

Anesthetic Management of a Neonate with Right-Sided Congenital Diaphragmatic Hernia and Significant Metabolic Acidosis: A Rare Neonatal Emergency

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ARTICLE INFO

Article history:

Received 25 February 2026

Revised 17 March 2026

Accepted 02 April 2026

Keywords:

Congenital diaphragmatic hernia;

Right-sided CDH;

Neonatal anesthesia;

Metabolic acidosis;

Pulmonary hypertension

Perioperative management

ABSTRACT

Congenital diaphragmatic hernia (CDH) is a neonatal surgical emergency associated with pulmonary hypoplasia, pulmonary hypertension, and significant perioperative anesthetic risk. Right-sided CDH is relatively uncommon and may present additional challenges due to liver herniation and cardiorespiratory compromise. We report the anesthetic management of a 3-day-old, 2.7 kg male neonate with antenatally diagnosed right-sided CDH who presented with respiratory distress and significant metabolic acidosis.

Preoperative workup revealed severe metabolic acidosis on arterial blood gas analysis, necessitating ventilatory support and NICU stabilization before definitive surgery. Once adequately optimized, the neonate underwent diaphragmatic repair via an abdominal approach under general anesthesia. Intraoperative priorities included lung-protective ventilation, prevention of barotrauma, maintaining satisfactory oxygenation and perfusion, and vigilant hemodynamic monitoring with continuous pre- and post-ductal saturation assessment. The herniated bowel and liver were reduced intraoperatively, and the diaphragmatic defect was closed. Throughout the perioperative course, hemodynamic stability was maintained with careful ventilatory and inotropic support.

Following surgery, the neonate was kept intubated and electively ventilated in the NICU, with uneventful extubation on postoperative day 5. This case underscores the value of thorough preoperative stabilization, gentle ventilation, careful monitoring, and a coordinated multidisciplinary approach in achieving favourable perioperative outcomes in neonates with right-sided CDH and metabolic acidosis.

Introduction

Congenital diaphragmatic hernia (CDH) occurs in approximately 1 in 2,500-3,000 live births. Right-sided defects account for only 10–15% of cases yet carry a higher perioperative risk due to frequent liver herniation and delayed diagnosis [1].

Managing anesthesia for right-sided congenital diaphragmatic hernia (RCDH) poses considerable challenges owing to pulmonary hypoplasia, V/Q mismatch, and the ever-present threat of pulmonary hypertension precipitating acute hemodynamic instability. We describe the perioperative anesthetic course of a neonate with RCDH who also had significant metabolic acidosis, with emphasis on the key intraoperative and perioperative considerations.

The authors declare no conflicts of interest.

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DOI:

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Case Report

A 3-day-old male neonate weighing 2.7 kg with an antenatal diagnosis of CDH was born via lower-segment cesarean section. He developed respiratory distress shortly after delivery and was electively intubated with a 3.5-mm endotracheal tube; bag-mask ventilation was deliberately avoided to prevent gastric insufflation. Passage of an orogastric tube was unsuccessful at the initial attempt.

He was transferred to the neonatal intensive care unit (NICU) and placed on synchronized intermittent mandatory ventilation. Ongoing sedation and analgesia were maintained via continuous infusions of midazolam (0.05-0.1 mg/kg/h) and fentanyl (1-2 µg/kg/h).

Initial arterial blood gas analysis revealed significant metabolic acidosis (pH 7.29, HCO₃⁻ 10.6 mmol/L) with hypoxemia, attributed to ventilation-perfusion mismatch and compromised pulmonary perfusion secondary to pulmonary hypoplasia and mediastinal shift. Ultrasonography of the thorax demonstrated gaseous bowel loops with herniation of a portion of the liver into the right hemithorax, causing compression of the right lung. Chest radiography corroborated these findings (Figure 1).

Transthoracic echocardiography revealed dextroposition of the heart; a small patent ductus arteriosus and patent foramen ovale with a left-to-right shunt; an intact interventricular septum; normal pulmonary artery pressures; and preserved biventricular function.



