ORIGINAL ARTICLE

Staged Gastrostomy Decompression, Total Parenteral-Enteral Nutritional Optimization, and Delayed Primary Thoracotomy Repair in a Late-Referred Term Neonate With Gross Type C Esophageal Atresia and Symmetric Intrauterine Growth Restriction

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ABSTRACT

Background: Late referral of neonates with Gross type C esophageal atresia and distal tracheoesophageal fistula (EA/TEF) can be complicated by aspiration pneumonia and nutritional depletion.

Objective: To describe physiological, nutritional, and surgical recovery in a late-referred term neonate with EA/TEF and reported symmetric intrauterine growth restriction.

Methods: This longitudinal observational case study at Dr. M. Djamil General Hospital, Padang, Indonesia, covered inpatient and outpatient care from June 16 to August 11, 2025. Serial clinical, biochemical, and anthropometric measurements were described using percentage changes, oxygenation ratios, and growth velocity. The manuscript also reports Bradford Hill and Naranjo assessments; no within-patient inferential testing was performed.

Results: A 7-day-old male neonate with a birth weight of 2,600 g presented with aspiration pneumonia, oxygen saturation of 88%, and weight loss to 2,200 g (15.38%). Stamm gastrostomy on hospital day 2, parenteral nutrition (84–110 kcal/kg/day), two albumin infusions (1.0 g/kg each), and trophic enteral feeding preceded weight recovery to 2,700 g. Thoracotomy, fistula ligation, and primary anastomosis were performed on day 17. Oral intake reached 280–320 mL/day by discharge on day 27, when weight was 2,955 g. Follow-up weight reached 3,700 g.

Conclusion: This case describes recovery following staged decompression, nutritional support, and delayed repair. A single case cannot establish comparative safety, treatment efficacy, or causality.

1. Introduction

Esophageal atresia (EA) with or without tracheoesophageal fistula (TEF) is a life-threatening congenital malformation of the primitive foregut, occurring at a global incidence of approximately 1 in 2,500 to 1 in 4,500 live births.[1,2] The anomaly arises during the fourth to sixth weeks of embryonic organogenesis due to incomplete dorsoventral septation of the lateral tracheoesophageal ridges into a ventrally positioned respiratory primordium and a dorsally positioned esophageal conduit.[3,4] Under normal developmental conditions, spatiotemporal gradients of Sonic hedgehog (SHH), SRY-box transcription factor 2 (SOX2), and NK2 homeobox 1 (NKX2-1) orchestrate the programmed remodeling of the foregut epithelium and surrounding splanchnic mesenchyme. Disruption of these molecular signaling networks arrests longitudinal esophageal canalization and leaves a persistent fistulous communication between the posterior membranous trachea and the rudimentary esophageal segments.[5,6] Within the classical anatomical taxonomy established by Gross and Vogt, Gross Type C (corresponding to Vogt Type 3B), characterized by a blind-ending hypertrophied proximal esophageal pouch and a distal esophageal segment communicating directly with the distal trachea or carina, accounts for 85% to 90% of all congenital tracheoesophageal malformations worldwide.[7,8]

From a pathophysiological standpoint, Gross Type C EA/TEF imposes an immediate, self-amplifying dual aerodigestive insult beginning with the neonate's first breaths and feeding attempts.[9,10] Because the blind upper esophageal pouch cannot conduct swallowed saliva or breast milk into the stomach, secretions accumulate rapidly within the cervical esophagus and spill over the aryepiglottic folds into the larynx and tracheobronchial tree, provoking paroxysmal choking, frothy oral sialorrhea, cyanosis, and anterograde bacterial aspiration.[11,12] Simultaneously, the patent distal tracheoesophageal fistula creates a low-resistance pneumatic and chemical shunt between the tracheobronchial airway and the stomach. During crying, tachypneic breathing, or positive-pressure mask ventilation, inspired gas is diverted through the distal fistula into the stomach, producing gastric distension that elevates intragastric pressure and restricts diaphragmatic excursion.[13,14] Conversely, acidic gastric juice and proteolytic pepsin reflux retrogradely through the distal fistula directly into the lower respiratory tract, inducing chemical pneumonitis, surfactant inactivation, atelectasis, and secondary bacterial bronchopneumonia.[15,16]

While contemporary neonatal intensive care and refined surgical techniques have elevated index-admission survival rates above 90% in high-income healthcare systems, perioperative mortality in low- and middle-income countries (LMICs) remains high, frequently ranging between 30% and 65%.[17,18] This survival disparity is driven

primarily by low rates of prenatal sonographic detection, delayed postnatal clinical recognition at peripheral birthing facilities, uncoordinated inter-hospital transport without continuous proximal pouch suction, and presentation beyond 72 hours of life after repeated oral feeding attempts have established severe aspiration pneumonia and protein-energy malnutrition.[19,20] Although prenatal high-resolution ultrasonography can detect suggestive features such as maternal polyhydramnios, an absent or micro-gastric bubble, and the dynamic upper neck or mediastinal esophageal pouch sign from 22 weeks of gestation onward, routine antenatal screening in resource-constrained settings often misses these subtle markers, particularly when a distal tracheoesophageal fistula permits partial amniotic fluid passage into the fetal stomach.[12,21]

Prognostic stratification in neonatal EA/TEF has historically relied on the Spitz classification, which designates neonates with a birth weight of at least 1,500 g and no major congenital cardiac anomaly as Group I, predicting survival rates approaching 95% to 98% in tertiary centers.[18,22] However, the classical Spitz framework treats birth weight as a binary threshold at 1,500 g and does not account for the profound physiological vulnerability of full-term neonates who suffer from symmetric intrauterine growth restriction (IUGR) and small-for-gestational-age (SGA) anthropometry despite having a birth weight numerically above 2,500 g.[11,23] Symmetric IUGR reflects chronic antenatal uteroplacental nutrient deprivation or intrauterine inflammatory stress beginning in the first or second trimester, resulting in proportional reductions across body weight, crown-heel length, and occipitofrontal head circumference, alongside depleted hepatic glycogen stores, diminished subcutaneous adipose insulation, and impaired humoral and cellular immunity.[24,25] When a term neonate with symmetric IUGR experiences a 7-day diagnostic delay with uncompensated vomiting and aspiration pneumonia, the convergence of antenatal growth restriction and acute postnatal starvation rapidly precipitates severe catabolic weight loss, hypoalbuminemia, vitamin K-dependent coagulopathy, and hyperbilirubinemia.[26,27]

In such physiologically decompensated, late-referred neonates, attempting immediate single-stage primary thoracotomy repair during active bilateral pneumonia and profound catabolism carries a prohibitive risk of intraoperative hypoxemic arrest, anastomotic tension, suture-line dehiscence, and fatal mediastinitis.[28,29] Conversely, a structured staged damage-control and nutritional rehabilitation paradigm—combining continuous proximal esophageal pouch suction, non-invasive positive-pressure airway recruitment, Stage-1 Stamm gastrostomy to vent gastric gas and eliminate trans-fistula acid reflux, central venous total parenteral nutrition (TPN) with composite fish-oil lipid emulsions, targeted human albumin replacement, and trophic gastrostomy enteral priming prior to delayed Stage-2 primary esophageal anastomosis—offers a rational physiological bridge to survival.[30,31] Despite its clinical importance for LMIC tertiary referral centers, detailed longitudinal data quantifying the day-by-day cardiorespiratory, hematologic-inflammatory, biochemical, and 50-week postmenstrual age Fenton catch-up growth kinetics of this staged approach in symmetric IUGR neonates remain scarce. Therefore, the novelty and primary aim of this observational analytic case study were to characterize the 63-day longitudinal physiological trajectory, staged gastrostomy-to-thoracotomy surgical mechanics, three-tier antimicrobial stewardship, and dual parenteral-enteral nutritional rehabilitation of a late-referred term neonate with Gross Type C EA/TEF and symmetric IUGR.

2. Methods

Study Design

This investigation was designed as a prospective-retrospective hybrid observational analytic longitudinal case study conducted within the Level III Neonatal Intensive Care Unit (NICU), Pediatric Surgery Operating Theater, and Outpatient Multidisciplinary Follow-Up Clinic of Dr. M. Djamil General Hospital, Faculty of Medicine, Universitas Andalas, Padang, West Sumatera, Indonesia. The study observation window spanned 63 consecutive days from the neonate's birth on June 9, 2025, through tertiary inpatient hospitalization between June 16, 2025 (chronological age 7 days; postmenstrual age [PMA] 42 weeks) and July 12, 2025 (chronological age 33 days; PMA 46.5 weeks), and continued through weekly outpatient anthropometric surveillance up to August 11, 2025 (chronological age 63 days; PMA 50 weeks). Clinical reporting and methodological architecture adhered strictly to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines integrated with the Consensus-based Clinical Case Reporting (CARE) analytical framework.[17,18]

Ethics Governance

The study protocol received formal ethical clearance from the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2025/0519), and was conducted under the institutional clinical governance of the Department of Child Health, Faculty of Medicine, Universitas Andalas, and Dr. M. Djamil General Hospital, Padang, Indonesia. All diagnostic, surgical, nutritional, and antimicrobial interventions were performed strictly in accordance with international evidence-based neonatal intensive care standards and the ethical principles of the Declaration of Helsinki (2013 Fortaleza revision). Prior to data curation and manuscript preparation, comprehensive verbal and written explanations regarding the clinical course, surgical procedures, radiological and intraoperative photographic documentation, and scientific publication were provided to the patient's legal guardians (father Mr. DA, aged 42 years, and mother Ms. D, aged 40 years), who signed a formal written informed consent for clinical care and anonymized publication. In strict compliance with International Committee of Medical Journal Editors (ICMJE) privacy mandates, all patient-identifying metadata on growth charts and PACS radiograph headers, as well as craniofacial and orbital regions on thoracic-abdominal babygrams, were permanently masked with solid black anonymization bars.

Participant Eligibility

Participant eligibility encompassed a full-term male neonate (born at 41 weeks and 0 days of gestation) referred from a secondary regional hospital (Dr. M. Zain Painan Regional Hospital) on day 7 of life with a clinical and radiographic diagnosis of Gross Type C (Vogt Type 3B) esophageal atresia with distal tracheoesophageal fistula, complicated by bilateral aspiration bronchopneumonia, early postnatal catabolic weight loss exceeding 15% of birth weight, Kramer Grade III neonatal hyperbilirubinemia, and baseline symmetric intrauterine growth restriction (birth weight < 3rd percentile, body length < 10th percentile, and head circumference < 10th percentile on the Fenton preterm/term growth chart). Exclusion of major cyanotic or duct-dependent congenital heart disease and verification of Spitz Group I prognostic eligibility were established via comprehensive two-dimensional transthoracic echocardiography with color Doppler interrogation performed by a consultant pediatric cardiologist on Hospital Day 5 (June 20, 2025).

Longitudinal Observation Protocol

Longitudinal clinical, cardiorespiratory, surgical, laboratory, and nutritional parameters were systematically ascertained across eight sequential, clinically defined observation epochs: Pre-Referral Perinatal Baseline (T_{-1}: June 9–15, 2025; age 0–6 days; PMA 41–41.9 weeks), Tertiary Emergency Triage and Preoperative Stabilization (T_0: Hospital Days 1–2, June 16–17, 2025; age 7–8 days; PMA 42 weeks), Stage-1 Stamm Gastrostomy, Peripherally Inserted Central Catheter (PICC) Placement, and Total Parenteral Nutrition Initiation (T_1: Hospital Days 3–7, June 17–22, 2025; age 8–13 days; PMA 43 weeks), Trophic Gastrostomy Enteral Priming, Intravenous Albumin Replacement, and Pre-Thoracotomy Optimization (T_2: Hospital Days 8–15, June 23–30, 2025; age 14–21 days; PMA 44 weeks), Stage-2 Right Posterolateral Thoracotomy, Tracheoesophageal Fistula Ligation, Primary Esophageal Anastomosis, and Early Extubation (T_3: Hospital Days 16–18, July 1–3, 2025; age 22–24 days; PMA 45 weeks), Post-Thoracotomy Leukocytosis Surge, Carbapenem Escalation, and Gastrostomy Refeeding (T_4: Hospital Days 19–22, July 4–7, 2025; age 25–28 days; PMA 45.5 weeks), Complete Room-Air Liberation, Oral Feeding Transition, and Gastrostomy Decannulation (T_5: Hospital Days 23–24, July 8–9, 2025; age 29–30 days; PMA 46 weeks), Full Oral Nutritional Autonomy and Hospital Discharge (T_6: Hospital Days 25–27, July 10–12, 2025; age 31–33 days; PMA 46.5 weeks), and Outpatient Longitudinal Fenton Catch-Up Surveillance (T_7: July 14–August 11, 2025; age 35–63 days; PMA 47–50 weeks).

Comparative Framework

To contextualize single-subject longitudinal findings without statistical over-extrapolation, a dual internal-external comparative framework was implemented. Internally, each serial physiological, hematologic, biochemical, and anthropometric measurement across T_0 through T_7 was benchmarked against the patient's own baseline admission state (T_0) and birth anthropometry (T_{-1}), as well as against standardized neonatal reference intervals, including the Fenton sex-specific growth standards (22 to 50 weeks PMA) and neonatal hematologic, coagulation, electrolyte, and arterial blood gas norms.[32,33] Externally, the patient's clinical presentation, surgical staging interval, trans-anastomotic and gastrostomy feeding kinetics, postoperative complication profile, and anthropometric recovery velocity were systematically benchmarked against contemporary international multicenter EA/TEF registries, randomized controlled trials, and LMIC cohort studies published between 2021 and 2026.[13,17,21,22,29,30,32,33]

Biostatistical Analysis

For single-subject (n = 1) observational analytic studies, fictitious inferential hypothesis testing—including Student's t-tests, analysis of variance (ANOVA), Mann-Whitney U tests, chi-square (χ^2) tests, and fabricated p-values—was not used for within-patient comparisons. Instead, rigorous deterministic longitudinal metrics were computed across all serial observation epochs. Relative temporal change between baseline (T_0) and subsequent timepoints (T_n) was quantified using the Percentage Delta Change formula: $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Neonatal body proportionality at birth was evaluated via Rohrer's Ponderal Index: PI = [Birth Weight (g) × 100] / [Crown-Heel Length (cm)]^3, where PI < 2.32 g/cm^3 indicates fetal wasting and symmetric proportional reduction with BW < P_3, BL < P_{10}, and HC < P_{10} confirms symmetric IUGR. Hemodynamic perfusion was tracked via Mean Arterial Pressure: MAP = [SBP + 2(DBP)] / 3. Pulmonary gas exchange was quantified via the non-invasive pulse oximetric saturation-to-inspired oxygen ratio (SpO_2/FiO_2), the arterial oxygen tension-to-inspired oxygen ratio (PaO_2/FiO_2), and the Oxygenation Index: OI = [Mean Airway Pressure (cmH_2O) × FiO_2 × 100] / PaO_2 (mmHg). Systemic cellular inflammation was indexed via the immature-to-total neutrophil (I/T) ratio, the Neutrophil-to-Lymphocyte Ratio (NLR = [% band + % segmented neutrophils] / % lymphocytes), and the Systemic Immune-Inflammation Index (SII = [Platelet count ($\times 10^3/\mu L$) × NLR]). Parenteral carbohydrate delivery was standardized via the Glucose Infusion Rate: GIR (mg/kg/min) = [Dextrose concentration (%) × Infusion rate (mL/h)] / [6 × Body weight (kg)], while somatic recovery was quantified via absolute daily weight velocity (g/day = [$W_2 - W_1$] / Δt) and weight-indexed velocity (g/kg/day). Finally, causal relationships between anatomical/clinical insults, therapeutic interventions, and intercurrent complications were evaluated using the nine-criterion Bradford Hill epidemiological causality matrix, the World Health Organization–Uppsala Monitoring Centre (WHO-UMC) causality taxonomy, and the Naranjo Adverse Drug Reaction (ADR) probability scale.

