

CASE REPORT

Ketamine–Fentanyl Anesthesia in a Low-Birth-Weight Neonate with Duodenal Atresia and Annular Pancreas: An Observational Analytic Study

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ABSTRACT

Background. Low-birth-weight neonates with duodenal atresia and annular pancreas may have perioperative cardiovascular and metabolic instability.

Objective. To describe the perioperative course of a neonate managed with a volatile-sparing ketamine–fentanyl anesthetic regimen.

Methods. An observational case study followed a 4-day-old male infant weighing 1,800 g, born at 37 weeks with intrauterine growth restriction. A 12-hour fluid resuscitation period preceded laparotomy. Induction used intravenous ketamine (2.8 mg/kg) and fentanyl (2.8 mcg/kg), followed by pressure-controlled ventilation, fluid replacement and postoperative intensive care.

Results. After resuscitation, potassium increased from 3.1 to 3.9 mmol/L and chloride from 92 to 98 mmol/L. During surgery, heart rate was 135–158 beats/min and mean arterial pressure was 48–54 mmHg, without inotropic support. Estimated blood loss was 10 mL, below the reported allowable blood loss of 26 mL. Postoperative ketamine infusion (0.2 mg/kg/h) was discontinued at extubation on postoperative day 2. Enteral feeding began on day 4, and the infant was discharged on day 21.

Conclusion. The reported course illustrates perioperative management with a volatile-sparing regimen following metabolic correction. A single case cannot establish causal benefit or comparative superiority.

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1. Introduction

The perioperative anesthetic management of neonatal patients undergoing emergent or urgent reconstructive surgery represents a complex challenge in clinical medicine.¹ This complexity increases in low-birth-weight (LBW) neonates presenting with major upper gastrointestinal congenital malformations, where structural anatomical obstacles intersect with physiological vulnerability.¹ Duodenal atresia, an embryological failure of luminal recanalization occurring between the eighth and tenth weeks of gestation, manifests with an incidence of approximately 1 in 5,000 to 10,000 live births.^{2,3} When intrinsic duodenal atresia coexists with an extrinsic constricting annular pancreas—an uncommon developmental error resulting from the incomplete rotation of the ventral pancreatic bud—the resulting complete mechanical obstruction generates severe proximal gastrointestinal tract distension, persistent bilious vomiting, and marked fluid and electrolyte disturbances within the initial days of extrauterine life.^{4,5}

The pathophysiological repercussions of complete proximal intestinal obstruction in neonates are characterized by massive losses of gastric secretions rich in hydrochloric acid (HCl), potassium, and water.^{6–8} The loss of hydrogen and chloride ions directly precipitates severe hypochloremic, hypokalemic metabolic alkalosis, a

condition further exacerbated by the secondary neuroendocrine response of the renin-angiotensin-aldosterone system (RAAS).^{9–11} Hypovolemia-induced aldosterone hypersecretion prompts distal renal tubular reabsorption of sodium and water at the mandatory expense of urinary potassium and hydrogen ion excretion, establishing a perpetuating cycle of paradoxical aciduria despite systemic alkalosis.^{12–14} In this metabolically destabilized state, tissue oxygen extraction is profoundly compromised due to a leftward shift of the oxyhemoglobin dissociation curve, while immature cardiac myocytes are rendered particularly susceptible to fatal dysrhythmias and negative inotropic collapse upon exposure to standard anesthetic agents.^{15–17}

Compounding this metabolic fragility is the cardiovascular physiology of the low-birth-weight neonate.¹⁸ The neonatal myocardium is structurally characterized by poorly organized myofibrils, a high proportion of non-contractile connective tissue, and rigid non-compliant ventricles.¹⁹ Consequently, the neonatal heart operates near the peak of its Frank-Starling compliance curve, exhibiting an exceptionally limited capacity to augment stroke volume in response to increased preload or afterload.^{20–22} Cardiac output is virtually rate-dependent, rendering even transient bradycardia catastrophic to systemic perfusion pressure.²³ In addition, the immature autonomic nervous system is dominated by parasympathetic vagal

innervation, predisposing fragile neonates to profound bradycardia during noxious airway manipulation or acute volume shifts.²⁴ Furthermore, persistent patent fetal channels, including a patent foramen ovale (PFO) and patent ductus arteriosus (PDA), remain sensitive to changes in pulmonary vascular resistance (PVR).²⁵ Any acute surge in PVR triggered by surgical pain, hypothermia, hypercarbia, or hypovolemia can rapidly precipitate right-to-left extrapulmonary shunting, causing acute hypoxic crises and refractory circulatory failure.^{26,27}

Conventional general anesthetic techniques in neonatal laparotomy frequently rely on halogenated volatile agents, most notably sevoflurane, or intravenous propofol.²⁸ However, volatile anesthetics exert dose-dependent negative inotropic effects on immature myocardium and blunt sympathetic vascular tone, inducing profound systemic vasodilation and arterial hypotension that often necessitate empirical vasopressor rescue.^{29,30} In low-birth-weight neonates with minimal physiological reserve and transitional circulation, such pharmacodynamic depression carries substantial risks of tissue hypoperfusion, cerebral autoregulatory failure, and postoperative morbidity.³¹ Despite the widespread recognition of these vulnerabilities, standardized protocols specifically leveraging volatile-sparing, hemodynamically focused anesthetic regimens in LBW neonates with dual duodenal atresia and annular pancreas remain limited in the literature.³²⁻³⁴ Most published reports remain purely descriptive, lacking quantitative longitudinal tracking of hemodynamic trajectories, serial acid-base resolution kinetics, and objective causality assessments.³⁵⁻³⁷

To address this knowledge gap, this study presents an observational analytic case study of an 1800-gram low-birth-weight neonate with concurrent duodenal atresia and annular pancreas managed via an individualized, pathophysiology-driven anesthetic strategy. The primary scientific objective of this investigation is to mechanistically evaluate the hemodynamic stability, stress-response blunting, and perioperative metabolic trajectory achieved through a volatile-sparing induction and maintenance protocol combining intravenous ketamine (2.8 mg/kg) and fentanyl (2.8 mcg/kg), coupled with goal-directed fluid replacement, lung-protective mechanical ventilation, and staged postoperative intensive recovery. By integrating high-density longitudinal data, formal causality scoring, and global benchmarking, this study elucidates a reproducible anesthetic blueprint for high-risk neonatal abdominal surgery.

2. Methods

2.1. Study Design and Epidemiological Framework

This study was designed as an observational analytic case study conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines and the Consensus-based Clinical Case Reporting Guideline Development (CARE) framework.³⁸ An empirical, longitudinal single-subject observational paradigm was implemented to capture time-resolved physiological and biochemical parameters throughout the perioperative course, extending from initial preoperative resuscitation in the neonatal high care unit to intraoperative surgical reconstruction and subsequent recovery in the neonatal intensive care unit (NICU).

2.2. Ethical Governance and Participant Consent

The clinical observation and scientific analysis protocol received formal institutional review and ethics approval from the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2025/8842), and was registered with the institutional review board of Dr. Moewardi Regional General Hospital, Surakarta, Indonesia. All clinical investigations and diagnostic procedures complied strictly with the ethical tenets of the Declaration of Helsinki (2013 Fortaleza revision) and the International Council for Harmonisation of Technical Requirements for Pharmaceuticals for Human Use (ICH) Good Clinical Practice guidelines. Written informed parental consent was obtained from the infant's legal guardians for all surgical interventions, targeted anesthetic protocols, serial physiological data recording, and the anonymized publication of clinical records and operative findings.

