e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

Hemodynamic-Focused Anesthetic Strategy for Duodenal Atresia with Annular Pancreas in a Low-Birth-Weight Neonate: A Case Report and Pathophysiological Review

Anggia Rarasati Wardhana[1*], Ardana Tri Arianto[1], Heri Dwi Purnomo[1]

[1]Department of Anesthesiology and Intensive Therapy, Dr. Moewardi Regional General Hospital, Surakarta, Indonesia

ARTICLE INFO

Keywords:
Duodenal atresia
Hemodynamic stability
Ketamine
Low birth weight
Neonatal anesthesia

***Corresponding author:**
Anggia Rarasati Wardhana

E-mail address:
anggiararasatiw@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v6i2.802

A B S T R A C T

Introduction: The anesthetic management of low-birth-weight (LBW) neonates with complex congenital anomalies like duodenal atresia presents a profound physiological challenge. These patients exhibit immature organ systems, precarious fluid balance, and heightened sensitivity to anesthetic agents. This case report describes a successful hemodynamically-focused anesthetic strategy in a particularly high-risk neonate with the combined pathology of duodenal atresia and a constricting annular pancreas. **Case presentation:** A 4-day-old, 1800-gram male infant, born at 37 weeks with intrauterine growth restriction, presented with prenatally diagnosed duodenal atresia. Preoperative stabilization focused on correcting a severe hypochloremic, hypokalemic metabolic alkalosis. A hemodynamically stable anesthetic induction was achieved using intravenous fentanyl (2.8 mcg/kg) and ketamine (2.8 mg/kg), avoiding myocardial depressant volatile agents. Anesthesia was maintained with 60% oxygen in air and intermittent opioid boluses. Intraoperative management was centered on meticulous, goal-directed fluid therapy, rigorous maintenance of normothermia, and lung-protective ventilation. The surgery, a duodenojejunostomy, was completed successfully with remarkable hemodynamic stability. The infant was transferred to the NICU for planned postoperative ventilation and was extubated on the second postoperative day. Postoperative analgesia was achieved with a continuous sub-anesthetic ketamine infusion, later transitioned to intermittent metamizole. **Conclusion:** The successful outcome in this fragile neonate underscores the value of a tailored anesthetic approach grounded in neonatal pathophysiology. A strategy utilizing hemodynamically stable induction agents, proactive correction of metabolic derangements, goal-directed fluid therapy, and a planned, staged recovery can effectively mitigate the significant perioperative risks associated with major abdominal surgery in LBW infants with complex congenital anomalies.

1. Introduction

The perioperative management of neonates, particularly those with low birth weight (LBW) and significant congenital anomalies, remains one of the most intricate and high-stakes subspecialties within anesthesiology. The neonatal period is a phase of dramatic physiological transition, characterized by profound adjustments in the cardiovascular, respiratory, renal, and metabolic systems as the infant adapts from fetal to extrauterine life.[1] These systems are functionally immature, rendering the neonate exquisitely vulnerable to the physiological trespass of surgery and the pharmacological effects of anesthetic agents. When prematurity or LBW is superimposed on

a major surgical pathology, the margin for error narrows to a razor's edge.[2]

Duodenal atresia, a congenital obstruction of the duodenum, is a classic neonatal surgical emergency, occurring in approximately 1 in 5,000 to 10,000 live births.[3] It is frequently associated with other significant conditions, including Trisomy 21 and various cardiac defects, or as part of the VACTERL association. The pathognomonic "double bubble" sign on abdominal radiography, caused by trapped air in the stomach and proximal duodenum, confirms the diagnosis. The complete obstruction results in an inability to tolerate enteral feeding, persistent vomiting, and a high risk of dehydration and critical electrolyte disturbances.[4] The characteristic biochemical derangement is a hypochloremic, hypokalemic metabolic alkalosis, stemming from the loss of voluminous quantities of gastric acid.[5] This is often preceded by maternal polyhydramnion due to impaired fetal swallowing of amniotic fluid, which can contribute to premature labor.

Anesthetizing an LBW infant (defined as <2500g) for duodenal atresia repair represents a confluence of formidable challenges. The cardiovascular system is characterized by a relatively fixed stroke volume, making cardiac output almost entirely dependent on heart rate; thus, bradycardia can be catastrophic.[6] The neonatal myocardium is less compliant and possesses fewer contractile elements, limiting its ability to respond to inotropic stimulation or increased afterload.[7] Elevated pulmonary vascular resistance (PVR) can persist, predisposing the infant to right-to-left shunting through a patent foramen ovale (PFO) or ductus arteriosus (PDA) in response to stimuli like hypoxia, hypercarbia, or acidosis.[8] Respiratory mechanics are inefficient, hepatic and renal systems are immature, leading to altered drug metabolism, and thermoregulation is a constant struggle due to a large surface area-to-mass ratio.[9]