3. Results

Perinatal Baseline, Symmetric IUGR Phenotype, and Pre-Referral Catabolic Trajectory

The index patient (anonymized code: Patient MRA) was a full-term male neonate born on June 9, 2025, at 10:00 WIB to a 40-year-old multigravida mother (G4P2A1H2) at 41 weeks and 0 days of gestation via spontaneous vaginal delivery assisted by a community midwife. Antenatal care had been restricted to routine visits at a primary healthcare clinic and midwife practice without evaluation by an obstetrician-gynecologist; obstetric ultrasonography had been performed only once at a primary facility at an unspecified gestational age, failing to identify fetal polyhydramnios or an upper mediastinal esophageal pouch sign. Maternal prenatal history was notable for persistent, partially treated yellowish, clumpy, pruritic, and malodorous vaginal discharge beginning in the fourth month of gestation and continuing until delivery, compounded by uninvestigated third-trimester maternal dysuria. No family history of congenital foregut or cardiovascular anomalies was present. The father (Mr. DA, aged 42 years, elementary school education) worked as a traditional coastal fisherman earning IDR 2,000,000 to 3,000,000 per month, and the mother (Ms. D, elementary school education) was a homemaker; the patient was the youngest of three living siblings residing in a non-permanent dwelling with adequate sanitation.

At birth (Epoch T_{-1}), the neonate cried immediately and vigorously, achieving Apgar scores of 8 at 1 minute and 9 at 5 minutes with clear amniotic fluid and no peripartum cyanosis. Routine intramuscular vitamin K prophylaxis and the birth dose of hepatitis B vaccine were administered. Despite a birth weight of 2,600 g—which places the infant within the nominal normal birth weight category (≥ 2,500 g) and Spitz Group I threshold (≥ 1,500 g)—plotting of birth anthropometry onto the sex-specific Fenton growth chart at 41 completed gestational weeks revealed severe, proportional intrauterine growth restriction: birth weight of 2,600 g fell below the 3rd percentile ($P_3 \approx 2{,}750$ g), crown-heel body length of 49.0 cm fell below the 10th percentile ($P_{10} \approx 49.8$ cm), and occipitofrontal head circumference of 34.0 cm fell below the 10th percentile ($P_{10} \approx 34.2$ cm), yielding a Rohrer's Ponderal Index of PI = [2,600 × 100] / 49.0^3 = 2.21 g/cm^3 and confirming a diagnosis of Small for Gestational Age (SGA) with Symmetric Intrauterine Growth

Restriction (IUGR). Clinical symptoms of foregut discontinuity emerged immediately after birth, characterized by continuous frothy oral mucus discharge, followed at 8 hours of age by recurrent non-bilious vomiting and choking after every breastfeeding attempt. By day 2 of life (5 days prior to tertiary referral), the neonate developed progressive tachypnea, copious viscid airway secretions, and cephalo-caudal jaundice extending to the lower extremities (Kramer Grade III). Initial evaluation at a secondary facility (Dr. M. Zain Painan Regional Hospital) documented failure to advance a nasogastric catheter, suspected aspiration pneumonia, and suspected right pneumothorax; the infant received intravenous Dextrose 12.5% (12 drops/min), parenteral amino acids (Aminoinfant 10% at 4.3 mL/h), intravenous ranitidine (2.2 mg twice daily for 5 days), intravenous ampicillin (125 mg twice daily), and intravenous gentamicin (10 mg once daily) before transfer. Across the 7-day pre-referral interval, uncompensated feeding failure and hypercatabolic respiratory distress precipitated a progressive weight decline from 2,600 g at birth to 2,380 g at referral (−8.46%) and 2,200 g upon tertiary NICU admission (Δ = −400 g; Δ% = −15.38%), with the complete perinatal, obstetric, socioeconomic, and baseline emergency triage parameters detailed in Table 1.

Table 1. Perinatal, Obstetric, Socioeconomic, and Baseline Emergency Triage Ascertainment of the Index Neonate With Gross Type C Esophageal Atresia and Symmetric Intrauterine Growth Restriction.

Clinical Domain	Parameter / Variable	Observed Patient Value	Reference Standard / Threshold	Analytical & Prognostic Classification
Demographics & Timeline	Patient Code / Sex / Gestational Age	Patient MRA / Male / 41 wk 0 d (Term)	Term gestation: 37–41 wk 6 d	Full-term male neonate; born June 9, 2025 (10:00 WIB)
Admission & Follow-Up	Age at Referral / Inpatient LOS / Follow-Up	7 days (PMA 42 wk) / 27–28 days (Jun 16–Jul 12, 2025) / 50 wk PMA (Aug 11, 2025)	Optimal surgical referral: < 24–48 h of birth	Delayed 7-day postnatal referral via Dr. M. Zain Painan Hospital; 63-day longitudinal tracking
Obstetric & Antenatal History	Gravidity-Parity / Delivery / ANC Surveillance	G4P2A1H2 mother (40–41 yr) / Spontaneous vaginal (midwife) / 1 primary-care ultrasound	≥ 4 OB-GYN ANC visits; targeted 2nd-trimester fetal anatomy scan	Suboptimal prenatal screening missed fetal polyhydramnios and proximal esophageal pouch sign
Maternal Perinatal Morbidity	Maternal Genitourinary Infections & Blood Group	Persistent 2nd-trimester yellowish pruritic vaginitis + untreated 3rd-trimester dysuria; Blood type A	Sterile maternal genitourinary tract during gestation	Chronic maternal urogenital inflammation predisposing to uteroplacental insufficiency and symmetric IUGR
Socioeconomic Determinants	Parental Age, Education & Occupation	Father Mr. DA (42 yr, elementary, fisherman; IDR 2–3 million/mo); Mother Ms. D (40–41 yr, elementary, housewife)	Regional minimum household income & secondary education	Youngest of 3 siblings in coastal fishing household; delayed tertiary diagnostic recognition
Perinatal Resuscitation	Apgar Score / Amniotic Fluid / Prophylaxis	8 at 1 min and 9 at 5 min; clear amniotic fluid; IM Vitamin K + Hepatitis B birth dose given	Apgar ≥ 7 at 1 and 5 min; routine neonatal prophylaxis	Vigorous immediate postnatal transition masking underlying foregut discontinuity
Baseline Anthropometry (Birth vs. Admission)	Birth Weight / Admission Weight / Length / Head Circumference / Ponderal Index	BW: 2,600 g (< P_3) → Admission: 2,200 g (Δ = −400 g; −15.38%); BL: 49.0 cm (< P_{10}); HC: 34.0 cm (< P_{10}); PI = 2.21 g/cm^3	Fenton 41-wk boys P_{10}–P_{90}: BW 2,950–4,150 g; BL 50.0–54.5 cm; HC 34.2–37.5 cm; PI ≥ 2.32 g/cm^3	Normal birth weight category (≥ 2,500 g) but Small for Gestational Age (SGA) with Symmetric IUGR and acute −15.38% catabolic weight loss
Aerodigestive & Triage Presentation (T_0)	Orogastric Tube Arrest / Oxygenation / Vital Signs / Kramer Jaundice	OGT arrested at 10.0 cm from gum line; Room-air SpO_2 88–90% (SpO_2/FiO_2 = 419.0–428.6) → 94–97% on CPAP PEEP 6, FiO_2 25%; HR 140 bpm, RR 45/min, Temp 37.3→38.0 °C; Kramer Grade III	OGT passage to stomach (18–22 cm); SpO_2 ≥ 95% on room air; RR 30–40/min; Temp 36.5–37.5 °C	Gross Type C / Vogt Type 3B EA/TEF complicated by bilateral aspiration bronchopneumonia and Kramer III neonatal hyperbilirubinemia
Cardiopulmonary Risk Stratification	Transthoracic Echocardiography (June 20, 2025) & Risk Scores	Situs solitus, Patent Foramen Ovale (PFO), good LV/RV systolic function, left aortic arch	Spitz Group I: BW ≥ 1,500 g without major congenital heart disease	Spitz Group I, Waterston Group B, Montreal Class I (favorable survival reserve modified by symmetric IUGR)

Abbreviations: ANC, antenatal care; BL, body length; BW, birth weight; CPAP, continuous positive airway pressure; EA/TEF, esophageal atresia with tracheoesophageal fistula; FiO_2, fraction of inspired oxygen; HC, head circumference; HR, heart rate; IDR, Indonesian Rupiah; IM, intramuscular; IUGR, intrauterine growth restriction; LOS, length of stay; LV/RV, left/right ventricle; OB-GYN, obstetrician-gynecologist; OGT, orogastric tube; PEEP, positive end-expiratory pressure; PFO, patent foramen ovale; PI, ponderal index ([weight in g × 100] / [length in cm]^3); PMA, postmenstrual age; RR, respiratory rate; SGA, small for gestational age; SpO_2, peripheral oxygen saturation.

Emergency Triage Stabilization, Serial Babygram Imaging, and Stage-1 to Stage-2 Surgical Execution

Upon arrival at the Emergency Department and Level III NICU of Dr. M. Djamil General Hospital on June 16, 2025 (Epoch T_0, Hospital Day 1; chronological age 7 days, PMA 42 weeks), the neonate appeared severely ill, irritable but consolable, with thin subcutaneous adipose tissue, generalized Kramer Grade III icterus, nasal flaring, and prominent epigastric and intercostal chest wall retractions. Baseline vital signs demonstrated a heart rate of 140 beats/min (fluctuating up to 160 beats/min during agitation), a respiratory rate of 45 breaths/min, an axillary temperature of 37.3 °C at triage that peaked at 38.0 °C on Hospital Day 2, and a capillary refill time of less than 2 to 3 seconds. On ambient room air (FiO_2 21%), peripheral pulse oximetry revealed significant arterial hypoxemia with SpO_2 of 88% to 90% (SpO_2/FiO_2 ratio = 419.0 to 428.6). Immediate application of non-invasive nasal Continuous Positive Airway Pressure (CPAP) at a Positive End-Expiratory Pressure (PEEP) of 6 cmH_2O and FiO_2 of 25% restored SpO_2 to 94%–97% (SpO_2/FiO_2 = 382.0), and settings were subsequently titrated to PEEP 7 cmH_2O and FiO_2 30% (SpO_2/FiO_2 = 318.3) to stabilize alveolar recruitment. Attempted passage of an orogastric catheter arrested reproducibly at 10.0 cm from the upper alveolar gum line, where continuous low-pressure proximal pouch suction was instituted. A diagnostic anteroposterior thoracic-abdominal babygram obtained on June 16, 2025 confirmed the radiopaque catheter coiled within a dilated, air-filled blind proximal esophageal pouch in the superior mediastinum, bilateral perihilar and lower-zone parenchymal pulmonary infiltrates consistent with aspiration bronchopneumonia, and positive intestinal gas distribution reaching the distal pelvis, establishing a definitive diagnosis of Gross Type C (Vogt Type 3B) EA/TEF. Following surgical optimization on Hospital Day 2 (June 17, 2025), the pediatric surgery team performed a Stage-1 decompressive and feeding Stamm gastrostomy with minimal blood loss, while a Peripherally Inserted Central Catheter (PICC) was placed via the left subclavian vein. Serial post-gastrostomy babygram (June 18, 2025) and chest radiography (June 19, 2025) verified proper subdiaphragmatic gastrostomy tube positioning, persistent bilateral pneumonic infiltrates, and stable intestinal gas distribution, while transthoracic echocardiography on June 20, 2025 demonstrated situs solitus, a hemodynamically

non-restrictive patent foramen ovale (PFO), preserved left and right ventricular systolic contractility, and a left aortic arch (confirming Spitz Group I status); these serial diagnostic and post-gastrostomy thoracic-abdominal babygrams alongside the longitudinal oxygenation trajectory are illustrated in Figure 1.

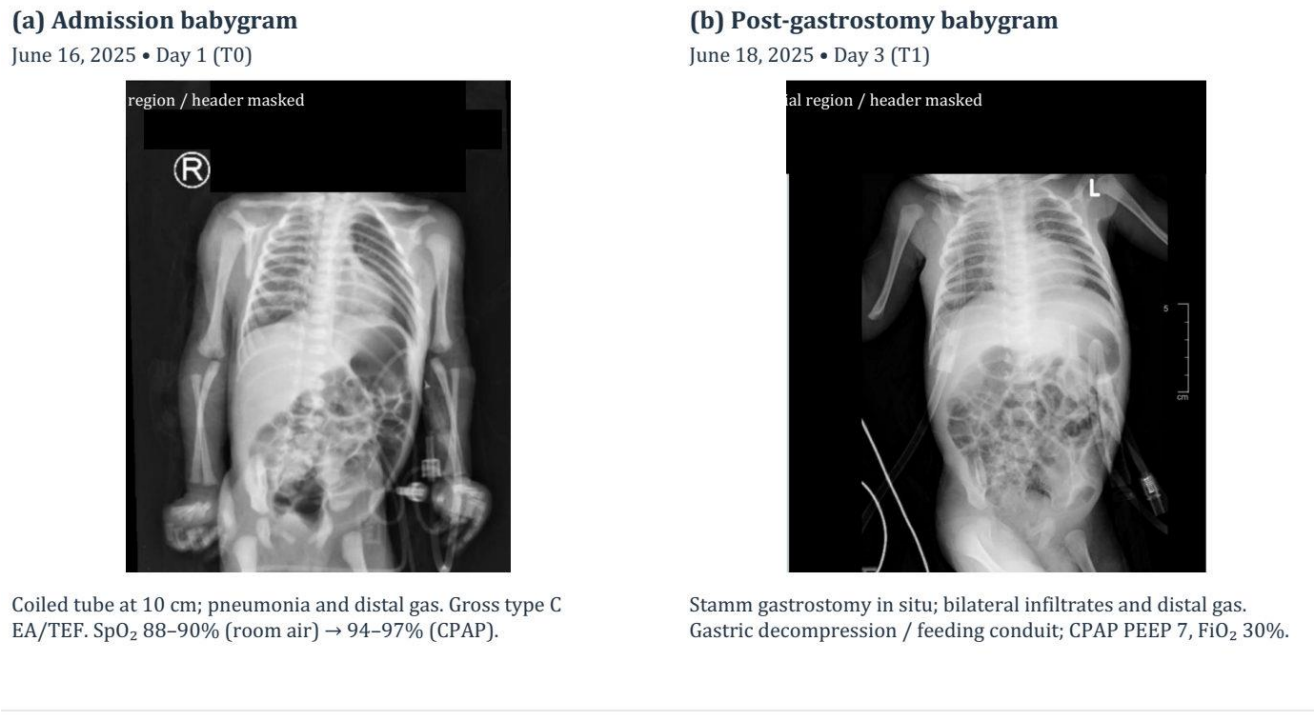


Figure 1. Serial Diagnostic and Preoperative Thoracic-Abdominal Babygrams With Mandatory Black Anonymization Bars (ICMJE / Helsinki Compliant) and Longitudinal Cardiorespiratory Oxygenation Trajectory Across Hospitalization. (a) Baseline admission babygram on June 16, 2025 (Hospital Day 1, T_0) showing the coiled radiopaque orogastric tube arrested in the dilated blind proximal esophageal pouch (10 cm from the gum line), bilateral aspiration bronchopneumonia infiltrates, and distal intestinal gas confirming Gross Type C EA/TEF. (b) Post-gastrostomy babygram on June 18, 2025 (Hospital Day 3, T_1) confirming subdiaphragmatic Stamm gastrostomy tube placement and distal bowel gas. (c) Preoperative chest radiograph on June 19, 2025 (Hospital Day 4, T_1) showing bilateral parenchymal infiltrates and the newly inserted left subclavian PICC line. (d) Longitudinal SpO_2/FiO_2 ratio and inspired oxygen (FiO_2) trajectory demonstrating stabilization on nasal CPAP, < 24-hour extubation after Day 17 right thoracotomy (PaO_2/FiO_2 = 217.1 mmHg, OI = 3.68), and complete room-air liberation on Day 23.