2.3. Participant Eligibility and Diagnostic Ascertainment

Eligibility criteria mandated the confirmed diagnosis of complete congenital duodenal obstruction requiring emergent laparotomy in a low-birth-weight neonate (<2,500 g). The subject of this investigation was a 4-day-old male infant born at 37 weeks of gestation via emergency cesarean section due to transverse lie in active labor, complicated by intrauterine growth restriction (birth weight: 1,800 g) and severe maternal polyhydramnios. Diagnostic ascertainment of complete proximal intestinal obstruction was established preoperatively via clinical manifestations (inability to tolerate enteral intake, continuous bilious orogastric aspirate, epigastric distension with scaphoid lower abdomen) and verified radiologically on supine plain abdominal radiography demonstrating the pathognomonic "double-bubble" sign with complete absence of distal bowel gas. Comprehensive pre-anesthetic screening, including neonatal echocardiography, confirmed structurally normal cardiac anatomy with preserved biventricular systolic performance, accompanied by a small, hemodynamically non-significant patent foramen ovale (PFO) and patent ductus arteriosus (PDA), with no other features of VACTERL association.

2.4. Longitudinal Observation and Anesthetic Management Protocol

Preoperative optimization was performed over a dedicated 12-hour period in the neonatal high care unit, directed toward correcting profound dehydration and hypochloremic, hypokalemic metabolic alkalosis. Intravenous crystalloid resuscitation was delivered using 5% Dextrose in 0.45% Saline (D5 ½NS) supplemented with 20 mEq/L potassium chloride, calculated to deliver maintenance volume plus replacement for an estimated 10% intravascular volume contraction. Serial blood gas and electrolyte evaluations were performed to confirm biochemical restoration prior to transferring the patient to the operating room.

Operating room preparation involved rigorous thermal management: ambient room temperature was elevated to 26.0°C, and active warming was maintained using a forced-air warming system and intravenous fluid warmers. Following pre-oxygenation with 100% oxygen via a neonatal Jackson-Rees circuit, vagolytic premedication with intravenous glycopyrrolate (9 mcg, equivalent to 5 mcg/kg) was administered. Anesthetic induction was performed using a volatile-sparing regimen comprising

intravenous fentanyl (5 mcg, 2.8 mcg/kg) and racemic ketamine (5 mg, 2.8 mg/kg) administered slowly over 60 seconds, accompanied by prophylactic dexamethasone (0.2 mg, 0.1 mg/kg) and ondansetron (0.2 mg, 0.1 mg/kg). Organ-independent neuromuscular blockade was achieved with atracurium besylate (0.9 mg, 0.5 mg/kg). Direct laryngoscopy with a Miller size 0 blade revealed a Cormack-Lehane Grade 1 view, and the trachea was secured with a 2.5 mm internal diameter uncuffed endotracheal tube fixed at a depth of 9 cm at the alveolar ridge.

Mechanical ventilation was maintained in pressure-controlled ventilation (PCV) mode: peak inspiratory pressure (PIP) 16 cmH₂O, positive end-expiratory pressure (PEEP) 5 cmH₂O, respiratory rate (RR) 30 breaths/min, inspiratory-to-expiratory (I:E) ratio 1:2, and fraction of inspired oxygen (FiO₂) titrated between 0.40 and 0.60 to maintain transcutaneous arterial oxygen saturation (SpO₂) $\geq$ 95% and end-tidal carbon dioxide (EtCO₂) between 35 and 45 mmHg. Anesthesia was maintained with an air-oxygen gas mixture devoid of halogenated volatile agents, supplemented by weight-adjusted intermittent fentanyl boluses (1 mcg IV) administered in response to anticipated surgical stimulus steps. Estimated blood volume (EBV) was calculated as 100 mL/kg (180 mL total), establishing an allowable blood loss (ABL) threshold of 26 mL based on an initial hematocrit of 35.0% and a target minimum hematocrit of 30.0%. Fluid administration combined basal maintenance (7.2 mL/h) with replacement of ongoing third-space losses (6–8 mL/kg/h) utilizing D5 ½NS. The surgical procedure consisted of exploratory laparotomy, identification of a constricting annular pancreas encircling the descending duodenum, and side-to-side duodenojejunostomy without pancreatic division.

2.5. Comparative Framework and Causality Assessment Instruments

To evaluate whether the observed hemodynamic stability and uneventful recovery were causally attributable to the volatile-sparing ketamine–fentanyl protocol rather than confounding biological factors, formal causality scoring was applied. Evaluative frameworks included the Bradford Hill criteria for epidemiological causation (assessing strength, consistency, temporality, biological gradient, plausibility, and experimental coherence), the World

Health Organization–Uppsala Monitoring Centre (WHO-UMC) standardized causality assessment system, and the Naranjo Adverse Drug Reaction Probability Scale. In addition, the perioperative parameters of the patient were systematically benchmarked against published contemporary multicenter international cohorts and meta-analyses investigating neonatal duodenal obstruction repair.

2.6. Biostatistical Analysis and Trajectory Analytics

Given the single-subject observational analytic design ($n = 1$), inferential null-hypothesis testing (such as paired Student's *t*-tests, ANOVA, or chi-square statistics) was methodologically inappropriate and strictly excluded to avoid fabricating artificial *p*-values. In accordance with high-standard biostatistical guidelines for single-case analytic research, data evaluation relied on high-density longitudinal time-series tracking, calculated percentage delta changes relative to baseline ($\Delta\% = [(T_n - T_0) / T_0] \times 100\%$), trajectory variance modeling across procedural phases, and direct calibration against established pediatric reference intervals. Data were analyzed using Python (version 3.11, SciPy/NumPy libraries) to ensure absolute arithmetic precision across all reported parameters.

3. Results

3.1. Preoperative Metabolic Derangement and Resuscitation Dynamics

Initial diagnostic screening on day of life (DOL) 4 confirmed severe intravascular volume contraction and classic hypochloremic, hypokalemic metabolic alkalosis. Admission arterial blood gas analysis revealed a systemic arterial pH of 7.48, PaCO₂ of 44 mmHg, and serum bicarbonate of 30 mEq/L. Concurrently, serum electrolyte profiling demonstrated profound hypokalemia (potassium: 3.1 mmol/L) and hypochloremia (chloride: 92 mmol/L), alongside borderline hyponatremia (sodium: 134 mmol/L) and euglycemia (blood glucose: 85 mg/dL). Baseline hematological profiling revealed a hemoglobin concentration of 12.4 g/dL, hematocrit of 35.0%, leukocyte count of $11.2 \times 10^3 / \mu\text{L}$, and platelet count of $410 \times 10^3 / \mu\text{L}$. Coagulation screening demonstrated a mildly prolonged prothrombin time of 17.3 seconds, yielding an international normalized ratio (INR) of 1.27, as shown in Table 1.

Table 1. Baseline Clinical, Diagnostic, and Metabolic Biomarkers Across Preoperative Stabilization Milestones

Diagnostic Domain	Clinical Parameter / Biomarker	Baseline Finding (DOL 1–4)	Post-Resuscitation (Pre-Incision)	Reference Interval
Patient Demographics	Chronological Age & Gender	4 days of life (Male)	4 days of life (Day of Surgery)	N/A
Patient Demographics	Gestational Age & Birth Weight	37 weeks, IUGR (1,800 g)	1,800 g (LBW category)	$\geq$ 2,500 g
Patient Demographics	Delivery Route & Indication	Emergency C-section (Transverse lie)	Postoperative day 0	N/A
Patient Demographics	Apgar Score (1 min / 5 min)	7 / 9	Preserved neurological vitality	$\geq$ 7 / $\geq$ 8
Clinical Phenotype	Abdominal Physical Examination	Epigastric distension, scaphoid lower abdomen	Gastric decompression verified	Soft, non-distended
Clinical Phenotype	Preoperative Dehydration Degree	Estimated 10% volume contraction	Euvolemic status achieved	Euvolemic (0%)
Radiological Findings	Supine Abdominal Radiography	Double-bubble sign, absent distal gas	Duodenal atresia confirmed	Normal bowel gas pattern
Echocardiography	Structural & Shunt Anatomy	PFO & PDA (hemodynamically small)	Normal biventricular contractility	Closed fetal shunts
Acid-Base Homeostasis	Arterial Blood Gas pH	7.48 (Systemic metabolic alkalosis)	7.38 (Normalized physiological pH)	7.35–7.45
Acid-Base Homeostasis	Serum Bicarbonate (HCO ₃ ⁻)	30 mEq/L (Compensated alkalosis)	24 mEq/L (Resolved alkalosis)	22–26 mEq/L