While the principles of managing duodenal atresia are well-established, this case report is instructive due to the unique convergence of multiple high-risk factors: LBW with intrauterine growth restriction (IUGR), a complex dual pathology of intrinsic duodenal atresia and extrinsic compression from an annular pancreas, and significant preoperative metabolic derangement.[10] The novelty of this report lies in its detailed description and justification of a hemodynamically-focused anesthetic protocol, intentionally avoiding potent myocardial-depressant inhalational agents in favor of a ketamine-based induction and maintenance strategy. This approach was specifically chosen to preserve cardiovascular stability in a patient with minimal physiological reserve. Therefore, the primary aim of this report is to provide a comprehensive, step-by-step account of this pathophysiology-driven anesthetic strategy. By dissecting the clinical decision-making process from preoperative optimization to postoperative recovery, we offer a valuable educational framework for managing similarly complex and fragile neonatal surgical patients.

2. Case Presentation

Written informed consent was obtained from the patient's legal guardians for the publication of this case report and any accompanying images. The case is reported in accordance with the CARE guidelines. A 4-day-old, 1800-gram male infant was scheduled for an exploratory laparotomy and duodenojejunostomy. The infant was an inpatient in the High Care Unit (HCU) Neonatus at Dr. Moewardi Regional General Hospital, having been born at the facility. He was born at 37 weeks of gestation via emergency Caesarean section due to a transverse lie, inpartu labor, and was complicated by severe polyhydramnion and IUGR. Antenatal ultrasound had been suggestive of a proximal bowel obstruction. The mother’s medical history was significant for mild anemia during pregnancy but was otherwise unremarkable.

Upon delivery, the infant's Apgar scores were 7 and 9 at 1 and 5 minutes, respectively. He was immediately transferred to the neonatal unit, where an orogastric tube (OGT) was placed, draining significant volumes of bilious fluid. He was kept nil per os. A workup for VACTERL association was negative. On the day of life (DOL) 4, a preoperative assessment was conducted. The infant was in a warmed incubator, appearing weak with a feeble cry. His vital signs were: heart rate (HR) 181 beats/minute, respiratory rate (RR) 50 breaths/minute, core temperature 36.5°C, and oxygen saturation (SpO_2)

97% on 0.5 L/min of humidified oxygen (Table 1). The epigastrium was distended while the lower abdomen was scaphoid. The infant's consciousness was initially lethargic but responsive, with a primitive Glasgow Coma Scale (pGCS) score of E4V5M6.

Table 1. Summary of patient's clinical course.

Day of Life (DOL) / Postop Day (POD)	Key Clinical Events	Vital Signs (HR/MAP/RR/Temp)	Key Laboratory Values	Management Plan / Interventions
DOL 1	Admission to NICU. Diagnosis of duodenal obstruction confirmed.	160 bpm / 48 mmHg / 55 / 36.8°C	Na 134, K 3.1, Cl 92, HCO3 29	NPO, OGT to low suction. IV access established. Start IV fluids (D5 ½NS + 20mEq/L KCl) for resuscitation. Vitamin K IM.
DOL 2-3	Continued preoperative stabilization. Bilious OGT output.	155 bpm / 50 mmHg / 50 / 36.7°C	(DOL 3) Na 136, K 3.9, Cl 98	Continue IV fluid resuscitation. Monitor electrolytes and acid-base status. Cross-match PRBCs. Surgical consult.
DOL 4 (Day of Surgery)	**Exploratory laparotomy and duodenojejunostomy.**	**(Preop) 181 bpm / - / 50 / 36.5°C**	**Hb 12.4, Hct 35, INR 1.27**	**Anesthetic induction with Fentanyl/Ketamine. Transfer to NICU post-op, intubated and ventilated. Start Ketamine infusion for analgesia.**
POD 1 (DOL 5)	Hemodynamically stable on ventilator. Minimal OGT output.	135 bpm / 54 mmHg / 30 (vent) / 36.9°C	Hct 32, Platelets 410k	Continue mechanical ventilation. Maintain Ketamine infusion. Monitor fluid balance. Broad-spectrum antibiotics.
POD 2 (DOL 6)	Improved respiratory effort. Awake and active. **Successful Extubation.**	140 bpm / 55 mmHg / 35 (spont.) / 36.8°C	ABG: pH 7.38, PCO2 40, PO2 88	Wean ventilator support. Extubated to High-Flow Nasal Cannula (HFNC) at 2 L/min. Discontinue Ketamine infusion. Start IV Metamizole.
POD 3 (DOL 7)	Weaned from HFNC. Stable on standard nasal cannula.	142 bpm / 56 mmHg / 40 / 37.0°C	Na 138, K 4.1, Cl 101	Wean to 0.5 L/min standard NC oxygen. Continue IV fluids and analgesia.
POD 4 (DOL 8)	Tolerating OGT decompression well. Bowel sounds present.	138 bpm / 55 mmHg / 42 / 36.9°C	-	Initiate trophic enteral feeding via OGT (1 mL/hr expressed breast milk).
POD 7 (DOL 11)	Feeds advanced slowly. Tolerating well. Off oxygen.	135 bpm / 58 mmHg / 40 / 37.1°C	-	Gradually increase enteral feeds as tolerated. Wean IV fluids accordingly. Discontinue IV metamizole, switch to oral PRN.
POD 14 (DOL 18)	Approaching full enteral feeds. Gaining weight.	130 bpm / 60 mmHg / 38 / 37.0°C	Hb 11.8, Hct 34	Discontinue IV fluids. OGT removed. Encourage oral feeding attempts.
POD 21 (DOL 25)	**Discharged home.**	Stable	-	Tolerating full oral feeds, gaining weight appropriately. Outpatient surgical follow-up scheduled.