Following 15 days of preoperative cardiorespiratory stabilization, central parenteral nutrition, intravenous albumin supplementation, and trophic gastrostomy priming across Epochs T_1 and T_2, the neonate's respiratory support requirement decreased to nasal CPAP PEEP 5 cmH_{2O} on room air (FiO_2 21%; SpO_2 94%–97%; SpO_2/FiO_2 = 454.8) by Hospital Day 16 (July 1, 2025). On Hospital Day 17 (July 2, 2025; chronological age 23 days, PMA 45 weeks, body weight 2,700–2,750 g), the patient underwent Stage-2 definitive surgical repair via a right posterolateral thoracotomy. Intraoperative exploration identified the distal tracheoesophageal fistula entering the posterior membranous trachea; the fistulous tract and punctum were meticulously isolated using traction sutures, transfixed, ligated, and divided, followed by mobilization of the hypertrophied blind proximal esophageal pouch and completion of a tension-calibrated, single-layer primary end-to-end esophago-esophageal anastomosis over a trans-anastomotic nasogastric catheter, with placement of a right intercostal thoracostomy drainage tube. Total operative

duration was 2.5 hours, intraoperative intravenous fluid administration was 42.0 mL of 5% Dextrose in 0.45% normal saline (D5 ½ NS) plus anesthetic medications, estimated blood loss was strictly minimal at 10.0 mL (3.64 mL/kg), intraoperative urine output was 10.0 mL (1.48 mL/kg/h), and net intraoperative fluid balance was +14.7 mL. Postoperative chest radiography on July 2, 2025 confirmed bilateral pulmonary hyperinflation with resolving aspiration infiltrates, a normal cardiothoracic silhouette, the gastrostomy button in the left upper quadrant, right chest tube drainage, and the left subclavian PICC tip projecting precisely at the superior vena cava–right atrial junction, with the intraoperative thoracotomy findings, postoperative chest radiograph, and hemodynamic stability profile illustrated in Figure 2.

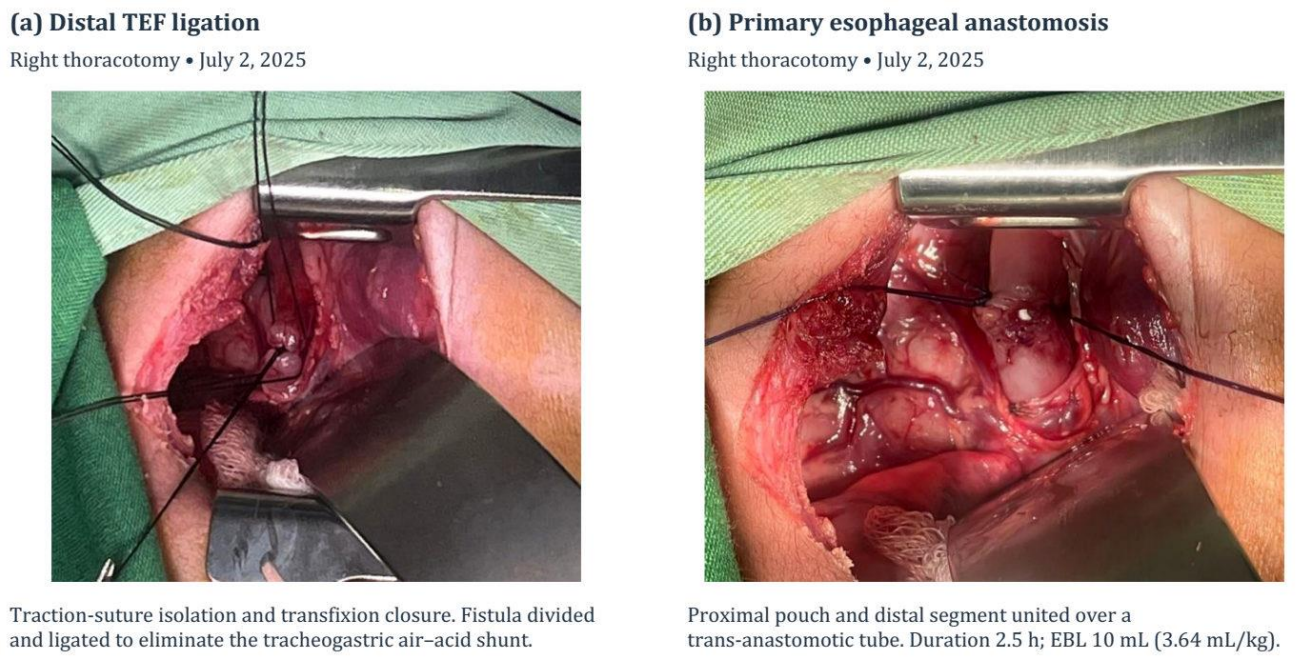


Figure 2. Intraoperative Right Posterolateral Thoracotomy Documentation (Fistula Ligation and Primary Esophageal Anastomosis), Post-Thoracotomy Chest Radiograph, and Longitudinal Hemodynamic & Intraoperative Fluid Balance Trajectory. (a) Intraoperative photograph on July 2, 2025 (Hospital Day 17, T_3) demonstrating dissection, traction-suture isolation, and transfixion ligation of the distal tracheoesophageal fistula punctum. (b) Completed tension-calibrated single-layer primary end-to-end esophageal anastomosis uniting the proximal pouch and distal esophageal segment over a trans-anastomotic catheter (operative duration 2.5 h, estimated blood loss 10.0 mL [3.64 mL/kg], urine output 1.48 mL/kg/h, net fluid balance +14.7 mL). (c) Immediate post-thoracotomy chest radiograph on July 2, 2025 with permanent black anonymization bar at the superior border, confirming left subclavian PICC tip projection into the right atrium, gastrostomy button in situ, and bilateral pulmonary hyperinflation. (d) Longitudinal systolic, diastolic, and mean arterial pressure (MAP ≥ 50 mmHg) and heart rate stability across T_0 to T_6.

Longitudinal Cardiorespiratory Liberation and Staged Parenteral-Gastrostomy-Oral Nutritional Recovery

Analysis of the longitudinal cardiorespiratory and hemodynamic trajectory across all eight observation epochs (T_0 to T_7) demonstrated tight physiological coupling between staged aerodigestive decompression, anastomotic repair, and ventilatory liberation. Immediately following transfer from the operating theater on July 2, 2025 at 18:00 WIB (Epoch T_3), the neonate was supported via an uncuffed endotracheal tube (internal diameter 3.0 mm, lip depth 8.5 cm) connected to Synchronized Intermittent Mandatory Ventilation (SIMV: Peak Inspiratory Pressure [PIP] 14 cmH_2O, respiratory rate 40 breaths/min, PEEP 6 cmH_2O, inspiratory time 0.4 s, FiO_2 35%), maintaining SpO_2 of 93%–95% (SpO_2/FiO_2 = 268.6) and a transient post-anesthetic tachycardia of 153 beats/min. Postoperative arterial blood gas analysis on SIMV confirmed well-compensated normocapnic ventilation and alveolar oxygenation, with pH 7.42, $PaCO_2$ 36.8 mmHg, PaO_2 76.0 mmHg (PaO_2/FiO_2 ratio = 217.1 mmHg; estimated mean airway pressure ≈ 8.0 cmH_2O, yielding a favorable Oxygenation Index of OI = 3.68), HCO_3^- 23.0 mmol/L, base excess −1.0 mmol/L, and SaO_2 95.0%. On Postoperative Day 1 (July 3, 2025), a restless episode caused the trans-anastomotic nasogastric catheter to fold within the oropharynx; in accordance with pediatric surgical directives to avoid blind re-advancement across the fresh anastomosis, the displaced catheter was removed while the pre-existing Stamm gastrostomy tube and right chest drain remained in situ. The infant was successfully extubated in the afternoon of July 3, 2025 (less than 24 hours after thoracotomy) to nasal CPAP (PEEP 6 cmH_2O, FiO_2 30%), which was rapidly weaned to PEEP 5 cmH_2O and FiO_2 21% (SpO_2/FiO_2 = 454.8) from July 4 to July 7, 2025 (Epoch T_4). Transition to unassisted breathing in room air (SpO_2 95%–97%; SpO_2/FiO_2 = 457.1) was achieved on Hospital Day 23 (July 8, 2025; Epoch T_5), with normotensive systemic perfusion maintained throughout hospitalization (MAP 50.0 to 66.0 mmHg) without vasopressor or inotropic support; this complete eight-epoch longitudinal cardiorespiratory, surgical, hemodynamic, and enteral-parenteral trajectory is detailed in Table 2.

Table 2. Longitudinal Cardiorespiratory, Surgical, Hemodynamic, and Aerodigestive Trajectory Across Seven Inpatient Epochs (T_0 to T_6) and Outpatient Follow-Up (T_7).

Epoch & Hospital Days (Dates)	Respiratory Support & Oxygenation Metrics	Hemodynamic & Thermal Parameters	Surgical & Airway Interventions	Enteral / Parenteral Route & Somatic Weight (Δ% vs. Nadir)
T_0: Admission & Stabilization Days 1–2 (Jun 16–17, 2025) Age 7–8 d (PMA 42 wk)	Room air SpO_2 88–90% (S/F = 419.0–428.6); initiated nasal CPAP PEEP 6→7 cmH_2O, FiO_2 25→30%; SpO_2 94–97% (S/F = 382.0→318.3); RR 45/min; retractions (+)	HR 140 bpm (range 135–160 bpm); CRT < 2–3 s; Temp 37.3 °C at triage → febrile peak 38.0 °C (Day 2); Kramer III jaundice	Continuous low-pressure proximal pouch suction via OGT/Replogle tube (arrested at 10 cm); pediatric surgery clearance for Stage-1 gastrostomy	Strict NPO; IVFD PG2 starter 330 mL/d (13.7 mL/h; 150 mL/kg/d); RBG 89→135 mg/dL. Weight nadir: 2,200 g (−15.38% vs. BW)
T_1: Stage-1 Gastrostomy & TPN Days 3–7 (Jun 17–22, 2025) Age 8–13 d (PMA 43 wk)	Nasal CPAP PEEP 7 cmH_2O, FiO_2 30%; SpO_2 94–97% (S/F = 318.3); vesicular breath sounds, no rales/wheezing; CXR (Jun 18–19): bilateral pneumonia	BP 75/48 mmHg (MAP 57.0 mmHg; monitor 55 mmHg); HR 130–145 bpm; CRT < 3 s; afebrile; Echo (Jun 20): PFO, normal LV/RV, left arch	Stage-1 Stamm gastrostomy performed (Jun 17, minimal bleeding); left subclavian PICC line placed to right atrium for central TPN	NPO for post-gastrostomy healing; TPN (150 mL/kg/d, GIR 8.2 mg/kg/min, 84 kcal/kg/d, N/NPC 1:152, RBG 91 mg/dL). Weight: 2,485→2,600 g (+12.95% to +18.18%)
T_2: Trophic Gastrostomy Priming Days 8–15 (Jun 23–30, 2025) Age 14–21 d (PMA 44 wk)	Nasal CPAP PEEP 7 cmH_2O, FiO_2 30%; SpO_2 94–97% (S/F = 318.3); venous/capillary BGA (Jun 30): pH 7.41, pCO_2 33.3, pO_2 34.0 mmHg, HCO_3^- 21.6, BE −3.0, SaO_2 67%	BP 75/42 mmHg (MAP 53.0 mmHg; monitor 54 mmHg); HR 130–147 bpm; CRT < 3 s; afebrile; Blood culture I (Jun 23): sterile	Continuous proximal pouch suction maintained; IV 20% Albumin transfusion (1.0 g/kg × 2 doses) for serum albumin 2.9 g/dL (Jun 24)	Trophic gastrostomy feeds: Pepti/EBM 8×2–3 mL (16–24 mL/d) + PICC TPN (84 kcal/kg/d, RBG 89 mg/dL). Weight: 2,645→2,700 g (+20.23% to +22.73%)
T_3: Right Thoracotomy & Repair Days 16–18 (Jul 1–3, 2025) Age 22–24 d (PMA 45 wk)	Jul 1: CPAP PEEP 5, FiO_2 21% (S/F = 454.8). Jul 2 (18:00 post-op): ETT No. 3.0 (8.5 cm), SIMV (PIP 14, RR 40, PEEP 6, Ti 0.4 s, FiO_2 35%), SpO_2 93–95% (S/F = 268.6); ABG: pH 7.42, $PaCO_2$ 36.8, PaO_2 76 mmHg (P/F = 217.1, OI = 3.68), SaO_2 95%. Jul 3: Extubated < 24 h to CPAP PEEP 6→5, FiO_2 30→21%	Jul 2 post-op HR 153 bpm. Jul 3: BP 70/40 mmHg (MAP 50.0 mmHg), HR 130–145 bpm, CRT < 3 s, afebrile. Intra-op (2.5 h): Intake 42 mL, EBL 10 mL (3.64 mL/kg), Urine 10 mL (1.48 mL/kg/h), Net +14.7 mL	Stage-2 right posterolateral thoracotomy (Jul 2): suture ligation/division of distal TEF punctum + primary end-to-end esophageal anastomosis + right chest tube; folded NGT repositioned Jul 3	Strict 48-h post-anastomotic fasting (Jul 2–4); intensified TPN (Dextrose N15% GIR 10.0 mg/kg/min, Protein 3.0 g/kg/d, Lipid 3.0 g/kg/d, RBG 103 mg/dL). Weight: 2,720 g (+23.64%)
T_4: POD 3 Surge & Refeeding Days 19–22 (Jul 4–7, 2025) Age 25–28 d (PMA 45.5 wk)	Nasal CPAP PEEP 5 cmH_2O, FiO_2 21%; SpO_2 94–97% (S/F = 454.8); bilateral vesicular breath sounds, no rales/wheezing; chest tube & gastrostomy in situ	BP 70/40 mmHg (MAP 50.0 mmHg); HR 130–145 bpm; CRT < 3 s; afebrile; POD 3 (Jul 5) WBC surge to 26,920/μL (+108.68%), I/T 0.02; Blood cultures I–II sterile	Escalated from Cefoperazone-Sulbactam (stopped Day 3) to IV Meropenem (3×110 mg) + Amikacin (3×20 mg) on Jul 5	Gastrostomy refeeding resumed at 48 h post-op: Pepti/EBM 8×5 mL (40 mL/d; 14.5 mL/kg/d) + parenteral nutrition. Weight: 2,750 g (+25.00%)
T_5: Oral Transition & GT Removal Days 23–24 (Jul 8–9, 2025) Age 29–30 d (PMA 46 wk)	Spontaneous unassisted room-air breathing (FiO_2 21%); SpO_2 95–97% (S/F = 457.1); no desaturation or retractions	BP 78/60 mmHg (MAP 66.0 mmHg; monitor 63 mmHg); HR 140–145 bpm; CRT < 3 s; afebrile; miliaria rubra & diaper rash (Gram stain negative)	Transitioned to oral feeding; gastrostomy tube removed on Jul 9, 2025; transferred to Level II NICU; topical 1% hydrocortisone BD	Full oral feeding: EBM/formula 4×30 mL + 4×35 mL (260 mL/d; 91.2 mL/kg/d; 150 mL/kg/d total fluids). Weight: 2,850 g (+29.55%)
T_6: Oral Autonomy & Discharge Days 25–27 (Jul 10–12, 2025) Age 31–33 d (PMA 46.5 wk)	Spontaneous room-air breathing (FiO_2 21%); SpO_2 94–97% (S/F = 454.8); complete resolution of pneumonia	BP 67/42 mmHg (MAP 50.3 mmHg); HR 134–145 bpm; CRT < 3 s; afebrile; cutaneous lesions resolved	Completed 7-day IV Meropenem course (Jul 5–12); discharged home in stable condition on Jul 12, 2025	Exclusive oral EBM/formula 8×35–40 mL (280–320 mL/d; 95–108 mL/kg/d; 110–125 kcal/kg/d). Discharge Weight: 2,955 g (+34.32% vs. nadir; +29.04 g/d)
T_7: Outpatient Fenton Catch-Up Days 35–63 (Jul 14–Aug 11, 2025) Age 35–63 d (PMA 47–50 wk)	Unassisted room-air breathing; no aspiration episodes, choking, or stridor during 5-week outpatient surveillance	Normotensive, afebrile, active motor tone; scheduled 3-month repeat cardiology echocardiography	Outpatient multidisciplinary surveillance (pediatric surgery, nutrition, pulmonology, gastroenterology)	Progressive ad libitum oral breast milk / formula feeding. Serial Fenton weights: Jul 14: 2,780 g → Jul 21: 3,120 g → Jul 28: 3,200 g → Aug 4: 3,320 g → Aug 11 (50 wk PMA): 3,700 g (+68.18% vs. nadir; +42.31% vs. BW)