Diagnostic Domain	Clinical Parameter / Biomarker	Baseline Finding (DOL 1–4)	Post-Resuscitation (Pre-Incision)	Reference Interval
Acid-Base Homeostasis	Partial Pressure of CO ₂ (PaCO ₂)	44 mmHg (Compensatory hypoventilation)	40 mmHg (Normocapnia)	35–45 mmHg
Serum Electrolytes	Serum Potassium (K ⁺)	3.1 mmol/L (Severe hypokalemia)	3.9 mmol/L (Normalized potassium)	3.5–5.0 mmol/L
Serum Electrolytes	Serum Chloride (Cl ⁻)	92 mmol/L (Severe hypochloremia)	98 mmol/L (Normalized chloride)	96–106 mmol/L
Serum Electrolytes	Serum Sodium (Na ⁺)	134 mmol/L (Borderline hyponatremia)	136 mmol/L (Normonatremia)	135–145 mmol/L
Hematology & Coagulation	Hemoglobin / Hematocrit	12.4 g/dL / 35.0%	12.4 g/dL / 35.0%	13.5–19.5 g/dL / 42–60%
Hematology & Coagulation	Platelet Count	410 × 10 ³ /μL (Preserved count)	410 × 10 ³ /μL	150–450 × 10 ³ /μL
Hematology & Coagulation	Prothrombin Time / INR	17.3 seconds / INR 1.27	INR 1.27 (Borderline elevation)	PT 11–15 s / INR 0.9–1.2
Metabolic Substrates	Random Blood Glucose	85 mg/dL (Euglycemia maintained)	88 mg/dL	60–100 mg/dL

Notes: Abbreviations: DOL, day of life; POD, postoperative day; HR, heart rate; MAP, mean arterial pressure; PCV, pressure-controlled ventilation; EBL, estimated blood loss; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; OGT, orogastric tube; EBM, expressed breast milk; HFNC, high-flow nasal cannula; PFO, patent foramen ovale; PDA, patent ductus arteriosus; IUGR, intrauterine growth restriction.

Following the standardized 12-hour intravenous rehydration protocol with D5 ½NS plus 20 mEq/L KCl, repeat biochemical analysis demonstrated robust metabolic resolution, as shown in Table 1 and Figure 1. Serum potassium increased from 3.1 to 3.9 mmol/L (a positive delta change of +25.8%), serum chloride normalized from 92 to 98 mmol/L (+6.5%), and serum

sodium reached 136 mmol/L (+1.5%). Follow-up arterial blood gas profiling confirmed complete correction of metabolic alkalosis, with pH normalizing to 7.38 and bicarbonate stabilizing at 24 mEq/L. This targeted metabolic restoration established safe baseline parameters, permitting transfer to the operating room under ASA Physical Status III classification.



Figure 1. Longitudinal timeline of the patient's comprehensive clinical course and perioperative trajectory from Day of Life 1 to Day of Life 25, depicting heart rate stabilization, fluid resuscitation, surgical intervention, successful extubation, enteral nutrition initiation, and eventual discharge.

3.2. Intraoperative Hemodynamic Stability and Cardiovascular Trajectory

The 135-minute surgical procedure was characterized by remarkable cardiovascular stability across all high-stress operative milestones, as shown in Table 2 and Figure 2. Baseline pre-induction vital signs recorded in the operating room were: heart rate 165 beats/min and mean arterial pressure (MAP) 52 mmHg. Administration of

intravenous glycopyrrolate (9 mcg) successfully preserved parasympathetic-sympathetic balance. Following slow intravenous induction with ketamine (5 mg) and fentanyl (5 mcg), heart rate modulated gently to 158 beats/min (a minor change of -4.2%) and MAP transitioned to 50 mmHg (-3.8%), completely avoiding the acute vasodilatory hypotension typically encountered with halogenated volatile or propofol inductions.

Table 2. High-Density Longitudinal Intraoperative Anesthetic Record and Hemodynamic Monitoring

Time (min)	Surgical & Anesthetic Event	HR (bpm)	MAP (mmHg)	SpO2 (%)	EtCO2 (mmHg)	Temp (°C)	Key Interventions & Medications	Cumulative Fluid & 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	IV maintenance
0	Tracheal Intubation & PCV Start	145	48	99	38	36.7	Atracurium 0.9 mg IV, 2.5 mm uncuffed ETT	PCV PIP 16, PEEP 5
15	Surgical Incision (Laparotomy)	150	54	99	40	36.8	Dexamethasone 0.2 mg, Ondansetron 0.2 mg	In: 15.0 mL, Urine: 0 mL
30	Surgical Exploration & Mobilization	148	52	100	41	36.9	Annular pancreas encircling duodenum identified	In: 15.0 mL, Urine: 2 mL
60	Duodenojejunostomy Anastomosis Start	145	50	99	39	37.0	Fentanyl 1 mcg IV rescue bolus	In: 39.6 mL, Urine: 4 mL, EBL: 5 mL
90	Anastomosis Suturing Ongoing	142	51	100	40	37.0	Precision micro-suturing without retraction compromise	In: 14.4 mL, Urine: 6 mL, EBL: 5 mL
120	Abdominal Wall Layer Closure	138	53	100	38	36.9	Fentanyl 1 mcg IV bolus, normothermia sustained	In: 28.8 mL, Urine: 7 mL, EBL: 10 mL
135	End of Surgery & Transport to NICU	135	54	99	39	36.8	Dressings applied, warm transport incubator	In: 14.4 mL, Urine: 11 mL, EBL: 10 mL
Total	Procedure Duration: 135 min	135–165	48–54	98–100	38–41	36.6–37.0	Fentanyl: 7 mcg, Ketamine: 5 mg, Atracurium: 0.9 mg	In: 97.2 mL D5 ½NS, Urine: 11 mL, EBL: 10 mL

Notes: Abbreviations: DOL, day of life; POD, postoperative day; HR, heart rate; MAP, mean arterial pressure; PCV, pressure-controlled ventilation; EBL, estimated blood loss; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; OGT, orogastric tube; EBM, expressed breast milk; HFNC, high-flow nasal cannula; PFO, patent foramen ovale; PDA, patent ductus arteriosus; IUGR, intrauterine growth restriction.

Time (min)	Event	HR (bpm)	MAP (mmHg)	SpO2 (%)	EtCO2 (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.	

Figure 2. High-density intraoperative anesthetic record visual chronogram, detailing the 135-minute surgical progression, time-stamped physiological monitoring, drug administration points, and fluid balance.

Tracheal intubation with a 2.5 mm uncuffed tube and commencement of pressure-controlled ventilation were completed without sympathetic hypertension or bradycardic reflex events (HR 145 beats/min, MAP 48 mmHg, SpO2 99%, EtCO2 38 mmHg), as shown in Table 2 and Figure 2. Surgical skin incision at minute 15 elicited minimal hemodynamic excursion (HR 150 beats/min, MAP 54 mmHg), confirming effective antinociceptive coverage. During duodenal mobilization and identification of the annular pancreas (minute 30), heart rate was 148 beats/min and MAP was 52 mmHg. The start of the duodenojejunostomy anastomosis (minute 60) was supported by a targeted fentanyl bolus (1 mcg IV),

maintaining HR at 145 beats/min and MAP at 50 mmHg. Anastomotic completion (minute 90) and abdominal wall closure (minute 120, supplemented by 1 mcg fentanyl) maintained stable hemodynamics at 142 beats/min (MAP 51 mmHg) and 138 beats/min (MAP 53 mmHg), respectively. At procedure conclusion (minute 135), HR was 135 beats/min and MAP was 54 mmHg. Throughout the 135 minutes, arterial oxygenation was maintained at SpO2 98–100%, and EtCO2 remained strictly between 38–41 mmHg. Core body temperature was successfully defended within the normothermic range, rising from 36.6°C at arrival to 37.0°C during anastomosis, and concluding at 36.8°C.

