chest radiograph, requested for confirmation, was obtained at HOL2 (hours of life) just before giving surfactant, which showed diffuse bilateral opacification, a white-out appearance (Figure 2), consistent with the LUS findings.

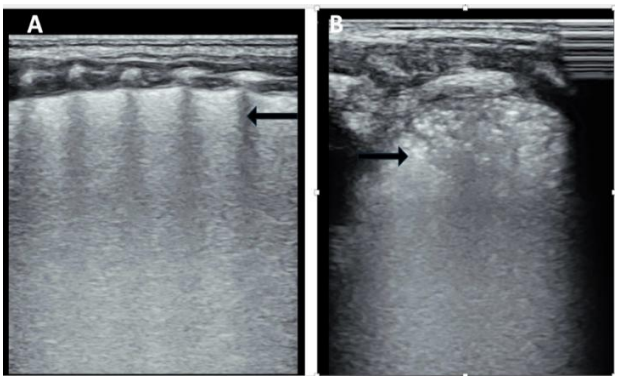


Figure 1: Lung ultrasound before surfactant. (A) Longitudinal view showing coalescent B-lines. (B) Transverse view showing subpleural consolidation (posterior lung field).

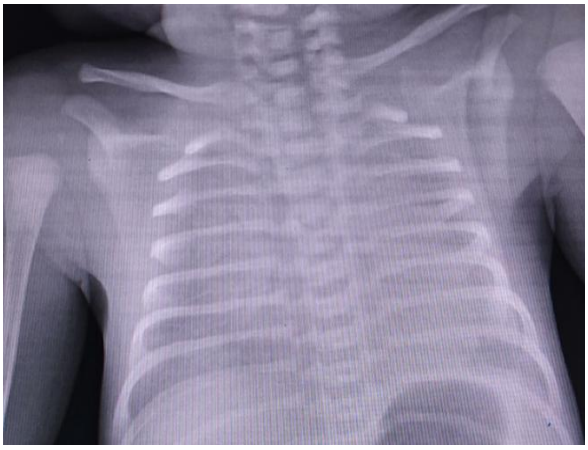


Figure 2: Chest radiograph obtained for confirmation at 2 hours of life prior to surfactant administration, showing diffuse bilateral opacification (white-out).

Functional echocardiography showed no pulmonary hypertension. A structurally normal heart was confirmed, and obstructive total anomalous pulmonary venous connection was specifically ruled out. Blood culture was taken before antibiotics were started. The sepsis screen was negative and the culture remained sterile. Cranial ultrasound was normal.

Surfactant and ventilation

At HOL2 baby was intubated and given poractant alfa (Curosurf) at 160 mg/kg once the surfactant had been procured, with the plan of extubating him straight back to non-invasive support (INSURE). His FiO_2 fell from 0.90 to 0.25 soon after the dose. He was extubated about 20 minutes later but failed to maintain adequate breathing on non-invasive support; his saturations fell, became cyanosed with increased work of breathing, and had to be re-intubated. He was then kept on mechanical ventilation for about six hours (assist control mode with peak inspiratory pressure (PIP) 20 cm H_2O, PEEP 6 cm H_2O, rate 40/min, inspiratory time 0.35s, and FiO_2 0.25). A venous blood gas on the ventilator, 1 hour after surfactant, showed adequate ventilation (pH 7.34, pCO_2 35 mmHg) with mild metabolic acidosis (bicarbonate 18.5 mmol/l, lactate 2.6 mmol/l).

Before the second extubation, he was on synchronized intermittent mandatory ventilation (SIMV) mode (PIP 16 cm H_2O, PEEP 6 cm H_2O, rate 40/min, inspiratory time 0.35s, and FiO_2 0.25) and had good spontaneous respiratory effort. He was then extubated to NIPPV (PIP 20 cm H_2O, PEEP 6 cm H_2O, FiO_2 0.25). Lung ultrasound 24 hours after surfactant showed the return of A-lines (Figure 3). As his breathing settled, he was weaned to room air on day 4 of life. He did not need a second dose of surfactant.

Other problems

Amikacin and cefotaxime were started after the blood culture was drawn. Because of increasing respiratory distress and suspected sepsis, cefotaxime was replaced with piperacillin-tazobactam at HOL8. Antibiotics were stopped after a total of five days, once the screen was negative and the blood culture sterile. He developed jaundice at HOL46 and received intensive phototherapy. He was initially kept nil by mouth.

Trophic feeds were initiated at HOL48 and increased as tolerated, reaching full enteral feeds by HOL82. Breastfeeding was initiated on day 4 of life after he was weaned to room air. He was discharged on breastfeeds with supplementary spoon feeds, with follow-up arranged as per unit protocol.