Abbreviations: ABG, arterial blood gas; BE, base excess; BGA, blood gas analysis; BP, blood pressure; BW, birth weight; CPAP, continuous positive airway pressure; CRT, capillary refill time; CXR, chest radiograph; EBL, estimated blood loss; EBM, expressed breast milk; ETT, endo tracheal tube; FiO_2, fraction of inspired oxygen; GIR, glucose infusion rate; GT, gastrostomy tube; HR, heart rate; I/T, immature-to-total neutrophil ratio; MAP, mean arterial pressure; NGT, nasogastric tube; N/NPC, nitrogen to non-protein calorie ratio; NPO, nil per os; OGT, orogastric tube; OI, oxygenation index; P/F, PaO_2/FiO_2 ratio; PICC, peripherally inserted central catheter; PIP, peak inspiratory pressure; PMA, postmenstrual age; POD, postoperative day; RBG, random blood glucose; S/F, SpO_2/FiO_2 ratio; SIMV, synchronized intermittent mandatory ventilation; TPN, total parenteral nutrition; WBC, white blood cell count.

Overcoming the profound nutritional deficit resulting from baseline symmetric IUGR (birth weight 2,600 g < P_3) and the acute 400 g pre-referral catabolic weight loss (admission nadir 2,200 g; Δ% = −15.38%) required a staged three-phase nutritional rehabilitation protocol tracked across the Fenton growth chart. During Phase I (Epochs T_0–T_1, June 16–22, 2025), following initial fluid resuscitation with parenteral starter solution PG2 (330 mL/day = 13.7 mL/h; 150 mL/kg/day) on Days 1–2, central TPN was delivered via the left subclavian PICC line using Dextrose N12.5% at 9.5 mL/h (Glucose Infusion Rate [GIR] = 8.2 mg/kg/min), crystalline amino acids (Aminoinfant 10% at 3.0 mL/h ≈ 3.0

g/kg/day), and composite four-oil intravenous lipid emulsion (SMOFlipid 20% at 1.2 mL/h ≈ 2.3–3.0 g/kg/day), providing 84 kcal/kg/day at a nitrogen-to-non-protein calorie ratio (N/NPC) of 1:152 and maintaining euglycemia (random blood glucose 89–91 mg/dL). This central parenteral regimen arrested tissue catabolism and increased body weight from 2,200 g to 2,485 g by Day 5 (+12.95% above nadir) and 2,600 g by Day 8 (June 23, 2025; regaining birth weight). During Phase II (Epochs T_2–T_4, June 23–July 7, 2025), trophic enteral priming via the Stamm gastrostomy was initiated using extensively hydrolyzed whey peptide formula (Pepti formula) alternating with expressed breast milk (EBM) at 8 × 2–3 mL/day (16–24 mL/day) alongside ongoing TPN, advancing weight to 2,645 g on June 24 and 2,700 g by June 30 (+22.73% above nadir). Following a mandatory 48-hour post-thoracotomy fasting window (July 2–4) supported by intensified Dextrose N15% TPN (GIR 10.0 mg/kg/min, protein 3.0 g/kg/day, lipid 3.0 g/kg/day; RBG 103 mg/dL), gastrostomy refeeding was escalated to 8 × 5 mL/day (40 mL/day; 14.5 mL/kg/day), bringing weight to 2,750 g on July 5–7 (+25.00% above nadir). During Phase III (Epochs T_5–T_7, July 8–August 11, 2025), intact esophageal anastomotic transit permitted transition to full oral feeding on July 8–9 at 4 × 30 mL plus 4 × 35 mL (260 mL/day; 91.2 mL/kg/day), enabling bedside removal of the gastrostomy tube on July 9, 2025 (weight 2,850 g; +29.55% above nadir). By discharge on July 12, 2025, exclusive oral intake reached 8 × 35–40 mL/day (280–320 mL/day; 95–108 mL/kg/day; 110–125 kcal/kg/day) and body weight reached 2,955 g (Δ = +755 g or +34.32% above admission nadir; Δ = +355 g or +13.65% above birth weight; net inpatient post-nadir growth velocity = +29.04 g/day or +11.67 g/kg/day over 26 days). Subsequent weekly outpatient Fenton growth plotting confirmed sustained somatic catch-up to 2,780 g (July 14, 46 wk PMA), 3,120 g (July 21, 47 wk PMA), 3,200 g (July 28, 48 wk PMA), 3,320 g (August 4, 49 wk PMA), and 3,700 g by August 11, 2025 at 50 weeks PMA (Δ = +1,500 g or +68.18% above admission nadir; Δ = +1,100 g or +42.31% above birth weight), with the clinical Fenton growth chart and 63-day somatic weight and enteral volume escalation trajectory illustrated in Figure 3.

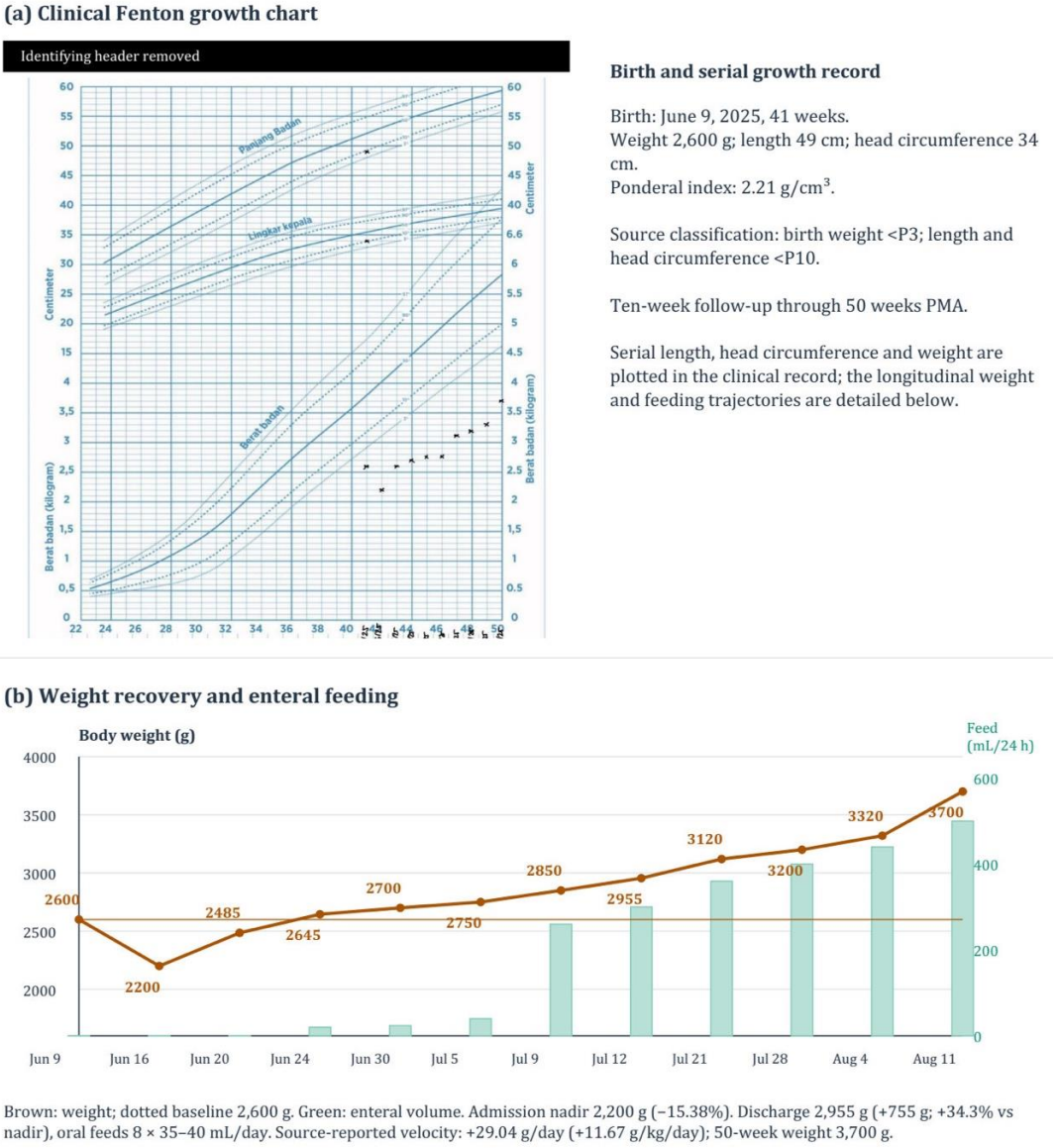


Figure 3. Longitudinal Fenton Preterm/Term Growth Chart With Mandatory Black Anonymization Bar (ICMJE Compliant) and Quantitative 63-Day Somatic Weight Recovery & Staged Parenteral-Gastrostomy-Oral Nutritional Escalation. (a) Clinical Fenton growth chart (boys, 22–50 weeks PMA) with permanent black bar masking the handwritten patient name, documenting baseline symmetric IUGR at 41 weeks gestation (birth weight 2,600 g [< P_3], body length 49.0 cm [< P_{10}], head circumference 34.0 cm [< P_{10}], Ponderal Index 2.21 g/cm³) and 10 serial weekly weight plots through 50 weeks PMA. (b) Quantitative 63-day weight trajectory and enteral volume escalation showing recovery from the 2,200 g admission nadir (−15.38% vs. birth weight) via PICC TPN (GIR 8.2→10.0 mg/kg/min, SMOFlipid 20% + Aminoinfant 10%), IV albumin replacement (2 × 1.0 g/kg), and trophic gastrostomy priming (16–40 mL/day) to 2,700 g pre-thoracotomy, gastrostomy removal on July 9 (2,850 g), hospital discharge on July 12 at 2,955 g (+34.32% above nadir; +29.04 g/day), and 50-week PMA outpatient catch-up to 3,700 g (+68.18%).

Serial Hematologic-Inflammatory, Coagulation, Hepatic-Metabolic, and Carbapenem Stewardship Kinetics

Serial hematologic-inflammatory, coagulation, hepatic-bilirubin, and metabolic laboratory panels across the five inpatient sampling timepoints (June 16, June 24, June 30, July 2, and July 5, 2025) revealed distinct pathophysiological phases corresponding to pre-referral starvation, preoperative optimization, and post-thoracotomy tissue stress. At admission on June 16, 2025 (T_0), the neonate exhibited mild neonatal anemia with hemoglobin of 14.6 g/dL (reference 16.5–21.5 g/dL) and hematocrit of 45.0% (reference 48.0%–68.0%), a normal erythrocyte count of 4.56 × 10^6/mm^3, a leukocyte count of 12,900/mm^3, and a platelet count of 421,000/mm^3. Coagulation screening demonstrated prolongation of both the extrinsic and intrinsic pathways, with Prothrombin Time (PT) of 12.1 seconds (1.1 × control) and Activated Partial Thromboplastin Time (APTT) of 31.0 seconds (1.2 × control), attributable to subclinical vitamin K depletion from 7 days of zero enteral intake and impaired hepatic synthesis. Concurrently, serum bilirubin profiling confirmed Kramer Grade III mixed (predominantly unconjugated) hyperbilirubinemia, with total bilirubin of 4.5 mg/dL, indirect bilirubin of 3.9 mg/dL, and direct bilirubin of 0.6 mg/dL (direct fraction 13.3%), alongside mild hypokalemia (K^+ 4.0 mmol/L) and hyperchloremia (Cl^- 113 mmol/L; Na^+ 142 mmol/L). By Hospital Day 9 (June 24, 2025; T_{2a}), parenteral vitamin K and TPN support completely normalized coagulation parameters to PT 10.6 seconds (Δ% = −12.40%) and APTT 25.9 seconds (Δ% = −16.45%) and corrected serum chloride to 96 mmol/L (Δ% = −15.04%; K^+ 3.6 mmol/L, Ca^{2+} 8.9 mg/dL), but uncovered mild dilutional/redistributive hyponatremia (Na^+ 134 mmol/L; Δ% = −5.63%) and clinically significant hypoalbuminemia of 2.9 g/dL. Administration of two targeted intravenous infusions of 20% human albumin (1.0 g/kg/dose) combined with 3.0 g/kg/day parenteral amino acids restored oncotic homeostasis and prevented anastomotic or peripheral tissue edema prior to surgery; the complete serial hematologic-inflammatory, coagulation, hepatic-bilirubin, electrolyte, and blood gas kinetics across all five inpatient sampling timepoints are detailed in Table 3.

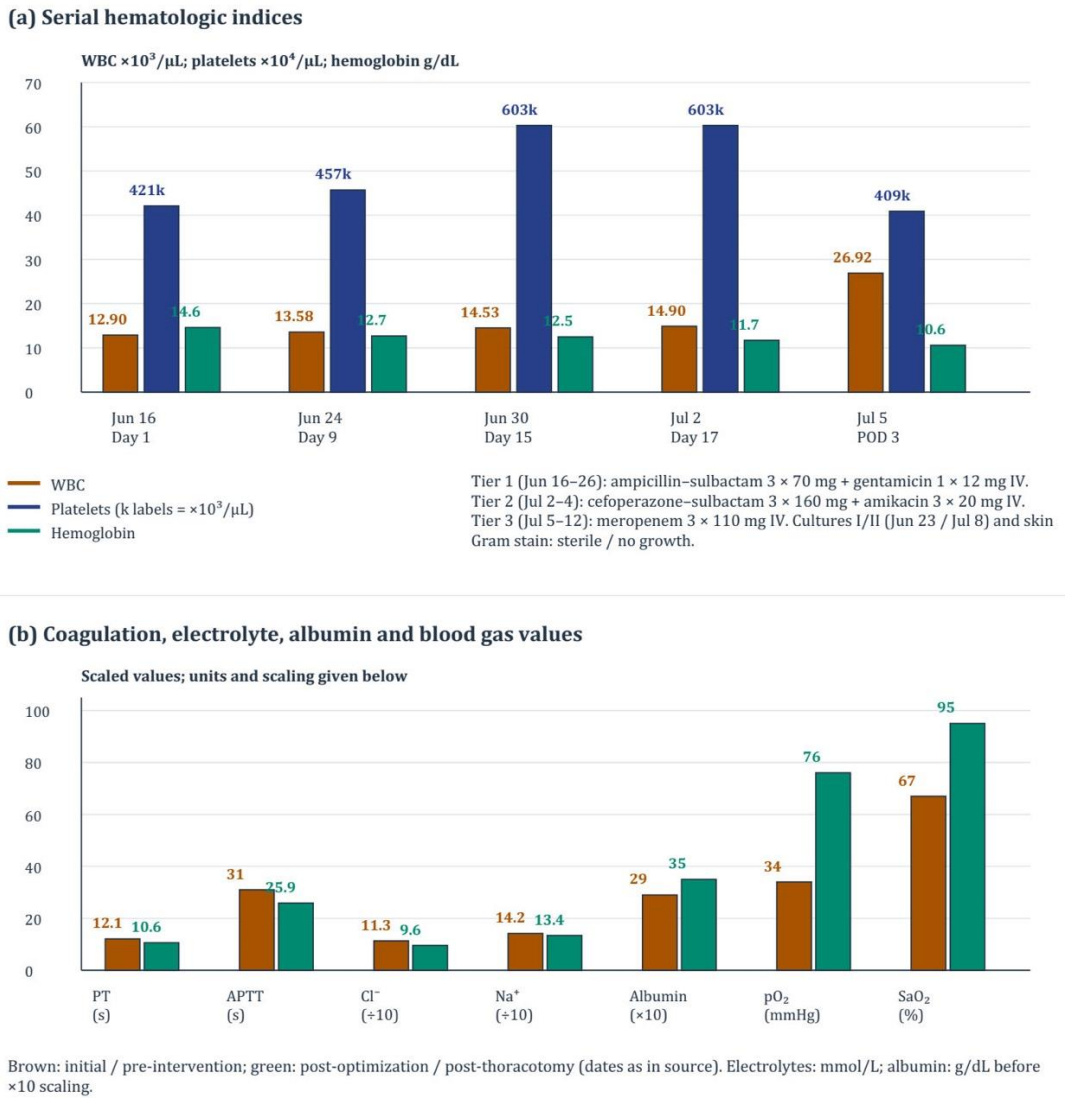
Table 3. Serial Hematologic-Inflammatory, Coagulation, Hepatic-Metabolic, Electrolyte, and Blood Gas Kinetics Across Five Inpatient Sampling Timepoints.