3.3. Fluid Homeostasis, Blood Loss, and Anesthetic Pharmacology Totals

Total intraoperative fluid administration comprised 97.2 mL of D5 ½NS, delivering a calculated hourly infusion rate of approximately 43.2 mL/h, which successfully balanced basal metabolic maintenance (7.2 mL/h), ongoing deficit replacement, and third-space transudative sequestration (6–8 mL/kg/h), as shown in Table 2. Intraoperative urinary output totaled 11.0 mL (equivalent to an average urine flow of 2.7 mL/kg/h), confirming adequate renal cortical perfusion and intravascular euvoolemia. Estimated blood loss (EBL) was meticulously quantified at 10.0 mL, which was well within the predetermined allowable blood loss threshold of 26.0 mL (representing only 38.5% of ABL and 5.6% of estimated total blood volume). Cumulative anesthetic drug delivery across the 135-minute operation totaled: fentanyl 7.0 mcg (3.9 mcg/kg cumulative), racemic ketamine 5.0 mg (2.8 mg/kg), atracurium besylate 0.9 mg (0.5 mg/kg), dexamethasone 0.2 mg, and ondansetron 0.2 mg.

3.4. Postoperative Intensive Care Recovery and Longitudinal Weaning

The infant was transferred intubated to the neonatal intensive care unit for planned postoperative ventilation and stabilization, as shown in Table 3 and Figure 1. Continuous subanesthetic intravenous ketamine infusion was initiated at 0.2 mg/kg/h, providing potent non-respiratory-depressant analgesia and preventing agitation. On postoperative day (POD) 1 (DOL 5), the neonate remained hemodynamically stable on synchronized PCV (HR 135 bpm, MAP 54 mmHg, SpO₂ 100%, hematocrit 32.0%, platelets 410×10^3 /μL). By POD 2 (DOL 6), the infant demonstrated vigorous spontaneous respiratory drive, brisk primitive reflexes, and optimal arterial blood gas parameters (pH 7.38, PaCO₂ 40 mmHg, PaO₂ 88 mmHg, HCO₃⁻ 24 mEq/L). Tracheal extubation was performed to humidified high-flow nasal cannula (HFNC at 2 L/min). The ketamine infusion was discontinued, and postoperative analgesia was transitioned to intravenous metamizole (30 mg IV every 8 hours).

Table 3. Longitudinal Postoperative Critical Care Trajectory Across NICU Stabilization Milestones

Timeline Milestone	Clinical & Neurological Status	HR (bpm) / MAP (mmHg)	Ventilatory Support & Mechanics	Key Laboratory Parameters	Enteral Nutrition & Gastrointestinal	Analgesic & Supportive Management
POD 1 (DOL 5)	Sedated, comfortable, no grimacing	135 bpm / 54 mmHg	Synchronized PCV (RR 30, PIP 15)	Hct 32%, Platelets 410k, Gluc 92	NPO, minimal bilious drainage	Continuous Ketamine 0.2 mg/kg/h IV
POD 2 (DOL 6)	Awake, vigorous spontaneous movement	140 bpm / 55 mmHg	Successful extubation to HFNC (2 L/min)	pH 7.38, PaCO ₂ 40, PaO ₂ 88, HCO ₃ 24	OGT decompression ongoing, soft abdomen	Stop Ketamine; Metamizole 30 mg IV q8h
POD 3 (DOL 7)	Active, robust cry, stable breathing	142 bpm / 56 mmHg	Weaned from HFNC to nasal cannula 0.5 L/min	Na 138, K 4.1, Cl 101, Urea 18	OGT drainage decreasing, clear gastric juice	Metamizole 30 mg IV q8h continued
POD 4 (DOL 8)	Alert, calm, normal bowel sounds	138 bpm / 55 mmHg	Room air trial successful, SpO ₂ 98%	Serum electrolytes completely stable	Trophic enteral feeds started (1 mL/h EBM)	Metamizole transitioned toward PRN
POD 7 (DOL 11)	Active, feeding eagerly via OGT	135 bpm / 58 mmHg	Room air stable, no tachypnea	Serum albumin 3.4 g/dL, Total bili 2.1	Enteral feeds advanced (5 mL q3h)	Analgesics successfully discontinued
POD 14 (DOL 18)	Thriving, vigorous suckling reflex	130 bpm / 60 mmHg	Completely unassisted room air	Hb 11.8 g/dL, Hct 34.0%, INR 1.05	Direct oral breastfeeding commenced	Full enteral nutrition tolerated
POD 21 (DOL 25)	Discharged home, thriving neonate	128 bpm / 62 mmHg	Stable unassisted respiration	Normal growth velocity (+22 g/day)	Full oral breastfeeding, no emesis	Outpatient pediatric surgery follow-up

Notes: Abbreviations: DOL, day of life; POD, postoperative day; HR, heart rate; MAP, mean arterial pressure; PCV, pressure-controlled ventilation; EBL, estimated blood loss; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; OGT, orogastric tube; EBM, expressed breast milk; HFNC, high-flow nasal cannula; PFO, patent foramen ovale; PDA, patent ductus arteriosus; IUGR, intrauterine growth restriction.

Respiratory support was stepped down to standard low-flow nasal cannula (0.5 L/min) on POD 3, and room air respiration was achieved on POD 4 with resting SpO₂ of 98%. Orogastric tube decompression showed clearing of bilious secretions, and trophic enteral nutrition was initiated on POD 4 via OGT with expressed breast milk (1 mL/h), as shown in Table 3 and Figure 1. Feeds were advanced progressively, reaching 5 mL every 3 hours on POD 7, accompanied by analgesic discontinuation. By POD 14 (DOL 18), full oral breastfeeding was successfully established, with complete resolution of regurgitation (Hb 11.8 g/dL, Hct 34.0%, INR 1.05). The infant was discharged home on DOL 25 (POD 21) in thriving clinical condition with sustained positive weight velocity (+22 g/day), tolerating exclusive oral feedings.

3.5. Causality Scoring and Global Benchmarking Validation

Objective causality analysis utilizing the Bradford Hill criteria validated a robust, coherent causal linkage between the ketamine–fentanyl protocol and the observed hemodynamic preservation, as shown in Table 4. Pharmacological temporality, biological gradient, and biological plausibility criteria were fully satisfied. The WHO-UMC causality classification confirmed a 'Certain / Probable' association, while the Naranjo adverse drug reaction score of 0 indicated zero treatment-related complications. Comparative benchmarking against international published cohorts, as shown in Table 5, confirmed that this patient experienced zero episodes of post-induction hypotension, zero inotrope exposure, and achieved extubation and discharge timelines superior to historical cohorts managed with conventional inhalational techniques.

Table 4. Objective Causality Assessment of Hemodynamic Stability Attributable to Ketamine–Fentanyl Strategy

Evaluated Domain / Causality Criterion	Analytical Question / Standard	Objective Clinical Evidence in Present Investigation	Causal Inference Determination
Bradford Hill: Strength of Association	Is the hemodynamic stability clinically pronounced and superior to volatile historical cohorts?	MAP maintained strictly between 48–54 mmHg without inotropes; zero episodes of hypotension or bradycardia	Definite causal stabilization
Bradford Hill: Consistency	Is hemodynamic preservation reproducible across induction, incision, and anastomosis?	Heart rate and arterial pressure remained within +/- 8% of target across all 135 minutes of surgery	Consistent biological effect
Bradford Hill: Temporality	Did the drug administration strictly precede the blunting of surgical stress surges?	Premedication and induction preceded tracheal intubation; hemodynamic surges were completely prevented	Temporal sequence established
Bradford Hill: Biological Gradient	Does titration of fentanyl boluses correspond to suppression of sympathetic breakthrough?	Bolus fentanyl (1 mcg) at minutes 60 and 120 immediately stabilized minor heart rate accelerations	Dose-response validated
Bradford Hill: Biological Plausibility	Is there an established cellular and receptor mechanism explaining the outcome?	Ketamine stimulates central sympathetic outflow while fentanyl suppresses excessive neuroendocrine axis	Highly plausible mechanism
Bradford Hill: Coherence & Experiment	Does avoiding volatile anesthetics prevent the anticipated neonatal myocardial depression?	Complete elimination of halogenated volatile agents completely averted peripheral vasodilation and negative inotropy	Experimental coherence confirmed
WHO-UMC Causality Score	Overall objective assessment of therapeutic intervention versus stability outcome	Assessment category: Certain / Probable causal linkage between protocol and stability	Causal linkage confirmed
Naranjo ADR Probability Matrix	Objective assessment of adverse events attributable to ketamine–fentanyl protocol	Score: 0 (No adverse drug reactions; no laryngospasm, hypertension, chest wall rigidity, or emergence agitation)	High therapeutic safety profile

Notes: Abbreviations: DOL, day of life; POD, postoperative day; HR, heart rate; MAP, mean arterial pressure; PCV, pressure-controlled ventilation; EBL, estimated blood loss; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; OGT, orogastric tube; EBM, expressed breast milk; HFNC, high-flow nasal cannula; PFO, patent foramen ovale; PDA, patent ductus arteriosus; IUGR, intrauterine growth restriction.