Initial investigations revealed a significant metabolic derangement consistent with the suspected diagnosis. Admission biochemistry showed hypokalemia (Potassium 3.1 mmol/L) and hypochloremia (Chloride 92 mmol/L), with a sodium of 134 mmol/L and an elevated bicarbonate of 29 mmol/L; blood glucose was normal at 85 mg/dL. An arterial blood gas analysis confirmed this picture, with a pH of 7.48 and a bicarbonate of 30 mEq/L, establishing the diagnosis of a partially compensated hypochloremic, hypokalemic metabolic alkalosis. The underlying anatomical cause was confirmed radiologically; a supine abdominal radiograph was pathognomonic, demonstrating the classic "double bubble" sign with no distal bowel gas, confirming complete duodenal obstruction. Further preoperative workup included hematological studies, which were largely unremarkable with a hemoglobin of 12.4 g/dL and a hematocrit of 35%, though the prothrombin time was slightly elevated at 17.3 seconds (INR 1.27). Finally, a screening echocardiogram revealed a structurally normal heart with preserved myocardial function, noting only a small, hemodynamically insignificant patent foramen ovale (PFO) and a patent ductus arteriosus (PDA).

The infant was classified as ASA physical status IIIE. The primary goal was preoperative stabilization. An intravenous infusion of Dextrose 5% in 0.45% Saline (D5 ½NS) with 20 mEq/L of potassium chloride was initiated. The rate was calculated to provide maintenance fluids plus correction for an estimated 10% dehydration. After 12 hours of resuscitation, repeat electrolytes showed significant improvement (Na 136, K 3.9, Cl 98), and the metabolic alkalosis had resolved. The patient was deemed optimized for surgery. A comprehensive timeline of the patient's hospital course is detailed in Table 1 and Figure 1.

Figure 1. Timeline of patient's clinical course.

The infant was transported to a pre-warmed operating room (26°C). Standard ASA monitoring was applied. A forced-air warming blanket was used, and all IV fluids were administered via a fluid warmer. After pre-oxygenation, premedication with glycopyrrolate 9 mcg (5 mcg/kg) was administered. Anesthesia was induced with intravenous fentanyl 5 mcg (2.8 mcg/kg) and ketamine 5 mg (2.8 mg/kg), administered slowly. Ondansetron 0.2 mg (0.1 mg/kg) and dexamethasone 0.2 mg (0.1 mg/kg) were also given. Neuromuscular blockade was achieved with atracurium 0.9 mg (0.5 mg/kg). Direct laryngoscopy revealed a Grade 1 Cormack-Lehane view, and the trachea was intubated with a 2.5 mm uncuffed endotracheal tube (ETT) to a depth of 9 cm.

Anesthesia was maintained with intermittent boluses of fentanyl (1 mcg) guided by hemodynamic responses. The lungs were ventilated using pressure-controlled ventilation (PCV) with PIP 16 cmH_2O, PEEP 5 cmH_2O, RR 30 breaths/min, and an I:E ratio of 1:2. FiO_2 was titrated between 0.4 and 0.6 to maintain SpO_2 >95%. End-tidal CO_2 ($EtCO_2$) was maintained between 35-45 mmHg. Normothermia (36.5°C to 37.0°C) was diligently maintained. Estimated Blood Volume (EBV) was 180 mL. Allowable Blood Loss (ABL) was calculated to be ~26 mL. Fluid management was guided by replacing maintenance needs (7.2 mL/hr), ongoing deficit, and third-space losses (estimated at 6-8 mL/kg/hr). Hemodynamics remained exceptionally stable throughout the procedure. A detailed summary of the intraoperative course is provided in Table 2.

The patient remained intubated and was transferred to the NICU. A plan for delayed extubation was made due to LBW, major abdominal surgery, and the presence of a PDA. In the NICU, a continuous intravenous infusion of ketamine was initiated for analgesia at 0.2 mg/kg/hour. On POD 2, the infant met extubation criteria and was successfully extubated to a humidified high-flow nasal cannula (HFNC) oxygen. The ketamine infusion was discontinued, and analgesia was transitioned to intermittent intravenous metamizole 30 mg every 8 hours. Trophic enteral feeding was initiated on POD 4 and advanced gradually. The infant was discharged home on DOL 25 in good condition, tolerating full oral feeds and gaining weight.