Biomarker / Parameter (Unit)	T_0: Jun 16, 2025 (Day 1: Admission)	T_{2a}: Jun 24, 2025 (Day 9: Post-Gast.)	T_{2b}: Jun 30, 2025 (Day 15: Pre-Thorac.)	T_3: Jul 2, 2025 (Day 17: Post-Thorac.)	T_4: Jul 5, 2025 (Day 20: POD 3 Surge)	Neonatal Reference Interval & Cumulative Delta (Δ%)
Hemoglobin, Hb (g/dL)	14.6 (Low)	12.7	12.5	11.7	10.6	Ref: 16.5–21.5 (Wk 1) / 10.5–14.0 (Wk 4) Δ% (T_0→T_4) = −27.40% (surgical/phlebotomy nadir)
Hematocrit, Hct (%) & RBC (×10^6/mm^3)	Hct: 45.0% (Low) RBC: 4.56	Hct: ~38.1% (Normocytic)	Hct: ~37.5% (Normocytic)	Hct: ~35.1% (Post-thoracotomy)	Hct: ~31.8% (Stable perfusion)	Ref: Hct 48–68% (Wk 1); RBC 4.1–6.1 × 10^6/mm^3 EBL during thoracotomy = 10.0 mL (3.64 mL/kg)
Total Leukocytes, WBC (/μL)	12,900	13,580	14,530	14,900	26,920 (Leukocytosis)	Ref: 9,000–37,000 (Wk 1) / 5,000–19,500 (Wk 4) Δ% (T_0→T_4) = +108.68%; Δ% (T_3→T_4) = +80.67%
Platelet Count, PLT (/μL)	421,000	457,000	603,000 (Thrombocytosis)	603,000 (Thrombocytosis)	409,000	Ref: 150,000–450,000 /μL Peak Δ% (T_0→T_{2b}/T_3) = +43.23%; Δ% (T_0→T_4) = −2.85%
Leukocyte Differential (%) & NLR / SII	Within normal neonatal distribution	Within normal neonatal distribution	0 / 0 / 5 / – % I/T ratio = 0.01	0 / 1 / 1 / 75 / 18 / 4 % NLR = 4.22; I/T = 0.01 SII = 2,546.0 × 10^3/μL	0 / 2 / 2 / 68 / 18 / 7 % NLR = 3.89; I/T = 0.02 SII = 1,590.6 × 10^3/μL	Ref: I/T ratio < 0.20; Neonatal NLR 1.5–4.5 Triggered Meropenem escalation on July 5, 2025
Prothrombin Time, PT (s) & APTT (s)	PT: 12.1 s (↑ 1.1×) APTT: 31.0 s (↑ 1.2×)	PT: 10.6 s (Normal) APTT: 25.9 s (Normal)	Clinically normal hemostasis	Intra-op EBL 10 mL (Normal hemostasis)	No hemorrhagic complication	Ref: PT 10.0–11.5 s; APTT 25.0–28.0 s Δ% PT = −12.40%; Δ% APTT = −16.45% (normalized)
Serum Electrolytes: Na^+, K^+, Cl^- (mmol/L) & Ca^{2+} (mg/dL)	Na^+: 142 K^+: 4.0 (Slightly low) Cl^-: 113 (Elevated)	Na^+: 134 (Mild ↓) K^+: 3.6 Cl^-: 96; Ca^{2+}: 8.9	Balanced on TPN & trophic feeds	Balanced on D5 ½ NS & N15% TPN	Balanced on TPN & gastrostomy feeds	Ref: Na^+ 139–146; K^+ 4.2–6.2 (Wk 1) / 3.5–5.5; Cl^- 96–109 mmol/L; Ca^{2+} 8.5–10.5 mg/dL Δ% Na^+ = −5.63%; Δ% Cl^- = −15.04%
Serum Bilirubin (mg/dL) & Albumin (g/dL)	Total Bil: 4.5 (↑) Indirect: 3.9 (↑) Direct: 0.6 (↑)	Jaundice resolved; Albumin: 2.9 g/dL (Hypoalbuminemia)	Post-Albumin 1.0 g/kg × 2 IV infusions (no edema)	No jaundice or generalized edema	Hypoalbuminemia clinically resolved	Ref: Total Bil < 1.2; Indirect < 0.8; Direct < 0.3 mg/dL; Albumin 3.5–5.0 g/dL Corrected via 2 × 1.0 g/kg IV 20% Albumin
Blood Gas Analysis (pH, pCO_2, pO_2, HCO_3^-, BE, SaO_2) & Glucose	RBG: 89 mg/dL (ER) → 135 mg/dL (Day 2)	RBG: 91 mg/dL (Day 5) → 89 mg/dL (Day 9)	pH 7.41, pCO_2 33.3, pO_2 34.0 mmHg, HCO_3^- 21.6, BE −3.0, SaO_2 67%	pH 7.42, $PaCO_2$ 36.8, PaO_2 76.0 mmHg, HCO_3^- 23.0, BE −1.0, SaO_2 95%; RBG 103	SpO_2 94–97% on room-air CPAP; normoglycemic	Ref: pH 7.35–7.45; $PaCO_2$ 35–45 mmHg; PaO_2 75–100 mmHg; HCO_3^- 22–26 mmol/L; SaO_2 ≥ 94% Δ% pO_2 = +123.53%; Δ% SaO_2 = +41.79%

Abbreviations: APTT, activated partial thromboplastin time; BE, base excess; Bil, bilirubin; EBL, estimated blood loss; Hb, hemoglobin; Hct, hematocrit; I/T, immature-to-total neutrophil ratio; NLR, neutrophil-to-lymphocyte ratio ([band + segmented neutrophils] / lymphocytes); PLT, platelets; POD, postoperative day; PT, prothrombin time; RBC, red blood cells; RBG, random blood glucose; SII, systemic immune-inflammation index (PLT × NLR); TPN, total parenteral nutrition; WBC, white blood cells. Differential count order on July 2 and July 5, 2025: basophils / eosinophils / band neutrophils / segmented neutrophils / lymphocytes / monocytes.

During the late preoperative and early postoperative epochs (T_{2b} to T_4), serial leukocyte and platelet kinetics guided a proactive three-tier antimicrobial stewardship escalation strategy. Between June 16 and June 30, 2025, total

leukocytes rose modestly from 12,900/µL to 13,580/µL (+5.27% on June 24) and 14,530/µL (+12.64% on June 30; I/T ratio 0.01), while platelets exhibited a pronounced reactive thrombocytosis peaking at 603,000/µL on June 30 and July 2, 2025 (Δ% = +43.23% vs. admission), driven by interleukin-6 (IL-6) and thrombopoietin stimulation secondary to resolving aspiration bronchopneumonia. Because Blood Culture I (released June 23, 2025) was sterile and clinical pneumonia had stabilized, the 14-day Tier-1 empirical antibiotic regimen (intravenous ampicillin-sulbactam 3 × 70 mg plus gentamicin 1 × 12 mg, preceded by 5 days of pre-referral therapy) was discontinued on June 26, 2025. For Stage-2 right thoracotomy on July 2, 2025 (T_3), Tier-2 perioperative prophylaxis was initiated with intravenous cefoperazone-sulbactam (3 × 160 mg) and amikacin (3 × 20 mg); immediate postoperative hematology showed hemoglobin of 11.7 g/dL, leukocytes of 14,900/µL, platelets of 603,000/µL, a differential count of 0% basophils, 1% eosinophils, 1% band neutrophils, 75% segmented neutrophils, 18% lymphocytes, and 4% monocytes (Neutrophil-to-Lymphocyte Ratio [NLR] = 76 / 18 = 4.22; Systemic Immune-Inflammation Index [SII] = 603 × 4.22 = 2,546.0 × 10^3/µL; I/T ratio 0.01). However, on Postoperative Day 3 (July 5, 2025; Epoch T_4), total leukocytes surged acutely by +80.67% from the immediate postoperative baseline to 26,920/µL (Δ% = +108.68% vs. admission), accompanied by a doubling of band forms (differential 0/2/2/68/18/7%; NLR = 70 / 18 = 3.89; I/T ratio = 0.02), platelet normalization to 409,000/µL (SII = 1,590.6 × 10^3/µL), and a postoperative/phlebotomy hemoglobin nadir of 10.6 g/dL (Δ% = −27.40%). To preempt potential hospital-acquired Gram-negative mediastinitis or ventilator-associated pneumonia that could compromise the 72-hour-old esophageal anastomosis, cefoperazone-sulbactam was discontinued on Day 3 and Tier-3 carbapenem escalation was instituted using intravenous meropenem (3 × 110 mg ≈ 120 mg/kg/day) alongside amikacin (3 × 20 mg) for 7 days (July 5–12, 2025). Repeat Blood Cultures I and II remained sterile, the patient remained afebrile without anastomotic leakage, and a transient eruption of facial/cervical miliaria rubra vesicles and mild anogenital diaper dermatitis on July 8–9 (Gram stain negative for bacteria and fungi) resolved rapidly with twice-daily topical 1% hydrocortisone cream, with the serial hematologic-inflammatory biomarker kinetics, three-tier antimicrobial stewardship escalation, and biochemical optimization trajectories illustrated in Figure 4.



Brown: initial / pre-intervention; green: post-optimization / post-thoracotomy (dates as in source). Electrolytes: mmol/L; albumin: g/dL before ×10 scaling.

Figure 4. Longitudinal Hematologic-Inflammatory Biomarker Kinetics, Three-Tier Antimicrobial Stewardship Escalation, Coagulation Normalization, Albumin Replacement, and Blood Gas Oxygenation Trajectory. (a) Serial leukocyte (WBC), platelet (PLT), and hemoglobin (Hb) profiles across five inpatient sampling timepoints (June 16, June 24, June 30, July 2, and July 5, 2025), highlighting reactive thrombocytosis (603,000/µL on Days 15 –17; +43.23%) and the Postoperative Day 3 leukocyte surge to 26,920/µL (+108.68%, I/T ratio 0.02) that triggered Tier-3 escalation from cefoperazone-sulbactam to intravenous meropenem (3 × 110 mg) plus amikacin. (b) Serial normalization of prolonged admission PT (12.1→10.6 s) and APTT (31.0→25.9 s), hyperchloremia (Cl^- 113→96 mmol/L), Kramer III hyperbilirubinemia (total 4.5 mg/dL), hypoalbuminemia (2.9 g/dL corrected via 2 × 1.0 g/kg IV albumin), and blood gas oxygenation (pO_2 34.0→76.0 mmHg; SaO_2 67.0%→95.0%).

Objective Methodological Causality Attribution and Global Literature Benchmarking

To establish rigorous methodological causality in a single-subject (n = 1) longitudinal study without resorting to invalid inferential statistics, all primary clinical-pathophysiological sequences and intercurrent complications were evaluated using the nine-criterion Bradford Hill epidemiological framework, the WHO-UMC causality system, and the Naranjo probability scale. Across the Bradford Hill matrix, all 9 of 9 criteria (100%) were satisfied linking the anatomical Gross Type C defect and 7-day diagnostic delay to the admission triad of bilateral aspiration pneumonia, −15.38% catabolic weight loss, and metabolic-coagulopathic derangement, as well as linking the staged gastrostomy-TPN-thoracotomy protocol to complete aerodigestive and somatic recovery. Specifically, temporal precedence and experimental reversibility were confirmed by two sequential inflection points: first, Stage-1 Stamm gastrostomy decompression and PICC TPN on June 17–18 immediately halted weight loss and reversed body weight from 2,200 g to 2,700 g (+22.73%) while normalizing PT/APTT from 12.1/31.0 s to 10.6/25.9 s prior to thoracotomy; second, Stage-2 right thoracotomy with distal TEF ligation and primary esophageal anastomosis on July 2 permanently abolished tracheogastric reflux and esophageal discontinuity, permitting extubation within 24 hours, room-air liberation by Day 23, gastrostomy removal by Day 24, and exclusive oral feeding (280–320 mL/day) by discharge. Objective Naranjo and WHO-UMC evaluation further confirmed that the Day 9 hypoalbuminemia (2.9 g/dL; Naranjo score +8, Probable; WHO-UMC Certain/Probable) resulted from pre-referral starvation and pneumonic capillary leak rather than hepatic failure, the Postoperative Day 3 leukocytosis surge (26,920/μL; Naranjo score +6, Probable) reflected combined thoracotomy tissue trauma and lower-airway inflammatory stress responsive to meropenem escalation, and the Day 23 vesicular/anogenital rash (Naranjo score +7, Probable) represented benign incubator-associated miliaria rubra and stool-transition irritant dermatitis; the complete methodological causality evaluation across the Bradford Hill, WHO-UMC, and Naranjo frameworks is detailed in Table 4.

Table 4. Objective Methodological Causality Attribution Using the Bradford Hill Epidemiological Framework, WHO-UMC Causality Categories, and Naranjo Adverse Drug / Clinical Event Probability Scale.