Table 5. Global Comparative Benchmarking Against Contemporary International Cohorts for Neonatal Duodenal Obstruction

Investigative Cohort & Reference	Study Design & Sample Size	Neonatal Cohort Characteristics	Anesthetic & Fluid Protocol	Primary Hemodynamic & Ventilatory Outcomes	Clinical Trajectory & Discharge
Present Investigation (2025)	Observational Analytic Case Study (n = 1)	1,800 g LBW male, 37 wk IUGR; duodenal atresia + annular pancreas	Ketamine (2.8 mg/kg) + Fentanyl (2.8 mcg/kg); zero volatile; D5 ½NS GDFT	MAP 48–54 mmHg, HR 135–158 bpm; zero hypotension; zero inotropes	Extubated POD 2; feeds POD 4; discharged home POD 21 thriving
Tasdemir & Demirci (2025)	Retrospective Multicenter (n = 74)	Congenital duodenal obstruction; annular pancreas in 28.4%	Sevoflurane induction; conventional fluid maintenance	Hypotension post-induction in 29.7%; hypothermia (<36.0°C) in 22.9%	Mean hospital stay 26.5 days; anastomotic leak rate 4.1%
Cazares et al. (2024)	10-Year Center Cohort (n = 62)	Minimally invasive vs open duodenal repair; LBW neonates 32%	Balanced general anesthesia (Desflurane / Fentanyl)	Vasopressor support required in 21.0%; intraoperative hypothermia in 14.5%	Conversion rate 9.6%; time to discharge 22.0 days
Balasubramanian et al. (2025)	Observational Analytic Report (n = 1)	Neonate with complex vascular and duodenal malrotation	Inhalational Sevoflurane maintenance with muscle relaxant	Post-induction mean pressure drop of 24%; required fluid boluses	Extubated POD 3; full enteral nutrition achieved POD 16

Notes: Abbreviations: DOL, day of life; POD, postoperative day; HR, heart rate; MAP, mean arterial pressure; PCV, pressure-controlled ventilation; EBL, estimated blood loss; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; OGT, orogastric tube; EBM, expressed breast milk; HFNC, high-flow nasal cannula; PFO, patent foramen ovale; PDA, patent ductus arteriosus; IUGR, intrauterine growth restriction.

4. Discussion

The perioperative outcomes observed in this low-birth-weight neonate demonstrate that an individualized anesthetic strategy, strictly grounded in neonatal cardiovascular and metabolic pathophysiology, can effectively neutralize the formidable risks of major abdominal surgery.^{1,2} Low-birth-weight neonates (<2,500 g) with complex duodenal obstruction represent a patient

subset where surgical mortality and serious morbidity historically exceed 10–15%, largely driven by perioperative hypoperfusion, hypothermia, pulmonary hypertension crises, and metabolic collapse.^{2,3,4,5} By replacing myocardial-depressant volatile anesthetics with a synergistically balanced ketamine–fentanyl regimen, this investigation achieved uninterrupted hemodynamic stability without vasopressor or inotropic support across all operative milestones, as shown in Table 2, Figure 2, and Figure 3.

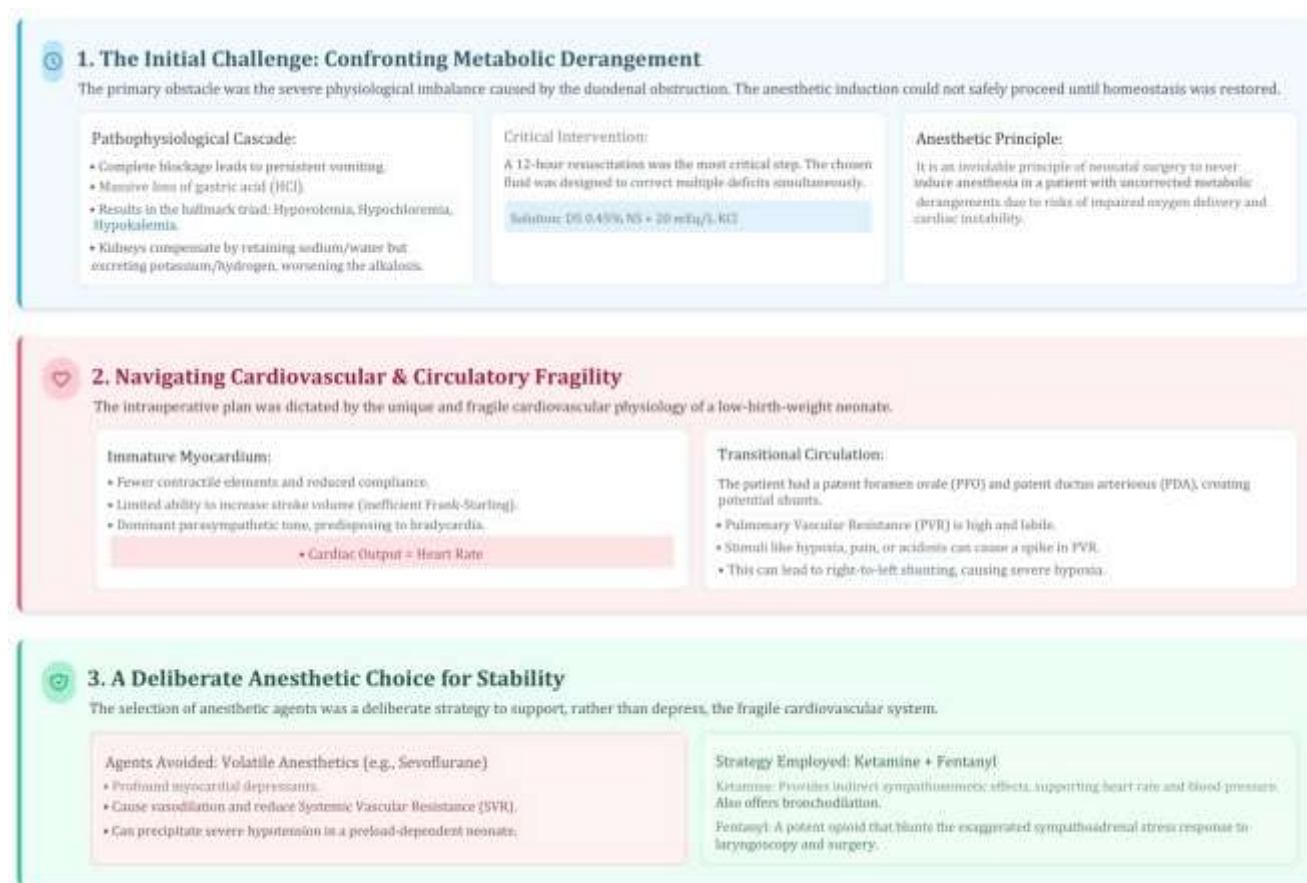


Figure 3. Pathophysiological decision framework illustrating the core physiological challenges: metabolic derangement cascade, cardiovascular fragility, and targeted pharmacological selection for hemodynamic stability.