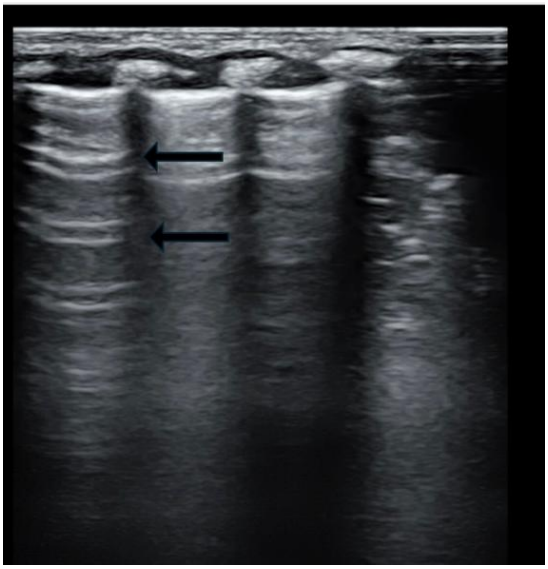


Figure 3: Lung ultrasound 24 hours post-surfactant, showing the return of A-lines.

DISCUSSION

This was an infant born at 37 weeks GA who became very sick immediately. Studies have shown clearly increased respiratory morbidity at early term with prelabor caesarean section.[9-11] Those studies looked at planned (elective) caesareans, not emergency caesareans, as in the case of our baby. The common factor is that both occurred prior to the onset of labor, which is thought to account for much of the risk.[12] Severe disease is not unknown in either situation; for example, in a Swiss series of 56 near-term and term infants ventilated for hyaline membrane disease following a caesarean section, disease severity was comparable whether the caesarean was an elective procedure prior to labor or if it was an emergency or occurred after the onset of labor; 27% received surfactant, seven met criteria for ECMO and one infant died, but notably there were no cases beyond 39 completed weeks.[23] This case is reported because of the severity of the illness. Surfactant-deficient RDS at 35 weeks or beyond is most often mild-to-moderate; of 117 such Qatari infants, only 6.8% had severe disease, and 26.5% received surfactant, most of whom needed a single dose.[24] Our patient, requiring FiO_2 of 0.90, was among

this severe minority and, as with most of the treated ones, responded to a single dose. In the early hours it would have been easily misdiagnosed as TTN, with a different management approach.

RDS vs TTN?

TTN is usually benign and self-limited with only a modest supplemental oxygen requirement and settles within 24-72 hours.[25] On LUS, TTN typically shows a clear difference between the upper and lower lung fields known as the “double lung point”, interstitial syndromes, disappearance of A-lines, and absence of consolidation.[16,26] RDS has the "white lung" pattern with coalescent B-lines, an irregular, thickened pleural line and the presence of sub-pleural consolidations with air bronchograms.[15,16,20]

Our patient had the second pattern. He also had a white-out chest radiograph and required 90% oxygen. LUS scores relate to oxygenation and can predict surfactant needs; an extended score (28/36) suggested that large areas of lung were poorly aerated.[17-19] Just after one dose of surfactant, his FiO_2 also fell from 0.90 to 0.25. Relative surfactant deficiency has been described in TTN as well, so this response is consonant with but not diagnostic of primary surfactant deficiency alone.[14] However, in an appropriate clinical picture, severe respiratory failure from birth, diffuse radiographic opacification, extensive loss of aeration on LUS with subpleural consolidation, and a marked fall in oxygen requirement after exogenous surfactant, all strongly supported RDS rather than isolated TTN. LUS is not, however, without limitations. Pneumonia and meconium aspiration also show consolidations, and LUS scores were recently found to be of decreased predictive value for the need of invasive ventilation for infants 34 weeks or older.[22] For this reason, we made our diagnosis keeping in mind the overall clinical picture, i.e., history, radiograph, ultrasound, negative sepsis workup, and response to surfactant.

The role of point-of-care lung ultrasound

In this infant, a bedside LUS was obtained by the treating neonatologist within 30 min of admission to the NICU, and the decision to give surfactant was made. A chest radiograph was obtained only subsequently just before giving surfactant and showed a white-out appearance consistent with the LUS findings but did not change the management plan.