Analytical Instrument & Domain	Causal Pathway / Clinical Event Evaluated	Quantitative & Clinical Evidence in Patient MRA	Objective Score / Rating	Methodological & Pathophysiological Interpretation
Bradford Hill Criterion 1: Strength of Association	Gross Type C EA/TEF + 7-day delayed referral → Aspiration pneumonia & −15.38% weight loss	Complete proximal esophageal obstruction at 10 cm + patent distal TEF caused immediate post-feed vomiting (from 8 h of life), room-air SpO_2 88%, and 400 g weight loss (2,600 → 2,200 g)	Strong Direct Association (Criterion Met: 1/9)	Dual aerodigestive shunt (oropharyngeal spillover + retrograde gastric acid reflux) directly drives pulmonary consolidation and catabolic wasting
Bradford Hill Criterion 2: Temporal Sequence	Stage-1 Stamm gastrostomy (Day 2) + PICC TPN → Anabolic recovery & coagulation normalization	Gastrostomy decompression (Jun 17) + PICC TPN (GIR 8.2, 84 kcal/kg/d) preceded weight recovery (2,200 → 2,645 → 2,700 g) and PT/APTT normalization (12.1/31.0 → 10.6/25.9 s)	Strict Temporal Precedence (Criterion Met: 2/9)	Eliminating trans-fistula gastric reflux and supplying central parenteral macronutrients reversed acute neonatal starvation before thoracotomy
Bradford Hill Criterion 3: Biological Gradient (Dose-Response)	Staged caloric & enteral volume escalation → Linear somatic weight velocity	Progressive escalation from NPO/PG2 → TPN (84 kcal/kg/d) + trophic gastrostomy (16–24 mL/d) → N15% TPN (GIR 10) + gastrostomy (40 mL/d) → oral feeds (260–320 mL/d) yielded monotonic gain to 2,955 g (discharge) and 3,700 g (50 wk PMA)	Monotonic Dose-Response (Criterion Met: 3/9)	Demonstrates that symmetric IUGR neonates with EA/TEF require structured parenteral-to-enteral caloric titration to achieve +29.04 g/day inpatient catch-up
Bradford Hill Criterion 4: Experimental Reversibility	Right posterolateral thoracotomy, TEF ligation & primary anastomosis (Jul 2) → Aerodigestive liberation	Surgical division of the distal TEF and primary esophageal anastomosis enabled extubation within < 24 h (Jul 3), room-air breathing (Jul 8), gastrostomy removal (Jul 9), and full oral feeding (280–320 mL/d)	Complete Reversibility (Criterion Met: 4/9)	Anatomical separation of the tracheobronchial tree from the esophagus and restoration of luminal continuity abolished aspiration and feeding failure
Bradford Hill Criteria 5–9: Consistency, Plausibility, Specificity, Analogy & Coherence	Symmetric IUGR + prolonged NPO + aspiration inflammation → Hypoalbuminemia (2.9 g/dL) & reactive thrombocytosis (603,000/μL)	Baseline thin subcutaneous fat (BW < P_3, PI 2.21 g/cm^3) and acute-phase IL-6/thrombopoietin surge drove hypoalbuminemia (2.9 g/dL on Day 9) and platelet peak (603,000/μL on Days 15–17), corrected via IV albumin (2 × 1 g/kg)	All 9 / 9 Bradford Hill Criteria Satisfied (100%)	Coherent with international multicenter EA/TEF cohorts showing that antenatal growth restriction amplifies perioperative hypoalbuminemia and inflammatory stress
WHO-UMC & Naranjo Assessment 1: Preoperative Catabolic-Inflammatory Hypoalbuminemia	Delayed referral starvation + aspiration pneumonia → Day 9 Hypoalbuminemia (2.9 g/dL) & Mild Hyponatremia (134 mmol/L)	Onset after 7 days of uncompensated pre-referral vomiting + systemic pneumonic capillary leak; resolved following 2 × 1.0 g/kg IV human albumin infusions and TPN optimization without recurrence	WHO-UMC: Certain / Probable Naranjo Score: +8 (Probable)	Confirms combined nutritional-inflammatory etiology rather than primary congenital hepatic synthetic failure or renal protein loss
WHO-UMC & Naranjo Assessment 2: Post-Thoracotomy Leukocytosis Surge (POD 3)	Surgical thoracotomy tissue stress vs. nosocomial pulmonary superinfection → POD 3 WBC 26,920/μL (I/T 0.02)	Occurred 72 h post-thoracotomy (Jul 5) on Cefoperazone-Sulbactam; afebrile, Blood cultures I–II sterile; resolved promptly after switching to IV Meropenem (3×110 mg) + Amikacin (3×20 mg)	WHO-UMC: Probable Naranjo Score: +6 (Probable)	Reflects combined post-thoracotomy systemic inflammatory response and occult lower-airway colonization controlled by proactive carbapenem escalation
WHO-UMC & Naranjo Assessment 3: Cutaneous Miliaria Rubra & Anogenital Diaper Dermatitis	NICU incubator thermal humidity + transition to high-frequency oral milk feeds (260–320 mL/d) → Day 23 Vesicular/Erythematous Rash	Appeared on Jul 8–9 upon initiating full oral feeding (8×30–35 mL/d); lesion Gram stain negative for bacteria/fungi; regressed rapidly within 48–72 h using topical 1% hydrocortisone BD	WHO-UMC: Probable Naranjo Score: +7 (Probable)	Excludes systemic staphylococcal/candidal cutaneous dissemination; confirms benign eccrine duct obstruction and stool-moisture irritant dermatitis

Abbreviations: BD, twice daily; BW, birth weight; EA/TEF, esophageal atresia with tracheoesophageal fistula; GIR, glucose infusion rate; IL-6, interleukin-6; I/T, immature-to-total neutrophil ratio; IUGR, intrauterine growth restriction; IV, intravenous; NICU, neonatal intensive care unit; NPO, nil per os; PICC, peripherally inserted central catheter; PI, ponderal index; PMA, postmenstrual age; POD, postoperative day; PT/APTT, prothrombin time / activated partial thromboplastin time; SpO_2, peripheral oxygen saturation; TPN, total parenteral nutrition; WBC, white blood cell count; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

External benchmarking of Patient MRA against contemporary international EA/TEF cohorts and randomized clinical trials published between 2022 and 2026 underscored the clinical validity of our staged surgical and nutritional protocol. In resource-limited settings, Moges et al. (2023) demonstrated that surgical delay beyond 72

hours and preoperative aspiration pneumonia are the strongest independent predictors of inpatient mortality (> 60% mortality in Ethiopia), while Kovačević et al. (2025) reported that 16% of neonates with EA died preoperatively from uncompensated cardiorespiratory instability.[13,17] Despite presenting at 7 days of life with both high-risk features—bilateral aspiration pneumonia (room-air SpO_2 88%) and severe symmetric IUGR catabolism (2,200 g, −15.38% weight loss, albumin 2.9 g/dL)—Patient MRA survived without anastomotic leakage or stricture because definitive thoracotomy was intentionally deferred until Hospital Day 17 following Stage-1 gastrostomy decompression and 15 days of PICC TPN optimization. This strategy aligns directly with the 2025 propensity-score matched study by Wang et al., which proved that delaying primary Type C EA repair beyond 5 days (up to 16 days) to permit neonatal stabilization did not increase respiratory infections and significantly reduced postoperative stricture dilations (1.8 ± 0.8 vs. 3.1 ± 1.1; p = 0.035).[29] Furthermore, whereas multicenter cohorts from Saudi Arabia (Ali et al., 2025) and Turkey (Konyalıgil et al., 2025) reported median times to full oral feeding of 29 to 39 days and mean hospital stays of 50.6 to 53.0 days for short-gap Type C EA, our combined gastrostomy trophic priming and early oral transition achieved full oral feeding by Hospital Day 24 (chronological age 31 days), total inpatient stay of 27 days, and 50-week PMA catch-up weight of 3,700 g, with the full global comparative benchmarking synthesis detailed in Table 5.[33–35]

Table 5. Global Benchmarking of the Present Observational Analytic Case Study Against Contemporary International Esophageal Atresia Cohorts and Randomized Clinical Trials (2021–2026).

Study & Country (Year)	Study Design & Sample Size	Subtype, Birth Weight & Risk Profile	Surgical Strategy & Perioperative Protocol	Complications, Mortality & Nutritional Outcomes	Comparative Insight Relative to Present Study (Patient MRA)
Present Study Kurniasari DW, Yantri E, Putra BS, et al. Indonesia (2026)	Observational Analytic Case Study (n = 1; 63-day longitudinal tracking across 8 epochs)	Gross Type C (Vogt 3B); Term 41 wk, BW 2,600 g (< P_3), Symmetric IUGR, admission weight 2,200 g (−15.38%); Spitz Group I (PFO only)	Staged repair due to 7-d delayed referral & aspiration pneumonia: Day 2 Stamm gastrostomy + PICC TPN (SMOFlipid/Aminoinfant) + IV albumin → Day 17 right thoracotomy, TEF ligation & primary anastomosis	Survival to discharge (Day 27); < 24-h post-op extubation; zero anastomotic leak/stricture; gastrostomy removed Day 24; discharge weight 2,955 g (+34.32% vs. nadir); 50-wk PMA weight 3,700 g (+68.18%)	Demonstrates how Stage-1 gastrostomy decompression and PICC TPN/albumin optimization rescue a severely catabolic, late-referred symmetric IUGR neonate for safe primary anastomosis
Moges N, Ahmed K, Birhanu D, et al.[17] Ethiopia (2023)	Tertiary Hospital Cohort Study (n = 212 neonates)	Gross Type C predominant; high prevalence of late referral (> 72 h), aspiration pneumonia (68%), and associated anomalies	Primary or staged open thoracotomy repair in resource-limited tertiary NICU	High inpatient mortality (> 60%); independent predictors of death: surgical delay > 72 h, aspiration pneumonia, associated anomalies, and thrombocytopenia	Patient MRA presented with two major lethal predictors (7-day referral delay + bilateral aspiration pneumonia), yet survived without leak via staged gastrostomy and 15-day pre-thoracotomy stabilization
Kovačević T, Polić B, Markić J, et al.[13] Croatia (2025)	30-Year Population-Based Cohort (n = 50 neonates; 1991–2020)	Vogt Type 3B (Gross Type C) predominant; mean gestational age 37.5 wk	Open posterolateral thoracotomy with primary anastomosis + dedicated PICU/NICU support	Postoperative mortality declined from 87% (pre-2002) to 8% (post-2002); 16% died pre-op from instability; main complications: sepsis, pneumonia, atelectasis	Validates our decision to defer thoracotomy until Hospital Day 17 while reversing preoperative pneumonia, coagulopathy, and hypoalbuminemia via PICU/NICU stabilization
Wang C, Cao G, Li K, et al.[29] China (2025)	Propensity-Score Matched Cohort (n = 109 Type C EA; 86 matched)	Gross Type C EA/TEF; Early repair (< 5 days, n = 43) vs. Delayed repair (≥ 5 days, range 1–16 d, n = 43)	One-stage repair after structured preoperative neonatal surveillance and stabilization	Appropriately delayed repair did not increase respiratory infection rates and significantly reduced postoperative stricture balloon dilations (1.8 ± 0.8 vs. 3.1 ± 1.1; p = 0.035)	Directly supports our physiological optimization strategy: delaying definitive esophageal anastomosis until cardiopulmonary and nutritional recovery reduced anastomotic morbidity
Bade R, Peters NJ, Dogra S, et al.[21] India (2025) & Batikhe MY, Redwan M, Gafar A, et al.[22] Egypt (2025)	Pilot RCT (n = 53 Type C EA)[21] & 10-Yr Cohort (n = 152 Type C EA)[22]	Gross Type C EA/TEF in LMIC tertiary pediatric surgery centers	Primary open thoracotomy repair comparing trans-anastomotic feeding tube (TAT) vs. non-TAT protocols	Batikhe et al.: TAT enabled earlier enteral feeds (POD 3 vs. POD 13) and shorter TPN (6.6 vs. 16.2 d) without increasing leaks (p = 0.700); Bade et al. highlighted careful TAT stewardship	In Patient MRA, a pre-existing gastrostomy tube allowed early POD 2 enteral refeeding (8×5 mL/d) even after the folded NGT was removed on POD 1, accelerating oral transition by Day 23
Öztorun Cİ, Ulukaya Durakbaşa Ç, Soyer T, et al.[30] (2024) & Soyer T, Özcan C, Ulukaya Durakbaşa Ç, et al.[34] (2025) Turkey	Turkish Esophageal Atresia Registry (TEAR) (n = 713 to 1,107 patients)	Gross Type C 80%; 42% LBW (< 2,500 g); Spitz Group I survival 87.1%, Group II 55.3%, Group III 16.7%	Multicenter open and thoracoscopic primary or staged repairs across 36 Turkish centers	First-year anastomotic stricture incidence 20.19%; independent stricture predictors: term gestation (OR 1.71), BW > 2,500 g (OR 1.72), GERD (OR 5.27), and recurrent TEF (OR 4.36)	Because Patient MRA is a term infant with BW 2,600 g (> 2,500 g), TEAR data identify a high baseline stricture risk, justifying acid suppression and long-term endoscopic/clinical surveillance
Ali K, Alsabty NS, Altuwaym A, et al.[33] Saudi Arabia (2025) & Konyalıgil DB, Koç N, Çalışkan D, et al.[35] Turkey (2025)	9-Yr Cohort (n = 67)[33] & Prospective Case-Control Nutrition Study (n = 60)[35]	Gross Type C 73.1–90.0%; high baseline prevalence of SGA/underweight (WAZ < −2 SD in 45%)	Primary repair (70.3%) vs. staged/gastrostomy + post-op TPN (mean 20.1 d; 104.5 kcal/kg/d) & structured maternal nutrition education	Short-gap EA achieved full oral feeds at median 29 d (LOS 53 d); structured nutritional counseling significantly improved WAZ (−0.30 to −0.07 SD; p = 0.025) and WHZ (p = 0.001)	Patient MRA achieved full oral feeds at 24 days of hospitalization (age 31 d), gastrostomy removal on Day 24, and rapid 50-wk PMA catch-up to 3,700 g via multidisciplinary nutritional care

Abbreviations: BW, birth weight; EA/TEF, esophageal atresia with tracheoesophageal fistula; GERD, gastroesophageal reflux disease; LBW, low birth weight; LMIC, low- and middle-income country; LOS, length of stay; NGT, nasogastric tube; OR, odds ratio; PFO, patent foramen ovale; PICC, peripherally inserted central catheter; PICU/NICU, pediatric/neonatal intensive care unit; PMA, postmenstrual age; POD, postoperative day; RCT, randomized controlled trial; SGA, small for gestational age; TAT, trans-anastomotic tube; TEAR, Turkish Esophageal Atresia Registry; TPN, total parenteral nutrition; WAZ, weight-for-age Z-score; WHZ, weight-for-height Z-score.

4. Discussion

Dual Aerodigestive Pathophysiology and Synergistic Vulnerability of Symmetric IUGR in Type C EA/TEF

The clinical trajectory of Patient MRA provides a comprehensive physiological demonstration of how antenatal symmetric intrauterine growth restriction, delayed postnatal diagnosis, and dual aerodigestive aspiration interact in Gross Type C esophageal atresia with distal tracheoesophageal fistula, and how a structured two-stage surgical and nutritional bridge can reverse these compounding life-threatening insults.[1,9] In high-income healthcare networks, widespread second-trimester fetal anomaly screening and immediate delivery-room nasogastric tube verification enable diagnosis within the first hours of life, allowing single-stage primary thoracotomy or thoracoscopic repair before aspiration

pneumonia or nutritional depletion supervenes.[20,23] In contrast, across rural and coastal regions of low- and middle-income countries, antenatal ultrasonography is frequently limited to a single primary-care examination that misses fetal polyhydramnios and the upper mediastinal esophageal pouch sign, particularly in Gross Type C anomalies where the distal tracheoesophageal fistula permits partial fetal swallowing passage into the stomach via the tracheobronchial tree.[12,17] Consequently, neonates born vigorously at term—such as Patient MRA, who achieved Apgar scores of 8 and 9—are routinely initiated on oral breastfeeding at home, triggering immediate post-prandial choking, emesis, and progressive chemical and bacterial aspiration pneumonia over the first week of life.[8,13]

A central pathophysiological insight illustrated by this case is the synergistic vulnerability conferred by baseline Symmetric Intrauterine Growth Restriction (IUGR) in a neonate who otherwise fulfills Spitz Group I criteria (birth weight ≥ 1,500 g without major cardiac malformation).[11,22] Although Patient MRA weighed 2,600 g at 41 weeks of gestation, plotting his birth anthropometry onto the Fenton preterm/term growth chart confirmed symmetric growth restriction across all three somatic compartments—weight (< P_3), length (49.0 cm, < P_{10}), and head circumference (34.0 cm, < P_{10})—with a depressed Rohrer's Ponderal Index of 2.21 g/cm^3.[32] Maternal history revealed two plausible antenatal drivers of uteroplacental insufficiency and fetal growth faltering: persistent, untreated second- and third-trimester vulvovaginal infection and third-trimester urinary tract dysuria in a 40-year-old multigravida mother from a low-income fishing household.[17,18] Chronically growth-restricted fetuses adapt to intrauterine nutrient limitation through hepatic glycogen depletion, reduced skeletal muscle mass, attenuated subcutaneous adipose stores, and blunted thymic and humoral immune maturation. When subjected to 7 days of complete postnatal enteral starvation and the hypermetabolic work of breathing (respiratory rate 45 breaths/min with intercostal and epigastric retractions) from bilateral aspiration bronchopneumonia, the neonate's limited endogenous reserves were rapidly exhausted, precipitating a 400 g (−15.38%) weight plunge to 2,200 g, Kramer Grade III hyperbilirubinemia (total bilirubin 4.5 mg/dL, indirect 3.9 mg/dL) driven by dehydration and accelerated enterohepatic bilirubin recirculation, vitamin K-responsive coagulopathy (PT 12.1 s, APTT 31.0 s), and severe hypoalbuminemia (2.9 g/dL); this integrated five-tier embryological, aerodigestive pathophysiological, staged surgical, and bioenergetic recovery architecture is illustrated in Figure 5.