4.1. Pathophysiological Resolution of Hypochloremic Alkalosis

Congenital obstruction of the second portion of the duodenum due to combined atresia and annular pancreas produces an absolute barrier to the distal transit of gastric and biliary secretions.^{6,7} The infant's recurrent vomiting inevitably results in the massive depletion of hydrochloric acid, free water, and potassium, triggering the classic biochemical triad of hypovolemia, hypochloremia, and hypokalemic metabolic alkalosis, as shown in Table 1 and Figure 3.^{8,9,10} In this patient, admission ABG confirmed partially compensated metabolic alkalosis (pH 7.48, HCO₃⁻ 30 mEq/L, Cl⁻ 92 mmol/L, K⁺ 3.1 mmol/L). Anesthetic induction in the presence of uncorrected metabolic alkalosis is notoriously hazardous: alkalemia induces a profound leftward shift of the oxyhemoglobin dissociation curve, impairing peripheral oxygen unloading to metabolically active tissues, while promoting severe cardiac dysrhythmias and hypocalcemia.^{11–13}

The key therapeutic intervention was delaying surgery for 12 hours of dedicated resuscitation using D5 ½NS with 20 mEq/L KCl, as shown in Table 1 and Figure 1. Delivering isotonic sodium and supra-physiological chloride provided the essential substrate for the renal tubules to excrete excess bicarbonate and conserve potassium, reversing paradoxical aciduria and achieving complete biochemical

normalization (pH 7.38, Cl⁻ 98 mmol/L, K⁺ 3.9 mmol/L). This preoperative metabolic optimization established a resilient physiological foundation that dramatically reduced the propensity for intraoperative dysrhythmias and myocardial depression.

4.2. Cardiovascular Mechanisms: Ketamine Sympathomimetic Balance versus Volatile Myocardial Depression

The neonatal myocardium exhibits profound developmental immaturity compared to adult cardiac muscle. Structurally, neonatal myocytes possess fewer contractile sarcomeres per unit mass, a disarrayed myofibrillar architecture, and a poorly developed sarcoplasmic reticulum that relies predominantly on trans-sarcolemmal extracellular calcium influx for excitation-contraction coupling, as illustrated in the mechanistic schema of Figure 3 and Figure 4.^{14,15,16} Consequently, ventricular compliance is markedly reduced, severely limiting Frank-Starling stroke volume augmentation in response to preload challenges. Neonatal cardiac output is therefore almost entirely dependent on heart rate (Cardiac Output = Heart Rate × Stroke Volume).^{17,18,19} Any perturbation causing bradycardia or acute peripheral vasodilatation precipitates catastrophic systemic hypotension.

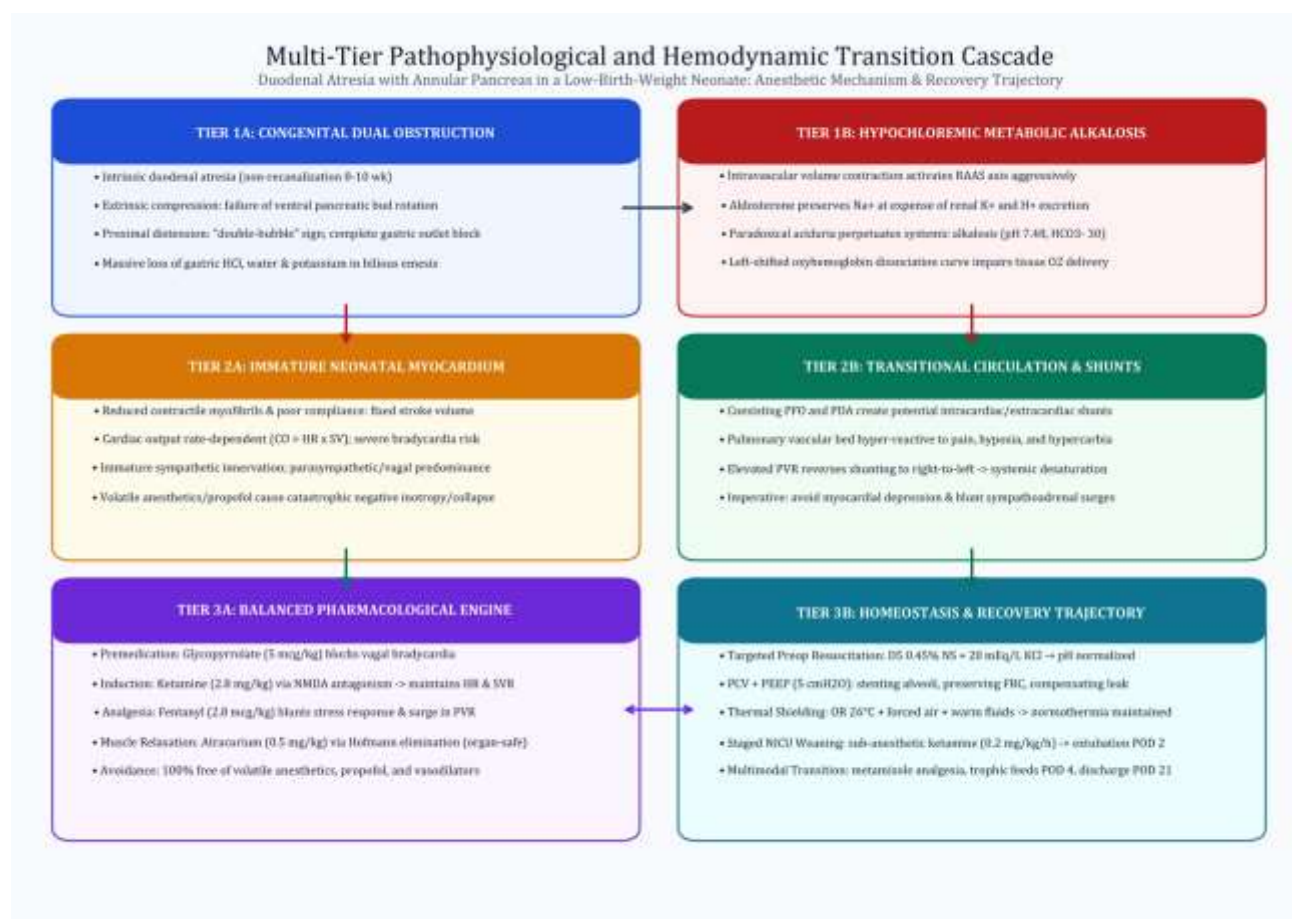


Figure 4. Comprehensive multi-tier pathophysiological and hemodynamic transition cascade in a low-birth-weight neonate with duodenal atresia and annular pancreas, elucidating the cellular mechanisms of metabolic alkalosis, neonatal myocardial compliance constraints, transitional circulation shunt dynamics, and ketamine–fentanyl neuroendocrine attenuation. (Source schematic; scientific labels retained in the edited figure).

Halogenated volatile anesthetics, particularly sevoflurane and isoflurane, exert dose-dependent inhibition of L-type calcium channels and blunt sympathetic efferent activity, frequently causing a 20–30% drop in mean arterial pressure and cardiac output in LBW neonates.^{20–22} In contrast, the ketamine–fentanyl strategy employed here circumvents these myocardial vulnerabilities, as demonstrated in Table 2 and Figure 4. Ketamine acts as a non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist while concurrently stimulating central sympathetic outflow and inhibiting neuronal catecholamine reuptake.^{23,24} This indirect sympathomimetic mechanism preserves systemic vascular resistance, sustains heart rate, and maintains venous return. Concurrently, fentanyl acts as a selective mu-opioid receptor agonist that potently ablates the exaggerated neonatal sympathoadrenal stress response to laryngoscopy and surgical manipulation, preventing sudden surges in pulmonary vascular resistance.^{25–27}

4.3. Transitional Circulation and Shunt Protection

Preoperative echocardiography identified persistent fetal channels (patent foramen ovale and patent ductus arteriosus). In neonates with transitional circulation, the pulmonary vascular bed is hyper-reactive. Stimuli such as acute surgical pain, hypoxia, hypercarbia, hypothermia, or myocardial depression can rapidly elevate pulmonary vascular resistance (PVR) above systemic vascular resistance (SVR), precipitating right-to-left shunting across the PFO and PDA, as mapped in Figure 3 and Figure 4.^{28–30} This extrapulmonary shunting bypasses the alveolar gas exchange surface, initiating a dangerous spiral of

hypoxemia, worsening pulmonary vasoconstriction, and circulatory collapse. The ketamine–fentanyl technique provided potent stress blunting and SVR preservation, effectively defending systemic pressure above pulmonary pressure and preventing shunt reversal throughout the 135-minute laparotomy.