At a one-month follow-up appointment, the patient's legal guardians expressed profound gratitude for the care provided. They reported that the infant was

thriving at home, feeding well without vomiting, and meeting developmental milestones appropriate for his corrected gestational age. They consented enthusiastically to the sharing of this case for educational purposes, hoping it could help other families in similar situations.

Table 2. Summary of intraoperative anesthetic record.

Time (min)	Event	HR (bpm)	MAP (mmHg)	SpO_2 (%)	$EtCO_2$ (mmHg)	Temp (°C)	Key Interventions & Medications	Fluid Balance (In/Out/EBL)
-15	Arrival in OR & Pre-oxygenation	165	52	98	-	36.6	Monitoring applied. Glycopyrrolate 9 mcg IV.	IV at TKO
-5	Anesthetic Induction	158	50	100	-	36.6	Fentanyl 5 mcg IV, Ketamine 5 mg IV.	–
0	Intubation	145	48	99	38	36.7	Atracurium 0.9 mg IV. Intubated with 2.5 ETT. Start PCV.	–
15	Surgical Incision	150	54	99	40	36.8	-	In: 15 mL
30	Surgical Exploration	148	52	100	41	36.9	Annular pancreas identified.	In: 15 mL
60	Anastomosis Start	145	50	99	39	37.0	Fentanyl 1 mcg IV bolus.	In: 39.6 mL, Out: 4 mL, EBL: 5 mL
90	Anastomosis Ongoing	142	51	100	40	37.0	-	In: 14.4 mL
120	Wound Closure	138	53	100	38	36.9	Fentanyl 1 mcg IV bolus.	In: 28.8 mL, Out: 7 mL, EBL: 10 mL
135	End of Surgery	135	54	99	39	36.8	Dressings applied. Prepare for transport.	In: 14.4 mL, Out: 11 mL, EBL: 10 mL
Total	Procedure Duration: 135 min	**Medication Totals: Fentanyl: 7 mcg, Ketamine: 5 mg, Atracurium: 0.9 mg**					**Input: 97.2 mL D5 ½NS Output: 11 mL Urine EBL: 10 mL**	

3. Discussion

The successful perioperative management of this 1800-gram neonate with a complex duodenal obstruction was not a matter of fortune, but the direct result of a planned and executed strategy, deeply rooted in the fundamental principles of neonatal pathophysiology.[11] This case represents a confluence of formidable challenges: low birth weight, intrauterine growth restriction, significant congenital abdominal pathology, and profound metabolic derangement. Navigating this high-risk landscape required a proactive and integrated approach to every phase of care. This discussion will provide a detailed, narrative exploration of the physiological challenges encountered and the specific rationale behind the anesthetic, fluid, and analgesic strategies employed, framing them within the broader context of contemporary neonatal anesthesiology and critically examining potential alternative management pathways.[12]

The journey began with confronting the immediate, life-threatening consequences of the congenital anomaly itself. Duodenal atresia, an embryological failure of the duodenal lumen to recanalize during the 8th to 10th week of gestation, creates a complete proximal bowel obstruction. In this patient, the intrinsic atresia was compounded by extrinsic compression from an annular pancreas—a distinct embryological error where the ventral pancreatic bud fails to rotate correctly, instead encircling and constricting the second part of the duodenum. The physiological cascade initiated by this complete blockage is both predictable and severe. The infant's persistent vomiting of gastric and bilious secretions leads to the hallmark triad of hypovolemia, hypochloremia, and hypokalemia. The voluminous loss of gastric fluid, rich in hydrochloric acid (HCl), directly depletes the body's stores of hydrogen and chloride ions.[13] This loss initiates a desperate compensatory response, primarily driven by the renin-angiotensin-

aldosterone system (RAAS), which is activated by the profound hypovolemia. Aldosterone, the final effector of this system, acts on the distal renal tubules to aggressively reabsorb sodium and water to preserve intravascular volume.[14] This volume preservation, however, comes at a steep metabolic cost: the reabsorption of sodium necessitates the excretion of potassium and hydrogen ions into the urine. This renal loss further exacerbates the pre-existing hypokalemia and perpetuates the metabolic alkalosis. In severe cases, this can lead to a state of paradoxical aciduria, where the kidneys excrete acidic urine despite systemic alkalosis, a sign of severe volume depletion where volume preservation overrides acid-base correction.[15] The admission arterial blood gas of our patient, with a pH of 7.48 and a bicarbonate level of 30 mEq/L, was a clear window into this disturbed physiology.