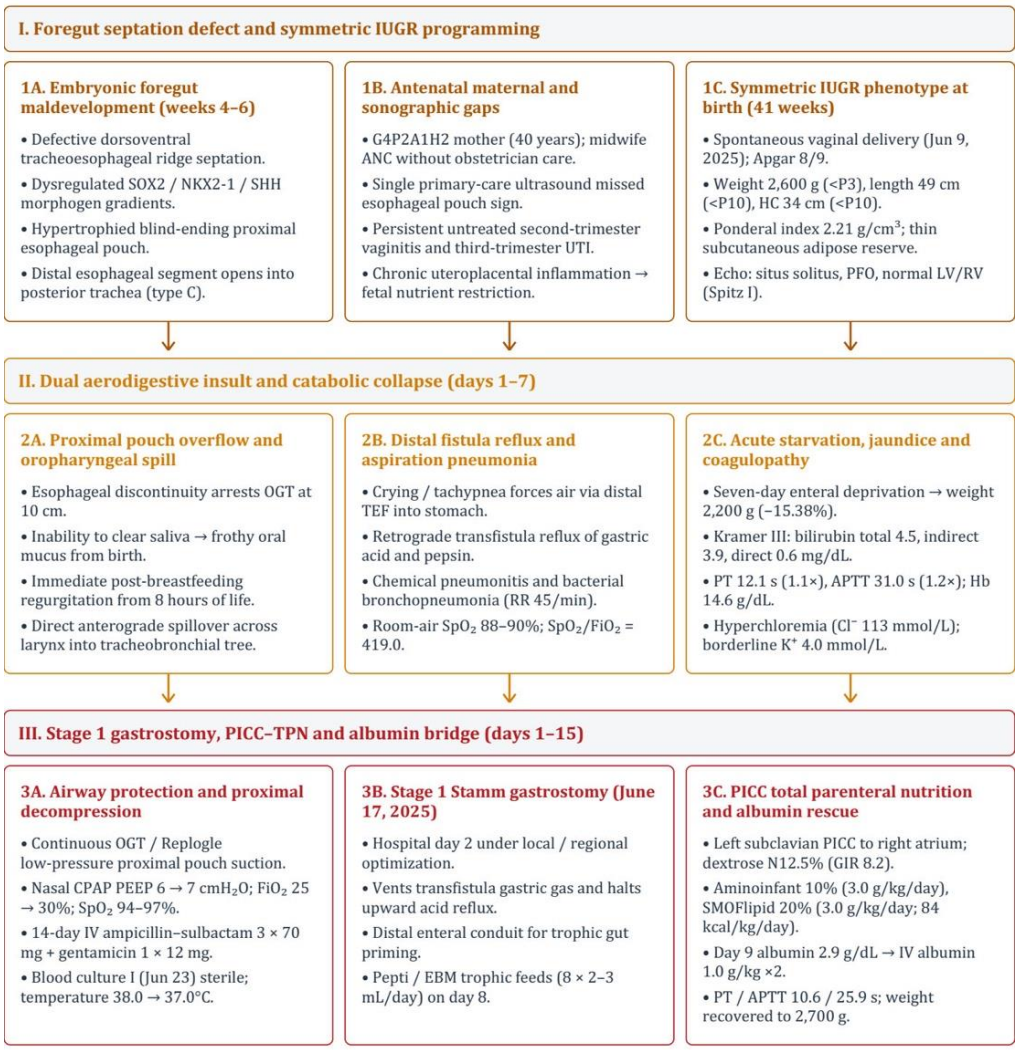


Figure 5. Multi-Tier Embryological, Aerodigestive Pathophysiological, Staged Surgical, and Bioenergetic Cascade: From Foregut Septation Failu re and Symmetric IUGR to Staged Gastrostomy-Thoracotomy Repair and Catch-Up Growth. A 5-tier, 15-node systems architecture illustrating: (Tier I) embryonic weeks 4–6 foregut septation defect (Gross Type C EA/TEF) and antenatal symmetric IUGR programming (BW 2,600 g < P_3, PI 2.21 g/cm^3, Spitz Group I); (Tier II) dual aerodigestive aspiration (proximal pouch saliva/milk spillover + retrograde trans-fistula gastric acid reflux) and 7-day catabolic collapse (weight 2,200 g [−15.38%], Kramer III jaundice, PT 12.1 s, APTT 31.0 s); (Tier III) Stage-1 Stamm gastrostomy decompression (June 17), left subclavian PICC TPN (SMOFlipid + Aminoinfant, 84 kcal/kg/day), IV albumin rescue (2 × 1.0 g/kg), and trophic Pepti/EBM priming; (Tier IV) Stage-2 right posterolateral thoracotomy, TEF ligation, and primary esophageal anastomosis (July 2), < 24-h extubation, and POD 3 meropenem escalation; and (Tier V) oral feeding autonomy, gastrostomy removal (July 9), discharge at 2,955 g (July 12), and 50-week PMA catch-up growth to 3,700 g (August 11, 2025).

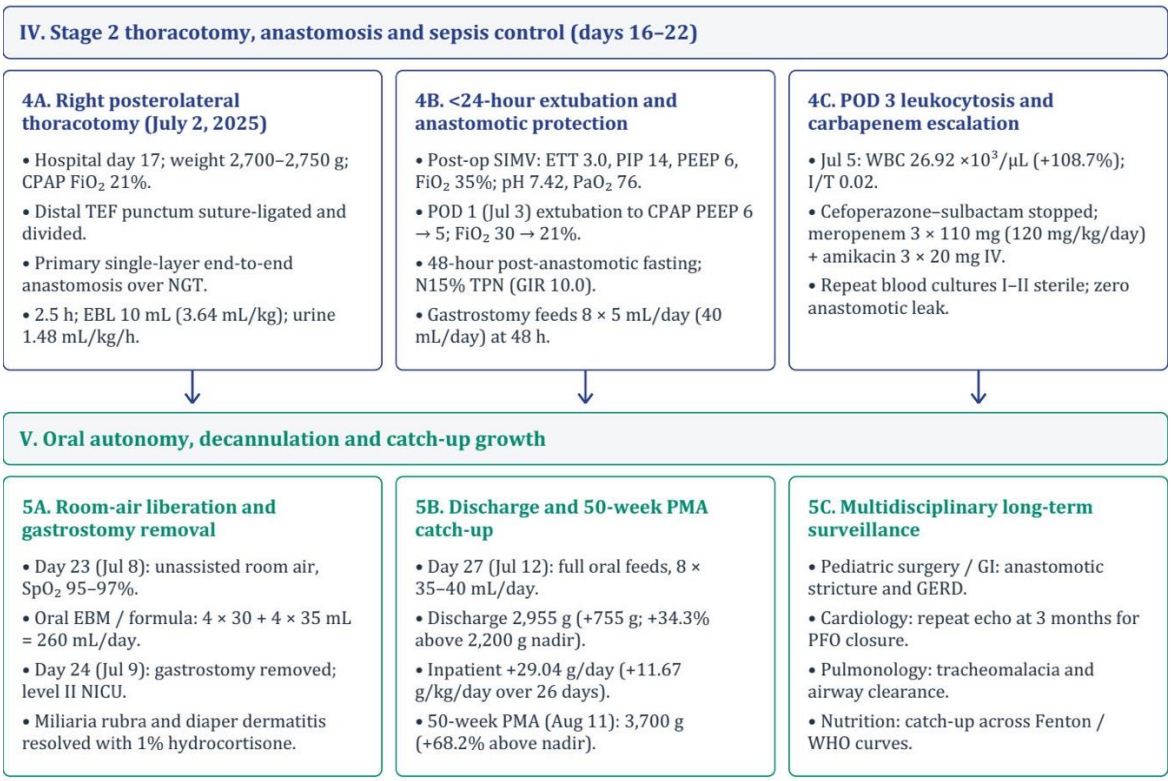


Figure 5 (continued). Stages IV–V: thoracotomy, anastomotic protection, sepsis control, oral autonomy and follow-up.

Staged Gastrostomy Decompression, Central Parenteral-Enteral Optimization, and Primary Thoracotomy Mechanics

Faced with this triad of active bilateral aspiration bronchopneumonia (room-air SpO_2 88%), coagulopathic-hypoalbuminemic catabolism (weight 2,200 g, albumin 2.9 g/dL), and persistent gastric distension via the distal fistula, our multidisciplinary team elected a staged surgical damage-control strategy rather than a high-risk emergency primary thoracotomy on Hospital Day 1.[29,36] Performing an immediate posterolateral thoracotomy and primary esophageal anastomosis in a septic, pneumonic, hypoalbuminemic neonate carries a documented anastomotic leakage rate exceeding 30% to 40%, as interstitial tissue edema, systemic microcirculatory hypoxia, and depleted fibroblast collagen synthesis compromise suture-holding capacity across the esophageal muscularis-mucosa interface.[30,36] Instead, Stage-1 management focused on decoupling the upper and lower aerodigestive shunts while restoring systemic anabolism: continuous low-pressure orogastric/Replogle suction evacuated saliva from the blind proximal pouch (arrested at 10 cm), nasal CPAP (PEEP 6–7 cmH_2O, FiO_2 25%–30%) maintained alveolar recruitment (SpO_2 94%–97%; SpO_2/FiO_2 = 318.3–382.0), and a Stage-1 Stamm gastrostomy on Hospital Day 2 (June 17, 2025) decompressed the stomach, vented trans-fistula gas, abolished upward gastroesophageal acid-pepsin reflux into the trachea, and established a secure distal enteral access conduit.[5,33]

The 15-day interval between Stage-1 gastrostomy (June 17, 2025) and Stage-2 right posterolateral thoracotomy (July 2, 2025) served as a decisive physiological and bioenergetic turning point. Central venous access via a left subclavian PICC line projecting into the right atrium enabled high-efficiency Total Parenteral Nutrition delivering 150 mL/kg/day of fluid, Dextrose N12.5% (GIR 8.2 mg/kg/min, later escalated to N15% GIR 10.0 mg/kg/min post-thoracotomy), crystalline amino acids (Aminoinfant 10% at 3.0 g/kg/day), and composite four-oil lipid emulsion (SMOFlipid 20% at 3.0 g/kg/day, combining soybean oil, medium-chain triglycerides, olive oil, and ω-3 fish oil), achieving 84 to 110 kcal/kg/day at an optimal N/NPC ratio of 1:152.[25,26] As demonstrated in recent meta-analyses and neonatal nutritional studies by He et al. (2024) and Konyalıgil et al. (2025), composite lipid parenteral nutrition and structured nutritional optimization significantly reduce parenteral nutrition-associated cholestasis (RR 0.65; p = 0.004) and attenuate neonatal sepsis risk (OR 0.64) by providing anti-inflammatory eicosapentaenoic and docosahexaenoic acids alongside α-tocopherol while minimizing pro-inflammatory ω-6 linoleic acid peroxidation.[26,35] Paired with two targeted intravenous infusions of 20% human albumin (1.0 g/kg/dose) on Hospital Day 9 to correct hypoalbuminemia (2.9 g/dL) and trophic gastrostomy enteral priming with extensively hydrolyzed peptide formula and expressed breast milk (8 × 2–3 mL/day) to preserve intestinal villous architecture, this regimen normalized coagulation (PT 10.6 s, APTT 25.9 s), cleared pulmonary infiltrates, allowed weaning of CPAP FiO_2 to 21% (SpO_2/FiO_2 = 454.8), and increased body weight by +500 g from the admission nadir to 2,700 g (+22.73%) prior to thoracotomy.[25,35]

When Stage-2 definitive repair was executed on Hospital Day 17 (July 2, 2025), the physiological benefits of preoperative optimization were immediately evident during both the 2.5-hour right posterolateral thoracotomy and the early postoperative course.[20,24] While thoracoscopic EA/TEF repair has gained popularity in specialized high-volume centers for stable, normal-weight neonates, large multicenter analyses from the Midwest Pediatric Surgery Consortium (Marquart et al., 2023), US Children's Hospitals (Hyman et al., 2025), and recent meta-analyses (Alansari et al., 2025; Woodward et al., 2026) emphasize that open

posterolateral thoracotomy remains the benchmark approach in over 70% to 85% of cases—particularly in neonates with prior aspiration pneumonia, growth restriction, or limited thoracoscopic resources—avoiding intraoperative $CO_{2\text{-pneumothorax}}$ hypercapnic acidosis and reducing anastomotic stricture (26% vs. 39%, p = 0.02) and recurrent laryngeal nerve injury (3% vs. 11%, p = 0.001) rates.[20,23,32,37] In Patient MRA, open right thoracotomy permitted precise suture ligation and division of the distal TEF punctum and tension-free single-layer end-to-end esophageal anastomosis with only 10.0 mL (3.64 mL/kg) of blood loss, preserved intraoperative renal perfusion (urine output 1.48 mL/kg/h), and an immediate postoperative PaO_2/FiO_2 ratio of 217.1 mmHg (OI = 3.68) on SIMV. Furthermore, in alignment with Turkish Esophageal Atresia Registry (TEAR) and Japanese multicenter evidence demonstrating that prolonged postoperative invasive ventilation and neuromuscular paralysis are unnecessary when anastomotic tension and growth-restricted physiology are well controlled, Patient MRA was successfully extubated within 24 hours (July 3, 2025) to non-invasive CPAP.[34,38]