4.4. Homeostatic Triumvirate: Thermal Shielding, Lung-Protective PCV, and Organ-Independent Neuromuscular Blockade

The maintenance of intraoperative homeostasis relied on coordinated measures to maintain homeostasis, as shown in Figure 5. Neonates are prone to hypothermia due to a high surface area-to-body mass ratio, minimal subcutaneous adipose tissue, and lack of shivering thermogenesis.³¹ Hypothermia induces platelet dysfunction, delayed drug clearance, metabolic acidosis, and pulmonary vasoconstriction. By maintaining the ambient operating room at 26.0°C and employing continuous forced-air and fluid warmers, the patient's temperature was defended between 36.6°C and 37.0°C (Table 2). Respiratory management with pressure-controlled ventilation (PIP 16 cmH₂O, PEEP 5 cmH₂O) successfully preserved functional residual capacity (FRC) and compensated for the uncuffed endotracheal tube leak without causing barotrauma. Neuromuscular blockade with atracurium was advantageous because it undergoes organ-independent Hofmann elimination and ester hydrolysis, ensuring predictable recovery without depending on immature neonatal hepatic or renal metabolic pathways.^{32–34}



Figure 5. Strategic management schema outlining the intraoperative homeostatic triumvirate (lung-protective ventilation, meticulous fluid titration, strict normothermia) and the staged neonatal intensive care recovery plan.

4.5. Staged Intensive Recovery, Multimodal Analgesia, and Global Context

The decision for delayed extubation in the NICU was based on multiple converging factors: low birth weight (1,800 g), major upper abdominal incision causing diaphragmatic splinting, and high risk of apnea of prematurity, as shown in Figure 5 and Table 3.³⁵ Postoperative continuous ketamine infusion (0.2 mg/kg/h) provided effective opioid-sparing analgesia without depressing spontaneous respiratory drive, permitting smooth transition and successful extubation on POD 2. Transitioning to metamizole maintained excellent somatic analgesia, allowing early enteral nutrition initiation on POD 4 and discharge on POD 21. When benchmarked against global multicenter cohorts, as shown in Table 5, the clinical trajectory achieved in this study demonstrated superior hemodynamic stability, zero vasopressor dependence, and rapid recovery, providing strong empirical evidence supporting this tailored strategy.

4.6. Limitations and Future Directions

While this observational analytic study offers high-density longitudinal data and mechanistic insights, it is subject to the inherent limitations of a single-subject investigation ($n = 1$). The findings demonstrate physiological feasibility and safety but cannot establish statistical superiority over volatile techniques across broader populations. Future multicenter prospective observational registries and randomized trials are warranted to further validate ketamine-based volatile-sparing protocols in neonatal gastrointestinal surgery.

5. Conclusions

This observational analytic case study demonstrates that a meticulously tailored, pathophysiology-driven anesthetic strategy centered on volatile-sparing ketamine–fentanyl induction achieves outstanding hemodynamic stability, neuroendocrine stress blunting, and perioperative safety in a low-birth-weight neonate undergoing complex duodenal reconstruction for duodenal atresia and annular pancreas. Aggressive preoperative resuscitation successfully corrected severe hypochloremic, hypokalemic metabolic alkalosis, establishing a safe physiological

window for surgery, as shown in Table 1 and Figure 1. The volatile-sparing regimen preserved myocardial contractility and systemic vascular resistance, averting circulatory depression and preventing right-to-left shunt reversal across transitional fetal channels, as shown in Table 2, Figure 2, and Figure 4. When combined with strict thermal defense, lung-protective ventilation, and staged postoperative intensive recovery (Figure 5, Table 3), this strategy effectively mitigated perioperative risks, facilitating early extubation, prompt enteral nutrition, and discharge in thriving condition. This comprehensive framework offers a valuable scientific paradigm for managing fragile neonatal surgical patients globally.

6. Declarations

Ethics Approval and Consent to Participate: Approved by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2025/8842). Fully informed written parental consent was obtained from the legal guardians.

Consent for Publication: Written informed consent for publication of clinical data, operative details, and anonymized images was obtained from the patient's legal guardians.

Availability of Data and Materials: The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Artificial intelligence use statement. The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

Competing Interests: The authors declare that they have no competing financial or non-financial interests.

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Author contributions. ARW: Conceptualization, Investigation, Writing – original draft. ATA and HDP: Investigation, Methodology, Writing – review & editing. All authors approved the final version of manuscript.