It is an inviolable principle of neonatal surgery that anesthetic induction in a patient with an uncorrected metabolic derangement is a perilous undertaking. The systemic alkalosis shifts the oxyhemoglobin dissociation curve to the left, increasing hemoglobin's affinity for oxygen and thereby impairing its release to the tissues. It can also cause compensatory hypoventilation and potentiates the negative inotropic and arrhythmogenic effects of many anesthetic agents.[15] Therefore, the initial 12 hours of aggressive resuscitation with a carefully chosen intravenous fluid—Dextrose 5% in 0.45% Saline with 20 mEq/L of potassium chloride—was the most critical intervention. This solution was designed to simultaneously address multiple deficits: the dextrose to prevent hypoglycemia in a neonate with scant glycogen stores; the free water and sodium to begin repleting intravascular volume; the chloride to correct the core deficit of the metabolic alkalosis; and the potassium to correct the severe hypokalemia. Only once homeostasis was restored, as evidenced by the normalization of electrolytes, was it safe to proceed to the operating room.

The core of the intraoperative plan was a hemodynamically-focused anesthetic strategy, a decision directly informed by the unique and fragile cardiovascular physiology of the LBW neonate.[16] The neonatal myocardium is structurally and functionally immature. It contains fewer organized contractile myofibrils and more non-contractile tissue compared to the adult heart, resulting in significantly reduced compliance. This inherent stiffness means the neonatal heart has a very limited ability to increase its stroke volume in response to increased preload, rendering the Frank-Starling mechanism underdeveloped and inefficient. Consequently, cardiac output is almost entirely dependent on heart rate (CO=HR×SV). Any clinical event that precipitates bradycardia—be it vagal stimulation during laryngoscopy, hypoxia, acidosis, or the direct effect of anesthetic drugs—can lead to a catastrophic fall in cardiac output and systemic perfusion. Furthermore, the autonomic innervation of the heart is also immature, with a dominant parasympathetic (vagal) tone and an incompletely developed sympathetic system, making the neonate even more prone to bradycardia.[17] This physiological reality was the primary driver for the prophylactic administration of glycopyrrolate and for the selection of anesthetic agents that would support, rather than depress, the heart rate.

Compounding this myocardial immaturity is the persistence of a transitional circulation. At birth, pulmonary vascular resistance (PVR) is high. While it falls dramatically in the first few days of life, it remains labile and exquisitely sensitive to a variety of stimuli, including pain, surgical stress, hypoxia, hypercarbia, and acidosis. Our patient had both a patent foramen ovale (PFO) and a patent ductus arteriosus (PDA), creating potential shunts between the pulmonary and systemic circulations. An acute rise in PVR can overwhelm the right ventricle and reverse the direction of blood flow through these shunts, causing deoxygenated blood to bypass the lungs and enter the systemic circulation. This right-to-left shunting initiates a dangerous, self-perpetuating cycle of hypoxia, worsening acidosis, and escalating pulmonary hypertension. A central goal of our anesthetic plan, therefore, was to avoid every known trigger for pulmonary vasoconstriction.

Given this fragile cardiovascular substrate, the decision to avoid potent inhalational agents, such as sevoflurane, for induction was deliberate. Although they provide rapid and smooth induction, volatile anesthetics are profound myocardial depressants and

vasodilators. Their dose-dependent depression of myocardial contractility, combined with a reduction in systemic vascular resistance (SVR), can precipitate severe hypotension in a preload-dependent neonate who cannot compensate by increasing stroke volume. This can lead to a rapid downward hemodynamic spiral. Propofol was similarly avoided due to its well-documented association with significant hypotension and apnea in this population. Instead, we chose a combination of ketamine and fentanyl, a technique that synergistically provides potent analgesia and amnesia while preserving cardiovascular stability. Ketamine, through its unique mechanism as an NMDA receptor antagonist, produces indirect sympathomimetic effects by inhibiting norepinephrine reuptake. This effect typically supports or even increases heart rate and blood pressure, a highly desirable quality in this context. It also offers the ancillary benefits of bronchodilation and profound analgesia. Fentanyl, a potent μ-receptor opioid agonist, was co-administered to potently blunt the profound sympathoadrenal stress response to laryngoscopy and surgical incision. This hormonal stress response, involving the release of catecholamines and cortisol, is particularly exaggerated in neonates and is a potent trigger for tachycardia, hypertension, and increased PVR. The combination of ketamine's supportive cardiovascular properties with fentanyl's ability to ablate the stress response provided an ideal and stable induction pathway for this high-risk infant.[18]

The management of the patient's inefficient respiratory system required an equally deliberate, lung-protective approach (Figure 2). The neonatal respiratory system is disadvantaged by a highly compliant cartilaginous chest wall and relatively non-compliant lungs.[19] This mismatch leads to a tendency for the chest to collapse inwards during inspiration, resulting in a low functional residual capacity (FRC). The FRC acts as the lung's oxygen reserve, and its small volume in neonates means that desaturation occurs with alarming rapidity during periods of apnea. Our ventilation strategy utilized pressure-controlled ventilation (PCV), a mode often preferred in neonates because it delivers a set inspiratory pressure, minimizing the risk of barotrauma to the delicate lung tissue. Importantly, PCV compensates for the variable air leak that is expected around an uncuffed endotracheal tube, which is the standard of care to prevent iatrogenic subglottic stenosis. The application of positive end-expiratory pressure (PEEP) at 5 cmH_2O was a critical component of this strategy. PEEP effectively stents the small airways and alveoli open at the end of expiration, preventing their collapse (atelectasis), thereby recruiting lung volume, preserving the precious FRC, improving ventilation-perfusion matching, and optimizing oxygenation.