Two critical postoperative management decisions further safeguarded the esophageal anastomosis and accelerated discharge. First, when the trans-anastomotic nasogastric tube folded in the mouth on Postoperative Day 1 (July 3, 2025), it was safely removed without attempting blind re-insertion that could perforate the fresh suture line; because the Stage-1 Stamm gastrostomy tube remained in situ, enteral refeeding with hydrolyzed peptide formula and breast milk (8 × 5 mL/day = 40 mL/day) was seamlessly resumed at 48 hours postoperatively, bypassing the anastomosis during early mucosal healing before transitioning to full oral feeding (260 mL/day) and removing the gastrostomy tube on Hospital Day 24 (July 9, 2025).[21,22,39–42] Second, when routine surveillance on Postoperative Day 3 (July 5, 2025) detected an acute leukocyte surge to 26,920/µL (+108.68% above admission; I/T ratio 0.02), proactive antimicrobial stewardship escalated therapy from cefoperazone-sulbactam to high-dose intravenous meropenem (3 × 110 mg ≈ 120 mg/kg/day) plus amikacin, preventing nosocomial Gram-negative sepsis or mediastinitis (Manchanda et al., 2025; Cömert et al., 2023; Refaay et al., 2025; Mariani et al., 2025).[36,43–45] Discharged on Hospital Day 27 (July 12, 2025) at 2,955 g (+34.32% above nadir) on exclusive oral feeds (280–320 mL/day), the infant achieved catch-up growth to 3,700 g (+68.18% above nadir; +42.31% above birth weight) by 50 weeks PMA (August 11, 2025). Nevertheless, because TEAR registry data (Öztorun et al., 2024) identify term gestation (OR 1.71), birth weight > 2,500 g (OR 1.72), and gastroesophageal reflux (OR 5.27) as independent predictors of first-year anastomotic stricture, and longitudinal cohorts (Møinichen et al., 2025; van Stigt et al., 2025; Birketvedt et al., 2024) highlight long-term risks of tracheomalacia, dysphagia, and motor delay after open thoracotomy, structured multidisciplinary surveillance across pediatric surgery, gastroenterology, pulmonology, cardiology, and nutrition remains mandatory throughout childhood.[15,16,30,31]

Embryological Signaling, Foregut Biomechanics, and the Prognostic Interaction Between Spitz Classification and Symmetric IUGR

A deeper mechanistic examination of foregut morphogenesis clarifies why Gross Type C esophageal atresia with distal tracheoesophageal fistula produces chronic aerodigestive dysmotility and tracheomalacia even after anatomically intact primary repair.[3,9] During normal human embryogenesis between Carnegie stages 13 and 16 (weeks 4 to 6 post-conception), the common primitive foregut tube undergoes dorsoventral patterning driven by reciprocal epithelial-mesenchymal crosstalk: ventral endodermal expression of NKX2-1 (TTF-1) and mesenchymal Bone Morphogenetic Protein 4 (BMP4) specify tracheal cartilage and respiratory epithelium, whereas dorsal expression of SOX2 and the BMP-antagonist Noggin specify stratified squamous esophageal differentiation.[4,9] In Gross Type C EA/TEF, focal failure of lateral epithelial ridge fusion leaves the distal esophagus arising directly from the posterior membranous wall of the distal trachea or carina, while the blind upper esophageal pouch undergoes compensatory smooth-muscle hypertrophy due to fruitless in utero swallowing against an obstructed lumen.[12,18] Crucially, this developmental defect is accompanied by intrinsic enteric nervous system hypoplasia—characterized by deficient Auerbach's myenteric plexus ganglia and interstitial cells of Cajal along the distal esophageal segment—as well as structural widening of the posterior tracheal membrane with U-shaped rather than C-shaped hyaline cartilage rings.[8,16] Consequently, even after surgical division of the fistula and primary end-to-end anastomosis, intrinsic esophageal aperistalsis and dynamic expiratory tracheal collapse (tracheomalacia) persist as lifelong structural substrates requiring coordinated aerodigestive surveillance.[7,28]

Simultaneously, our findings expose a critical clinical limitation of relying solely on the classical Spitz prognostic classification in low- and middle-income neonatal intensive care settings.[22,38] Under the Spitz schema, Patient MRA was categorized into Group I based on a birth weight of 2,600 g (≥ 1,500 g) and the absence of major congenital heart disease on echocardiography (which showed only a physiological patent foramen ovale with normal biventricular contractility and a left aortic arch).[11,39] While Spitz Group I predicts a survival rate exceeding 95% in high-income cohorts where diagnosis occurs on Day 1 of life, recent analyses from the Turkish Esophageal Atresia Registry (Soyer et al., 2025), Japanese multicenter cohorts (Miyake et al., 2023), and LMIC referral hospitals (Moges et al., 2023; Raus et al., 2025) demonstrate that functional physiological reserve is governed not merely by absolute birth weight, but by intrauterine growth trajectory, gestational maturity, and preoperative septic-pneumonic burden.[17,18,34,38] In Patient MRA, chronic maternal second- and third-trimester urogenital infections and suboptimal antenatal nutrition produced Symmetric Intrauterine Growth Restriction (birth weight 2,600 g [< P_3], length 49.0 cm [< P_{10}], head circumference 34.0 cm [< P_{10}], Ponderal Index 2.21 g/cm^3 at 41 weeks gestation), which stripped the neonate of subcutaneous adipose and hepatic glycogen reserves prior to birth.[32,40] When compounded by a 7-day diagnostic delay and 400 g (−15.38%) acute postnatal weight loss to an admission weight of 2,200 g, the infant's effective metabolic and oncotic reserve resembled that of a low-birth-weight neonate, explaining the rapid onset of hypoalbuminemia (2.9 g/dL) and vitamin K-responsive coagulopathy (PT 12.1 s, APTT 31.0 s) and validating the necessity of preoperative nutritional rehabilitation before thoracotomy.[39,42]

Bioenergetics of Composite Lipid Parenteral Nutrition, Oncotic Albumin Rescue, and Staged Gastrostomy-to-Oral Enteral Transition

Restoring anabolic growth in a catabolic, late-referred neonate with uncorrected Gross Type C EA/TEF requires precise stoichiometric balancing of fluid, glucose, amino acid, and lipid substrates alongside restoration of plasma

colloid osmotic pressure.[25,26] At tertiary admission on June 16, 2025 (weight 2,200 g), Patient MRA was trapped in a hypercatabolic state driven by elevated work of breathing (respiratory rate 45 breaths/min), systemic pneumonic inflammation (febrile peak 38.0 °C), and 7 days of complete enteral deprivation. Initiating central Total Parenteral Nutrition via a left subclavian PICC line on Hospital Day 3 allowed delivery of hypertonic Dextrose N12.5% at 9.5 mL/h (GIR 8.2 mg/kg/min, later increased to N15% GIR 10.0 mg/kg/min during the 48-hour post-thoracotomy fasting window), crystalline neonatal amino acids (Aminoinfant 10% at 3.0 g/kg/day), and composite four-oil intravenous lipid emulsion (SMOFlipid 20% at 3.0 g/kg/day), supplying 84 to 110 kcal/kg/day at a nitrogen-to-non-protein calorie ratio of 1:152.[26,44] Maintaining an N/NPC ratio between 1:120 and 1:160 is biochemically essential in surgical neonates: if non-protein carbohydrate and lipid calories are insufficient, infused amino acids are oxidized via hepatic gluconeogenesis rather than incorporated into skeletal muscle and anastomotic collagen synthesis.[31,35] Furthermore, using SMOFlipid—which blends 30% soybean oil (essential linoleic and α-linolenic acids), 30% medium-chain triglycerides (rapid carnitine-independent β-oxidation), 25% olive oil (monounsaturated oleic acid resistant to lipid peroxidation), and 15% fish oil (anti-inflammatory ω-3 eicosapentaenoic and docosahexaenoic acids)—prevented parenteral nutrition-associated liver disease and accelerated resolution of the admission hyperbilirubinemia (4.5 mg/dL to normal) within the first week of care.[26,44]

Equally decisive was the prompt recognition and correction of preoperative hypoalbuminemia (2.9 g/dL) and mild hyponatremia (Na^+ 134 mmol/L) on Hospital Day 9 (June 24, 2025) prior to scheduling definitive thoracotomy.[11,36] In neonates with EA/TEF complicated by aspiration pneumonia and symmetric IUGR, serum albumin falls below 3.0 g/dL through two convergent mechanisms: diminished hepatic albumin gene transcription during acute-phase cytokine (IL-6 and TNF-α) prioritization of C-reactive protein and fibrinogen synthesis, and increased transcapillary escape of albumin into the pulmonary and splanchnic interstitium.[18,36] Uncorrected hypoalbuminemia reduces intravascular Starling colloid osmotic pressure, promoting submucosal edema at the esophageal ends that renders primary anastomotic sutures friable and prone to dehiscence.[19,36] By administering two targeted intravenous infusions of 20% human albumin (1.0 g/kg/dose) alongside 3.0 g/kg/day parenteral amino acids and initiating trophic gastrostomy enteral priming (8 × 2–3 mL/day of extensively hydrolyzed whey peptide formula [Pepti] and expressed breast milk), our protocol restored oncotic balance, stimulated gut mucosal glucagon-like peptide-2 (GLP-2) trophic signaling, and propelled somatic weight from the 2,200 g nadir to 2,700 g (+22.73%) before thoracotomy, 2,955 g (+34.32%; +29.04 g/day) at discharge, and 3,700 g (+68.18%) at 50 weeks PMA.[25,35]

Perioperative Antimicrobial Stewardship, Trans-Anastomotic Catheter Mechanics, and Long-Term Multidisciplinary Surveillance

The perioperative course of Patient MRA also offers important lessons regarding trans-anastomotic catheter management and proactive carbapenem stewardship in neonatal thoracic surgery.[21,22,45] During the July 2, 2025 right thoracotomy, a trans-anastomotic nasogastric catheter was placed across the end-to-end esophageal anastomosis to stent the lumen; however, on Postoperative Day 1 (July 3), infant agitation caused the catheter to fold in the oropharynx, requiring removal. Recent literature reflects lively debate over the risks and benefits of trans-anastomotic feeding tubes (TATs): while a 10-year retrospective cohort of 152 Type C EA repairs by Batikhe et al. (2025) found that TATs enabled earlier enteral feeding (median POD 3 vs. POD 13) and shorter TPN duration (6.6 vs. 16.2 days) without increasing anastomotic leaks (23.0% vs. 18.1%; p = 0.700), a prospective pilot randomized controlled trial of 53 Type C EA neonates by Bade et al. (2025) reported that avoiding indwelling TATs significantly reduced anastomotic leakage (0% vs. 20%; p = 0.03), lowered gastroesophageal reflux and stricture trends, and decreased brief resolved unexplained events (11.1% vs. 48.0%; p = 0.01) by eliminating mechanical foreign-body friction and lower esophageal sphincter stenting.[21,22] In Patient MRA, the presence of the Stage-1 Stamm gastrostomy tube provided an ideal synthesis of both strategies: removing the folded trans-anastomotic NGT on POD 1 relieved mechanical friction across the fresh anastomosis while the gastrostomy tube allowed safe enteral refeeding at 48 hours postoperatively (8 × 5 mL/day), culminating in rapid transition to full oral feeding (260–320 mL/day) and gastrostomy removal on Postoperative Day 7 (Hospital Day 24, July 9, 2025).[33,41]

Concurrently, the acute leukocyte surge to 26,920/μL (+108.68% above admission; +80.67% above immediate post-thoracotomy baseline; I/T ratio 0.02) on Postoperative Day 3 (July 5, 2025) illustrates the imperative of vigilant hematologic surveillance and rapid antimicrobial escalation after neonatal thoracotomy.[18,36] In retrospective audits of Type C EA repairs in LMIC tertiary centers, Manchanda et al. (2025) and Raus et al. (2025) demonstrated that uncontrolled perioperative sepsis is the single most lethal determinant of anastomotic breakdown and inpatient mortality.[18,36] Because neonates hospitalized in intensive care units for more than two weeks face high colonization rates with extended-spectrum β-lactamase (ESBL)-producing Enterobacterales and non-fermenting Gram-negative bacilli, the emergence of marked leukocytosis (26,920/μL) after 72 hours of perioperative cefoperazone-sulbactam prompted immediate switch to intravenous meropenem (3 × 110 mg ≈ 120 mg/kg/day) combined with amikacin.[45] As shown by Mariani et al. (2025), neonates exhibit a lower glomerular filtration rate (median eGFR 33 mL/min/1.73 m^2) than older infants, allowing high-dose meropenem regimens to achieve robust plasma trough concentrations (median C_{trough} 8.9 mg/L) that exceed the minimum inhibitory concentration (fT > MIC) of nosocomial Gram-negative pathogens, securing complete clinical resolution and sterile blood cultures in our patient.[45] Finally, structured long-term follow-up across pediatric surgery, gastroenterology, pulmonology, cardiology, and nutrition has been instituted to monitor for anastomotic stricture, gastroesophageal reflux, tracheomalacia, PFO closure at 3 months, and sustained neurodevelopmental and somatic catch-up growth.[7,15,30,35]

5. Conclusion

Staged surgical decompression and multidisciplinary nutritional optimization successfully rescued a late-referred (7-day-old) full-term male neonate presenting with Gross Type C (Vogt Type 3B) esophageal atresia with distal tracheoesophageal fistula, bilateral aspiration bronchopneumonia, Kramer Grade III hyperbilirubinemia, subclinical coagulopathy, and severe catabolic weight loss (−15.38%, nadir 2,200 g) superimposed on baseline symmetric intrauterine growth restriction (birth weight 2,600 g [< P_3], length 49.0 cm [< P_{10}], head circumference 34.0 cm [< P_{10}], Ponderal Index 2.21 g/cm^3; Spitz Group I).

Rather than performing an immediate high-risk thoracotomy during active pneumonia and acute starvation, instituting continuous proximal pouch suction, non-invasive nasal CPAP, Stage-1 Stamm gastrostomy decompression on Hospital Day 2, central PICC total parenteral nutrition with SMOFlipid 20% and Aminoinfant 10% (84 to 110 kcal/kg/day), targeted intravenous human albumin replacement (2 × 1.0 g/kg for serum albumin 2.9 g/dL), and trophic peptide formula priming restored coagulation homeostasis and increased somatic weight to 2,700 g (+22.73%) prior to Stage-2 right posterolateral thoracotomy, fistula ligation, and primary end-to-end esophageal anastomosis on Hospital Day 17. Rapid extubation within 24 hours, proactive carbapenem escalation (meropenem 3 × 110 mg IV) for Postoperative Day 3 leukocytosis (26,920/µL), early gastrostomy refeeding at 48 hours post-thoracotomy, transition to full oral feeding (260 to 320 mL/day) with gastrostomy removal on Hospital Day 24, and hospital discharge on Day 27 at 2,955 g (+34.32% above nadir; +29.04 g/day inpatient velocity) culminated in sustained outpatient Fenton catch-up growth to 3,700 g (+68.18% above nadir; +42.31% above birth weight) by 50 weeks postmenstrual age.

Declarations

Ethics approval and consent to participate

This observational analytic case study was formally reviewed and approved by the Central Medical & Health Committee (CMHC) Ethics Committee, Indonesia (Protocol Approval Number: 2025/0519) and conducted under the institutional clinical supervision of the Department of Child Health, Faculty of Medicine, Universitas Andalas, and Dr. M. Djamil General Hospital, Padang, Indonesia, in strict accordance with the Declaration of Helsinki. Written informed consent for clinical care, surgical procedures, and study participation was obtained from the patient's legal guardians (father Mr. DA and mother Ms. D).

Consent for publication

Written informed consent for the publication of anonymized clinical data, growth charts, radiographic babygrams, and intraoperative surgical photographs was formally signed by the patient's parents. All identifying patient names, medical record headers, and craniofacial/orbital borders on radiographs and growth charts were permanently masked with solid black anonymization bars in compliance with ICMJE guidelines.

Data availability

All longitudinal clinical, laboratory, radiographic, intraoperative, and anthropometric datasets generated and analyzed during this study are fully integrated within the manuscript, Tables 1–5, and Figures 1–5. De-identified source records are available from the corresponding author upon reasonable academic request.

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Conflict of interest

The authors declare that they have no commercial, financial, or personal competing interests.

Author contributions

DWK and EY: Conceptualization; Methodology; Investigation; Data curation; Formal analysis; Writing – original draft. BSP, FIH, and DN: Investigation; Validation; Writing – review and editing. The source states that all authors approved the submitted version; approval of this edited version remains pending.

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Use of artificial intelligence

The authors declare no generative artificial intelligence were used in preparation of this manuscript.

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