Figure 1- Preoperative chest radiograph showing right-sided congenital diaphragmatic hernia with bowel loops in the right hemithorax and mediastinal shift.

Discussion

Surgery was deferred until clinical stabilization was achieved, in keeping with current evidence-based guidelines that recommend delaying repair until physiological parameters are optimized [2].

Ventilation and perfusion were optimised, and metabolic acidosis was corrected judiciously with sodium bicarbonate (1-2 mEq/kg, given as a slow infusion) only after confirming adequate ventilation and hemodynamic stability — given that indiscriminate bicarbonate administration risks worsening intracellular acidosis [3]. Repeat arterial blood gas analysis confirmed acid-base improvement (pH 7.42), and all routine investigations were within normal limits.

Induction was accomplished with fentanyl (2 µg/kg) and propofol (1-2 mg/kg), titrated slowly to clinical effect, and neuromuscular blockade was established with atracurium (0.5 mg/kg). A Jackson-Rees circuit was used for ventilation with a lung-protective approach: tidal volumes were kept low (4-6 mL/kg) while respiratory rate was increased to sustain adequate minute ventilation and limit both volutrauma and barotrauma.

Peak inspiratory pressure was maintained at ≤ 25 cmH₂O, with permissive hypercapnia (PaCO₂ 45-55 mmHg), in line with the gentle ventilation philosophy for CDH [4]. Sevoflurane (0.8-1 MAC) in an oxygen-air blend was used for maintenance; nitrous oxide was withheld. Dobutamine (5-10 µg/kg/min) was started and uptitrated as needed to sustain mean arterial pressure and protect end-organ perfusion.

Beyond routine monitoring, continuous pre-ductal (right upper limb) and post-ductal (lower limb) oxygen saturation readings, end-tidal CO₂, core body temperature, urine output, and serial arterial blood gas values were tracked throughout.

The gradient between pre- and post-ductal saturations served as a real-time surrogate for ductal shunting and nascent pulmonary hypertension, allowing early detection of hemodynamic deterioration and timely titration of ventilatory and inotropic support [5].

The repair was carried out through an abdominal incision. Herniated bowel and the partially herniated liver were methodically returned to the abdominal cavity; a gradual reduction was deliberate, as a sudden shift risks a sharp rise in intra-abdominal pressure and hemodynamic instability — a well-documented hazard during liver reduction in RCDH [6]. The diaphragmatic defect was then closed (Figure 2). The procedure lasted approximately three hours. Oxygenation and hemodynamics held steady throughout, with no desaturation events or significant hypotension recorded.

Maintenance fluids (1% dextrose Ringer's lactate) ran at 4-5 mL/kg/h. Intraoperative blood loss was negligible (around 15 mL) and urinary output was satisfactory from start to finish.



Figure 2- Intraoperative image during abdominal repair of a right-sided congenital diaphragmatic hernia after reduction of herniated contents and diaphragmatic closure.

Postoperatively, the infant was returned to the NICU on mechanical ventilatory support. Sequential arterial blood gas measurements showed stable acid-base indices and progressive improvement in oxygenation. He was extubated without complication on day 5.

RCDH poses distinct anesthetic challenges: liver herniation, distorted cardiopulmonary anatomy, and a heightened propensity for perioperative instability all compound the complexity of management. Our experience reinforces that rigorous preoperative optimisation, a lung-protective ventilatory strategy with strict pressure limits, vigilant pre- and post-ductal saturation surveillance, and seamless multidisciplinary collaboration are collectively indispensable for safe outcomes in this population.

Conclusion

1. Right-sided congenital diaphragmatic hernia is rare but carries increased anesthetic risk due to liver herniation and delayed diagnosis.
2. A lung-protective ventilatory approach — characterised by low tidal volumes, higher respiratory rates, strict airway pressure ceilings, and continuous pre- and post-ductal saturation monitoring — is fundamental to safe perioperative care in neonatal CDH.

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