Intraoperative homeostasis was a triumvirate of meticulous fluid management, glucose control, and thermoregulation. Neonatal fluid management is a delicate balancing act, as the immature neonatal kidney possesses a low glomerular filtration rate (GFR) and a limited ability to concentrate urine or excrete a large fluid or sodium load.[20] Both dehydration and fluid overload are poorly tolerated and are associated with significant morbidity, including necrotizing enterocolitis and intraventricular hemorrhage. Our fluid plan was therefore multi-faceted, calculated to replace maintenance needs, ongoing deficits from preoperative losses, and the substantial but unmeasurable third-space losses resulting from the surgical trauma of laparotomy. The intentionally positive fluid balance achieved was necessary to maintain adequate circulating volume and ensure end-organ perfusion in the face of this significant fluid translocation. Simultaneously, maintaining normothermia was a constant battle. Neonates are essentially poikilothermic, and the cold operating room environment is a hostile one. They lose heat rapidly through four mechanisms: radiation to cold surfaces, convection from air currents, conduction to the operating table, and evaporation from the respiratory tract and the open surgical field. Hypothermia is a major catalyst for perioperative morbidity, leading to metabolic acidosis, coagulopathy, delayed drug metabolism, and impaired immune function. Our multi-modal approach—a high ambient OR temperature, a forced-air warmer, and a fluid warmer—was the absolute standard of care and was vital to preserving core body temperature.