There are a number of reasons for using LUS as the preferred test in this instance. It was immediately available, not requiring an infant on 90% oxygen to be moved during analysis or waiting for a portable radiograph, and it involves no ionizing radiation.[20] It showed not only that aeration was severely reduced but also where; and posterior extension of the scan revealed subpleural consolidation not appreciated by anterior six-zone scanning. And it could be repeated at the bedside to follow recovery, as it was at 24 hours post-surfactant (Figure 3).[18]

The 2025 European guidelines stated that LUS is a reliable technique to diagnose RDS within 2 hours of age with appropriate training and to include LUS findings to indicate surfactant administration in treatment criteria.[1] In a quality-improvement study, adding LUS criteria to oxygen thresholds increased the proportion of infants receiving surfactant within 3 hours of birth.[27] In a small randomized controlled trial, LUS-guided treatment brought surfactant delivery from a median of 6 hours to 1 hour of life, with better oxygenation afterwards.[28] These studies were performed in preterm infants, but the same rationale also applied to our patient because the LUS picture preceded the radiographic image, and the decision to administer surfactant was based on LUS. In published series of RDS in full-term and near-term infants, the diagnosis rested on chest radiography rather than LUS.[23,24,29] However, LUS requires training and must be interpreted alongside the clinical picture.

Why at 37 weeks?

It was likely multifactorial. In a case-control study of 205 term infants with RDS, caesarean section, gestation of 38 weeks or less, and male sex were independent risk factors; our patient had all three, although that study also included infants with surfactant deficiency secondary to asphyxia or infection.[29] Lung maturity is variable, even in infants of the same gestation, with 37 weeks being at the lower end of term.[7] In the absence of labor, lung fluid clearance was likely impaired and term infants with TTN have been shown to have low lamellar body counts, suggesting that surfactant may already be marginal at this gestation.[12,14] The mother did not receive antenatal steroids. The ASTECS trial had shown that betamethasone given in advance of a planned caesarean at term reduced the admissions for respiratory distress.[13] The study included planned rather than emergency delivery. However, in our case it was an emergency caesarean at term, so there was no time for steroid administration. Maternal diabetes delays surfactant maturation; however, the mother gave no history of it, and her OGTT was normal.[30]

Surfactant beyond the preterm period

The majority of evidence for surfactant comes from preterm infants. The 2025 European consensus guidelines recommend surfactant for preterm infants with worsening RDS, and that treatment should be considered on non-invasive support with a mean airway pressure of ≥6 cm H_2O, an FiO_2 of ≥0.30, or where LUS suggests the need for surfactant (Grade A1).[1] However, they state that they currently do not have enough evidence to make any recommendations for the more mature infant, and that the recently completed SurfON trial should help decide whether to treat late-preterm and early-term infants.[1] The

American Academy of Pediatrics states that surfactant may be considered for term infants with respiratory failure due to secondary surfactant deficiency.[31] A systematic review of late-preterm and term infants with RDS showed that surfactant may reduce mortality (moderate certainty of evidence), air leak, PPHN, and duration of respiratory support (low to very low certainty of evidence) but not the need for invasive ventilation.[32] We did not feel it reasonable to wait with our patient on NIPPV with an FiO_2 of 0.90 (well above any treatment threshold).

Early extubation failure after surfactant

INSURE was planned to give surfactant and return the baby quickly to nasal CPAP.[33] In this baby, oxygenation improved within minutes, but he could not tolerate being off the ventilator at 20 minutes. A lower FiO_2 shows that lung recruitment has improved. It, however, does not show that the infant can breathe on his own against lungs that are still stiff and wet. Persisting lung fluid and the mean airway pressure he still needed may both have played a part. After six hours of ventilation, the baby was extubated without difficulty. Even in preterm infants, in whom it has been studied most, no single measure reliably predicts extubation readiness, and composite assessments perform best.[34] In a more mature infant with severe disease, readiness is likewise better judged by spontaneous respiratory effort, pressure requirement and blood gas together than by FiO_2 alone.

Other diagnoses considered

Congenital pneumonia was one of the alternative diagnoses. A negative sepsis screen and a sterile blood culture taken before antibiotics make it less likely, although they do not rule it out completely. Genetic surfactant disorders (*SFTPB*, *ABCA3* and *SFTPC*) typically appear as severe RDS in term or near-term infants.[35] However, in these conditions the response to exogenous surfactant is usually very transient, and the disease progresses despite surfactant administration. However, the response of this infant to a single dose, with discontinuation of all support by day 4 of life, makes a severe genetic surfactant disorder unlikely, although it does not exclude it. No genetic workup was performed.

Limitations

We did not carry out genetic or other tests of surfactant function and therefore inferred primary surfactant deficiency based on clinical, radiological, and sonographic findings and treatment response rather than measuring it directly.

CONCLUSION

An early-term infant born by LSCS prior to onset of labor can develop severe, surfactant-responsive RDS. In such a baby, with a high oxygen requirement, a white-out radiograph and an LUS pattern of subpleural consolidation, TTN should not be assumed, and surfactant should be considered early. In this case, point-of-care LUS, performed within 30 minutes, strongly favored the diagnosis and guided the decision to give surfactant, with subsequent chest radiography confirming it. A good oxygenation response to surfactant is reassuring, but the decision to extubate should be based on the overall clinical picture.

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