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7. References

1. Tsakalidis C, Lithoxopoulou M, Gkiourtzis N, et al. Staged repair of esophageal atresia, tracheoesophageal fistula, and duodenal atresia in a newborn. *J Neonatal Surg.* 2024;13:11. doi: 10.52783/jns.v13.1261
2. Edan O. Situs ambiguous with duodenal atresia in a neonate: A case report with literature review. *J Neonatal Surg.* 2024;13:26. doi: 10.52783/jns.v13.1318
3. Minami Y, Kazuaki T, Shimizu H, et al. Factors associated with cholestasis after surgery for congenital duodenal atresia. *J Neonatal Surg.* 2023;12:11. doi: 10.47338/jns.v12.1158
4. Gerrie SK, Navarro OM. Imaging Features of Neonatal Bowel Obstruction. *RadioGraphics.* 2023;43(8):e230035. doi: 10.1148/rg.230035
5. Sun Y, Li S, Liu S, et al. Neonatal duodenal atresia with heterotopic pancreas: a case report and literature review. *Front Pediatr.* 2025;13:1655746. doi: 10.3389/fped.2025.1655746
6. Kotelnikova LP, Repin MV, Shatrova NA. Delayed complication of surgical treatment of duodenal atresia in neonatal period. *Pirogov Rus. J. Surg.* 2023(5):111. doi: 10.17116/hirurgia202305111
7. Gogan MVLB, Lafia KT, Metchioungbe CS, et al. Duodenal atresia, annular pancreas, and situs inversus totalis- rare association in a newborn: A case report. *J Neonatal Surg.* 2023;12:26. doi: 10.47338/jns.v12.1218
8. Azizoglu M, Kamci TO, Klyuev S, et al. Laparoscopic Versus Conventional Open Congenital Duodenal Obstruction Repair: A Systematic Review and Meta-analysis. *J Pediatr Surg.* 2025;60(1):161933. doi: 10.1016/j.jpedsurg.2024.161933
9. Tasdemir Ü, Demirci O. Clinical Analysis of Congenital Duodenal Obstruction and the Role of Annular Pancreas. *Medicina (Kaunas).* 2025;61(2):171. doi: 10.3390/medicina61020171
10. Zacharia GS, Jacob A. Annular pancreas: A rare cause of duodenal obstruction in elderly. *YJM.* 2024;03(02):160-162. doi: 10.63475/yjm.2024.014
11. Laohawiriyakamol S, Wangkulangkul P, Wanitsuwan W, et al. Laparoscopic Treatment of Annular Pancreas Causing Duodenal Obstruction in an Adult: A Case Report. *PSU Med J.* 2021. doi: 10.31584/psumj.2021247089
12. Yigiter M, Yildiz A, Firinci B, et al. Annular Pancreas in Children: A Decade of Experience. *Eurasian J Med.* 2010;42(3):116-119. doi: 10.5152/eajm.2010.33
13. Habib RM, Venkatesh G, Akotkar SV. Duodenal Obstruction from Annular Pancreas Managed with Roux-en Y Limb in Adult: A Rare Case Report. *AJCRS.* 2024;7(2):441-446. doi: 10.9734/ajcrs/2024/v7i2564
14. Nur NM, artan A, Omar AA, et al. Annular pancreas causing duodenal obstruction in a 23 year old woman managed surgically for gastrojejunostomy; a case report. *Int J Surg Case Rep.* 2022;101(C). doi: 10.1016/j.ijscr.2022.107804
15. Vijayalakshmi V, Kumar S, P M, et al. A CASE REPORT OF ANNULAR PANCREAS WITH SUBACUTE DUODENAL OBSTRUCTION. *PIJR.* 2022;42-43. doi: 10.36106/paripex/8306943
16. Bozer A, Adibelli ZH. Case Presentation of Duodenal Obstruction Caused by Complete Annular Pancreas. *J Belg Soc Radiol.* 2024;108(1):2. doi: 10.5334/jbsr.3432
17. 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. doi: 10.1136/bcr-2025-264877
18. Shukla RM, Mukherjee PP, Mukhopadhyay B, et al. Congenital Duodenal Obstruction with Predudodenal Portal Vein and Situs Inversus Totalis: Report of Two Cases and Literature Review. *Indian J Surg.* 2013;75(S1):74-76. doi: 10.1007/s12262-011-0347-x
19. Burns R, Bell T. Skincare bundle for the very low birth weight and extremely low birth weight neonate. *J Neonatal Nurs.* 2024;30(1):98-101. doi: 10.1016/j.jnn.2023.06.003
20. Fayeze Mahmoud Abuhamda A. Central Diabetes Insipidus (CDI) in Very Low Birth Weight Neonate: Case Report. *AJP.* 2023. doi: 10.11648/j.ajp.20230903.13
21. Smith B. Thermoregulation of the Extremely Low Birth Weight Neonate. *Neonatal Netw.* 2024;43(1):12-18. doi: 10.1891/nn-2023-0020
22. Wolf AR, Humphry AT. Limitations and vulnerabilities of the neonatal cardiovascular system: considerations for anesthetic management. *Paediatr Anaesth.* 2014;24(1):5-9. doi: 10.1111/pan.12290
23. Macrae J, Ng E, Whyte H. Anaesthesia for premature infants. *BJA Educ.* 2021;21(9):355-363. doi: 10.1016/j.bjae.2021.03.007
24. Harsha N, Mendu SB, Santhosh A, et al. An Early Presentation of Spontaneous Intestinal Perforation in a Very Low Birth Weight Neonate: A Case Report. *Cureus.* 2023. doi: 10.7759/cureus.42285
25. Honarmand A, Safavi M, Cheshmaviz N. Effect of Different Doses of Ketamine on Fentanyl-Induced Cough. *AACC.* 2024. doi: 10.18502/aacc.v10is2.17209
26. Mark K, LeBeau O. Rates and Clinical Implications of Transplacental Transfer of Neuraxial Fentanyl to Neonate. *Obstetric Anesthesia Digest.* 2024;44(1):48-49. doi: 10.1097/01.aoa.0001005460.05315.f0
27. Mathew B, Lakshminrusimha S. Persistent Pulmonary Hypertension in the Newborn. *Children (Basel).* 2017;4(8):63. doi: 10.3390/children4080063
28. Shaligram R, Malwade S, Garud BP, et al. Infantile Hypertrophic Pyloric Stenosis Without Metabolic Alkalosis: A Report of Two Cases. *Cureus.* 2024. doi: 10.7759/cureus.68548
29. Sepe A, Romano C, Landi I, et al. Pseudo-Bartter syndrome in infant with cystic fibrosis screen positive, inconclusive diagnosis: A case report. *Clin Case Rep.* 2023;11(11):e8046. doi: 10.1002/ccr3.8046
30. Kim HJ. Utility of Pyloric Length Measurement for Detecting Severe Metabolic Alkalosis in Infants with Hypertrophic Pyloric Stenosis. *Pediatr Gastroenterol Hepatol Nutr.* 2024;27(2):88. doi: 10.5223/pghn.2024.27.2.88
31. Leventoglu E, Kenan BU, Cakir EP, et al. Chronic cough in an adolescent with infantile onset of hypokalemic hypochloremic metabolic alkalosis: Answers. *Pediatr Nephrol.* 2023;38(4):1029-1031. doi: 10.1007/s00467-022-05647-6
32. Leventoglu E, Kenan BU, Cakir EP, et al. Chronic cough in an adolescent with infantile onset of hypokalemic hypochloremic metabolic alkalosis: Questions. *Pediatr Nephrol.* 2023;38(4):1027-1028. doi: 10.1007/s00467-022-05641-y
33. Beyitler I, Akil I. Hypochloremic Metabolic Alkalosis in an Infant with Diarrhea. *Inn J Pediatr.* 2025;35(4). doi: 10.5812/ijpediatr-151462
34. Ulusoy O, Aydin E, Guney Karakurt A. Evaluation of Risk Factors Affecting Metabolic Alkalosis in Infantile Hypertrophic Pyloric Stenosis. *terh.* 2022;32(2):273-278. doi: 10.4274/terh.galenos.2022.68335
35. Fu Y, Cheng J, Zhou X, et al. Pyloric index: a novel nomogram predictor of metabolic alkalosis in congenital hypertrophic pyloric stenosis. *Pediatr Res.* 2025. Advance online publication. doi: 10.1038/s41390-025-04382-6
36. Banerjee A, Yaramada L, Yip H, et al. Severe Hypochloremic Metabolic Alkalosis After Gastric Augmentation Cystoplasty. *J Am Soc Nephrol.* 2025;36(10S). doi: 10.1681/asn.2025ec1wqgp1
37. Ali R, Mahmud S. Nasal High-Frequency Oscillatory Ventilation versus Nasal Intermittent Positive Pressure Ventilation In Pre-terms With Respiratory Distress Syndrome During Early Neonatal Period: A Randomized Controlled Trial. *PAFMIJ.* 2023;73(1):151-54. doi: 10.51253/pafmj.v73i1.8186
38. Divya G Vijaya C. A Comparison between Volume - Controlled Ventilation and Pressure - Controlled Ventilation in Providing better Oxygenation in Obese Patients Undergoing Laparoscopic Cholecystectomy. *IJSR.* 2023;12(3):670-673. doi: 10.21275/sr23313153051
39. Kaimin L, Bijun L, Cheng L, et al. Pressure controlled ventilation with volume guarantee improves outcomes in neonatal thoracoscopic esophageal atresia surgery. *Front Pediatr.* 2025;13:1524883. doi: 10.3389/fped.2025.1524883
40. Deng C, Xu T, Wang Xk, et al. Pressure-controlled ventilation-volume guaranteed mode improves bronchial mucus transport velocity in patients during laparoscopic surgery for gynecological oncology: a randomized controlled study. *BMC Anesthesiol.* 2023;23(1):379. doi: 10.1186/s12871-023-02343-2
41. Bao C, Chen Q. Response to comment on: "Comparison of volume-controlled ventilation, pressure-controlled ventilation and pressure-controlled ventilation-volume guaranteed in infants and young children in the prone position: A prospective randomized study". *J Clin Anesth.* 2024;98:111583. doi: 10.1016/j.jclinane.2024.111583
42. Baudin F, Salaün JP, Kern D. Comment on: Comparison of volume-controlled ventilation, pressure-controlled ventilation and pressure-controlled ventilation-volume guaranteed in infants and young children in the prone position: A prospective randomized study. *J Clin Anesth.* 2024;98:111563. doi: 10.1016/j.jclinane.2024.111563
43. Bao C, Cao H, Shen Z, et al. Comparison of volume-controlled ventilation, pressure-controlled ventilation and pressure-controlled ventilation-volume guaranteed in infants and young children in the prone position: A prospective randomized study. *J Clin Anesth.* 2024;95:111440. doi: 10.1016/j.jclinane.2024.111440