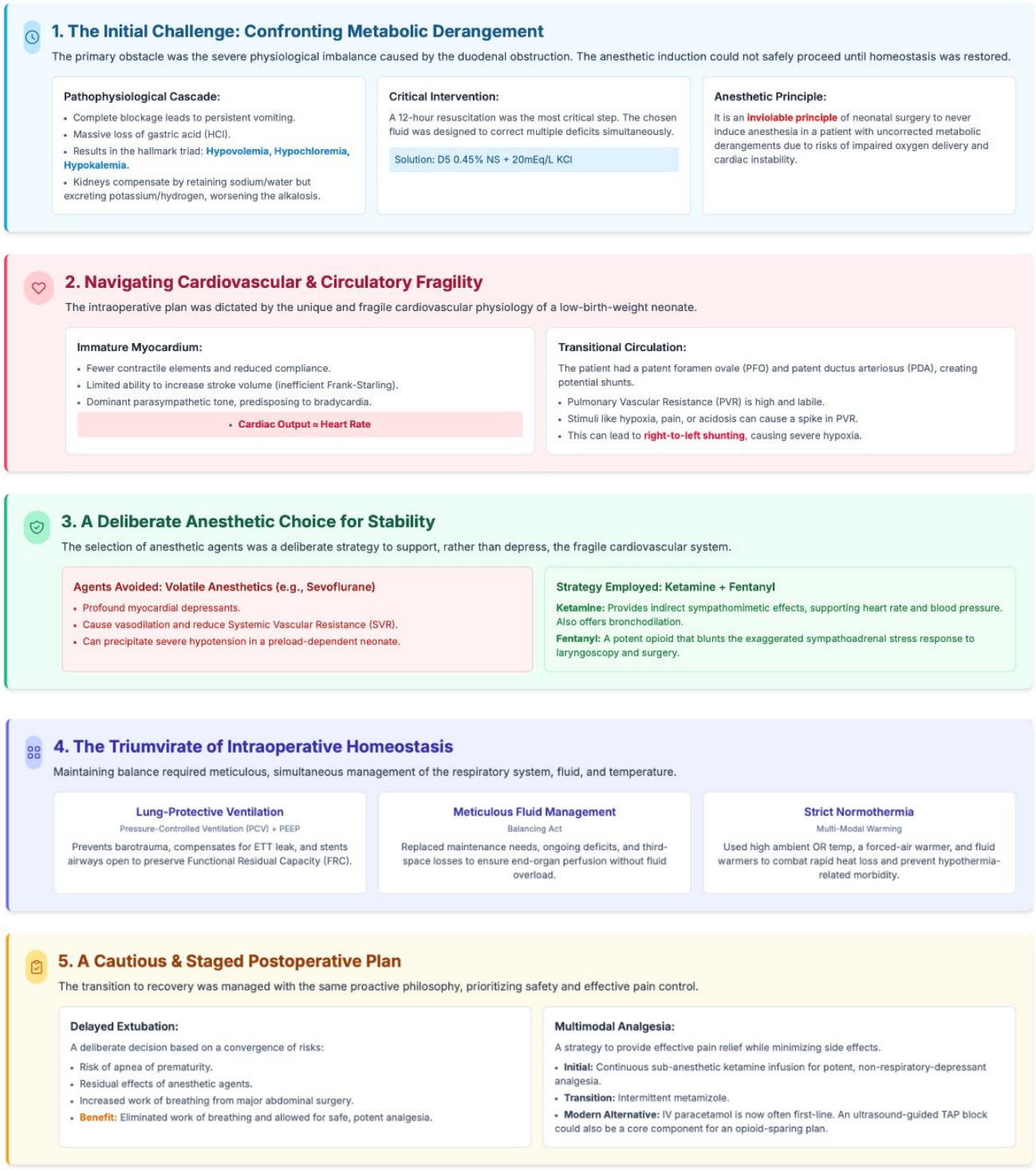

Figure 2. Analysis of a hemodynamically-focused anesthetic approach in a low-birth-weight neonate.

The transition to the postoperative period was managed with the same cautious, proactive philosophy. The decision for delayed extubation was based on a convergence of risk factors. Apnea of prematurity is common in infants born before 37 weeks, and residual anesthetic agents can exacerbate this by depressing the immature respiratory drive.[21] Major abdominal surgery further compromises respiratory function through diaphragmatic splinting due to pain and abdominal distension, increasing the work of breathing to a level that can overwhelm a neonate's easily fatigable respiratory muscles. Allowing the infant to recover on

the ventilator eliminated the work of breathing, permitted the safe use of potent analgesics, and mitigated the physiological stress of the immediate postoperative period.

Effective postoperative pain control was achieved with a multimodal strategy. The continuous sub-anesthetic ketamine infusion provided a baseline of potent, non-respiratory-depressant analgesia. The transition to metamizole, while effective, warrants careful consideration. While it is a potent non-opioid analgesic used in many regions, its association with the rare but fatal risk of agranulocytosis has led to its restriction in many countries. Today, intravenous paracetamol (acetaminophen) is widely considered the first-line non-opioid analgesic in the neonatal population due to its well-established safety profile. A notable alternative that was deferred in this case is regional anesthesia. An ultrasound-guided transversus abdominis plane (TAP) block could have provided excellent somatic analgesia, significantly reduced systemic opioid requirements, and possibly facilitated earlier extubation. The technique was deferred here due to the patient's borderline coagulopathy (INR 1.27) and a desire to avoid any potential hemodynamic confounding variables. However, in a stable neonate with normal coagulation, a truncal block should be considered a core component of a modern, opioid-sparing analgesic plan.

This report, of course, is subject to the inherent limitations of a single-case study. The successful outcome describes the management of one patient and does not establish causality or prove the superiority of this specific anesthetic regimen over others. The positive result is likely attributable to a combination of factors, including meticulous surgical technique, high-quality neonatal intensive care, and the patient's specific physiology, in addition to the anesthetic management. The findings, therefore, are not directly generalizable but serve to illustrate the successful application of a physiologically-sound, hemodynamically-focused approach in a highly challenging clinical scenario.

4. Conclusion

This case report details the successful perioperative management of an 1800-gram, low-birth-weight neonate with the complex dual pathology of duodenal atresia and annular pancreas. The favorable outcome was the result of a meticulously planned and executed strategy deeply rooted in an understanding of neonatal pathophysiology. Key elements contributing to this success included: (1) aggressive preoperative stabilization of fluid and electrolyte status; (2) a hemodynamically-focused anesthetic induction using a balanced ketamine-fentanyl technique that avoided myocardial depressants; (3) vigilant and proactive intraoperative management focused on goal-directed fluid therapy, strict normothermia, and lung-protective ventilation; and (4) a cautious, staged postoperative plan incorporating continued mechanical ventilation and multimodal analgesia. This case reinforces the principle that success in high-risk neonatal anesthesia is achieved through a synthesis of detailed physiological knowledge, pharmacological precision, and proactive management of anticipated challenges, serving as a comprehensive model for navigating the complexities of caring for our smallest and most vulnerable surgical patients.

5. References

1. Liu T, Wang Y, Xiao X, Chen Z, Li X, Liu C. Comparison of maternal and neonatal outcomes between general anesthesia and combined spinal-epidural anesthesia in cesarean delivery for pregnancy complicated with placenta previa. BMC Anesthesiol. 2025; 25(1): 294.
2. Pezzato S, Govindan RB, Beqiri E, Smielewski P, Waberski A, Nuszkowski M, et al. Intraoperative assessment of cerebral autoregulation with cerebral oximetry index in neonates undergoing cardiac surgery: a pilot study. J Cardiothorac Vasc Anesth. 2025; 39(8): 2049–56.
3. Prigge L, Bosenberg AT, van Schoor AN. Anatomical study of the superficial cervical plexus targeted for sensory nerve blocks in neonates. Reg Anesth Pain Med. 2025; rapm-

2025-106997.
4. Heinonen K, Saisto T, Gissler M, Sarvilinna N. Maternal and neonatal complications of shoulder dystocia with a focus on obstetric maneuvers: a case-control study of 1103 deliveries. Obstet Anesth Dig. 2025; 45(3): 163–163.
5. Badge M, Hasija S, Chauhan S, Gayatri S, Bisoi AK, Kumar A. Evaluation of intraoperative bivalirudin anticoagulation in neonates and infants undergoing arterial switch operation on integrated ECMO-CPB circuit: a prospective study. J Cardiothorac Vasc Anesth. 2025; 39(9): 2339–45.
6. Butt AL, Mazzeffi MA, Mishima Y, Tanaka KA. Fibrinogen replacement in neonatal cardiac surgery: Methodological challenges. Anesth Analg. 2025; 141(3): e37–8.
7. Diego MG. Manolo RL. Anesthetic management of esophageal atresia tipe iii with tracheoesophageal fistula in premature infant without invasive monitoring: a case report. J Anesth Crit Care. 2015; 2(4).
8. Shinozaki H, Nakao M, Sakurai Y, Unetani H, Kurokawa H, Sato N. Anesthetic management of congenital esophageal atresia with tracheomalacia using laryngeal mask airway. J Jpn Soc Clin Anesth. 2013; 33(2): 247–52.
9. Passi Y, Sampathi V, Pierre J, Caty M, Lerman J. Esophageal atresia with double tracheoesophageal fistula. Anesthesiology. 2013; 118(5): 1207.
10. Kajikawa Y, Taguchi S, Kato T, Oshita K, Hamada H, Tsutsumi Y. Ultrasound-guided thoracic paravertebral block after surgery for biliary atresia in a neonate. J Jpn Soc Clin Anesth. 2021; 41(1): 47–53.
11. Balasubramanian S, Ramasundaram M, Sundaram J. Congenital duodenal obstruction due to duodenal atresia along with annular pancreas, intestinal malrotation and preduodenal portal vein. BMJ Case Rep. 2025; 18(2): e264877.
12. Corujo Avila P, Woodward JM, L'Huillier JC, Mucci-Jackson B, Amin R, Wulkan ML, et al. Technique modifications: enabling laparoscopic repair of duodenal atresia in a preterm, very low birthweight infant. J Surg Case Rep. 2025; 2025(5): rjaf323.
13. Atiyat DK, Al-Nusair DA, Alhajahjeh A, Al-Awadi MY, Aborajooh E. Global prevalence of duodenal atresia in trisomy 21: a systematic review and meta-analysis. Eur J Pediatr Surg. 2025; 35(3): 208–18.
14. Tian C, Liu J, Yang Z, Sun X, Li B, Dai K. Type IIIa esophageal atresia, duodenal atresia, and intestinal atresia in one of the monochorionic, diamniotic twins: a multi-surgery approach for treatment. J Clin Med Res. 2025; 6(2): 228.
15. Davis RT, Baida IB, Ward KR, Brahmamdam P, Akay B, Stallion A, et al. Laparoscopic stapled anastomosis for duodenal atresia: Superior efficiency compared to hand-sewn techniques. J Pediatr Surg. 2025; (162550): 162550.
16. Chinthala S, Rathi C, Pati AB, Mahalik SK. Congenital duodenal band masquerading as duodenal atresia in a neonate: a diagnostic challenge. BMJ Case Rep. 2025; 18(8): e266928.
17. Li M, Xie G, Chu L, Li Y. Efficacy of low-dose hypobaric anesthetics in spinal anesthesia for cesarean delivery: systematic review and meta-analysis. J Matern Fetal Neonatal Med. 2025; 38(1): 2519655.
18. Kumar Gupta S, Krishnatray D, Kanani R. Anesthesia management in patients with acute Respiratory Distress Syndrome (ARDS). J Neonatal Surg. 2025; 14(8S): 498–502.
19. Smolkina EO, Lekmanov AU. Neonatal sepsis and septic shock: towards the implementation of phenotyping into clinical practice. Russian Journal of Pediatric Surgery, Anesthesia and Intensive Care. 2025; 15(1): 51–70.
20. Jackson KL, Smiley RM, Lee AJ. Neonatal acid-base status before and after discontinuing routine left uterine displacement for elective cesarean delivery: a retrospective cohort study (2014-2017). Int J Obstet Anesth. 2025; 62(104350): 104350.

21. Khan MJ, Hassan J, Karmakar A, Khan M, Dean CT, Scavone BM, et al. Closed-loop vasopressor systems for hemodynamic stability during cesarean delivery and maternal and neonatal outcomes: a systematic review and meta-analysis. Int J Obstet Anesth. 2025; (104768): 104768.