

CASE REPORT

Ketamine-Based Modified Rapid Sequence Induction and Torso-Ramped Airway Alignment in a Child with Giant Hydrocephalus, Obstructive Ileus, and Sepsis: An Observational Case Study

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ABSTRACT

Background. Giant hydrocephalus predicts a difficult airway and limited intracranial compliance. With obstructive ileus, sepsis and malnutrition, induction must protect cerebral perfusion, support the circulation and guard against aspiration.

Objective. To describe the anesthetic management, hemodynamic course and early outcome of one child with these conditions who underwent emergency laparotomy.

Methods. A 5-year-old boy (15 kg) had untreated communicating hydrocephalus (head circumference 81 cm), cerebral palsy, a four-day low small-bowel obstruction, sepsis physiology and dehydration. After resuscitation and nasogastric decompression, a modified rapid sequence induction used fentanyl 15 mcg, ketamine 7.5 mg (0.5 mg/kg) and rocuronium 8 mg (≈ 0.53 mg/kg), with gentle mask ventilation, torso ramping and a cuffed tube. Ventilation targeted an end-tidal carbon dioxide of 30–35 mmHg. Six epochs (T0–T5) were recorded. Shunting was deferred.

Results. Ramping allowed first-attempt cuffed intubation (Cormack-Lehane grade I). Mean arterial pressure rose from 60.0 to 65.0 mmHg after resuscitation and stayed at 65.3–70.7 mmHg without vasopressors. End-tidal carbon dioxide was 32–34 mmHg and lactate fell from 4.2 to 1.3 mmol/L. An adhesive band obstructing the terminal ileum was divided during a 2.5-hour laparotomy (blood loss 20 mL). The child went to intensive care intubated and sedated (Glasgow Coma Scale E2V(ett)M1) with no clinical sign of raised intracranial pressure. Follow-up beyond handover was not recorded.

Conclusion. In this child, a ketamine-based modified rapid sequence induction, torso ramping, normocapnic ventilation and deferral of the shunt were followed by hemodynamic stability and uncomplicated intubation. One case cannot show causation; intracranial pressure was not measured.

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1. Introduction

Hydrocephalus is a disorder of cerebrospinal fluid dynamics. Fluid accumulates inside the ventricles because outflow is structurally blocked, arachnoid granulations fail to reabsorb it, or production exceeds what the system can clear.¹ The distinction between non-communicating (obstructive) and communicating forms matters for both neurosurgical and anesthetic management. If communicating hydrocephalus is left untreated through early childhood, the ventricles keep enlarging, stretching the periventricular white matter tracts, thinning the cortical mantle and pushing outward on a cranial vault that has not yet fused.¹ A young child has a patent fontanelle and open metopic, sagittal and coronal sutures, so the skull grows along with the ventricles. Massive macrocephaly results, and it distorts craniofacial anatomy and upper airway mechanics.^{2,3} The expansion gives the brain temporary room. It shields the brain from sudden

intracranial hypertension, but only for a time, and the buffer is finite. Once the skull reaches its physical limit, intracranial compliance falls toward exhaustion.⁴ After that, small changes in cerebral blood volume, venous outflow or systemic carbon dioxide tension produce sharp rises in intracranial pressure. Little safe margin remains for cerebral perfusion.⁵⁻⁶

From the anesthetic point of view, a child with massive macrocephaly faces two opposing threats during emergency surgery. The first is mechanical. On a flat operating table the oversized occiput pushes the neck into involuntary hyperflexion, and the larynx sits behind the mandibular rim, the tongue falls back, and the oral, pharyngeal and laryngeal axes no longer line up.⁵ Clinical series of cranial dysraphism and giant encephaloceles report difficult tracheal intubation in approximately 48% to 50% of children with severe cranial-to-torso disproportion. Routine direct laryngoscopy can turn into an airway crisis.^{2,3,5} The second threat is hemodynamic. Cerebral perfusion pressure equals mean arterial pressure

minus intracranial pressure (CPP = MAP - ICP). Perfusion therefore falls when intracranial pressure rises or when systemic mean arterial pressure drops. Neuroanesthetic practice generally favors propofol for induction, since it lowers cerebral metabolic rate for oxygen and intracranial pressure.⁶ Propofol also dilates peripheral vessels and depresses the myocardium. In a patient with limited cardiovascular reserve, the resulting hypotension can cut cerebral blood flow.

The picture becomes harder when massive hydrocephalus coexists with acute obstructive ileus, bacterial sepsis and severe acute malnutrition. Fecal stasis, vomiting and diaphragmatic splinting from a distended abdomen create an imminent risk of pulmonary aspiration. That risk is the classic indication for rapid sequence induction and tracheal intubation.⁷ The technique is simple. The induction agent and muscle relaxant are given in quick succession, and the child is intubated at once without bag-mask ventilation.⁷ A malnourished, septic child has high metabolic demand and a reduced functional residual capacity. Standard apneic induction in such a child can cause arterial desaturation within seconds, and the hypoxemia, sympathetic stimulation and reactive hypercapnia that follow dilate the cerebral vessels and can drive intracranial pressure up uncontrollably.^{7,8} Abdominal sepsis adds an uncontrolled inflammatory response, with endothelial barrier breakdown, nitric oxide-mediated vasoplegia and absolute hypovolemia from fluid sequestered in the bowel lumen.^{9,10} A vasodilating induction agent such as propofol can cause immediate cardiovascular collapse in a child in this state. Mean arterial pressure falls below the autoregulatory threshold. Ischemic secondary brain injury follows.⁸

Severe acute malnutrition of the marasmic type compounds these conflicts, and it depletes hepatic glycogen, breaks down skeletal muscle, blunts autonomic cardiovascular reflexes and reduces tolerance of pharmacological stress and hypothermia. When several emergencies meet in one child, standard algorithms fail because guideline-directed care for one problem worsens another. The pediatric literature on hydrocephalus focuses almost entirely on early ventriculoperitoneal shunt insertion. That approach has a cost here. Placing a silicone shunt into an obstructed, distended and bacterially contaminated peritoneal cavity invites shunt colonization, ascending ventriculitis and fatal neurosurgical infection. An integrated strategy may therefore be needed. It would reorder surgical priorities, use positioning designed for the airway anatomy, replace conventional induction agents with a hemodynamic-preserving regimen and keep ventilation strictly normocapnic. Reports of anesthesia for this combination in one patient are few in the sources we

located. The objective of this case report is to describe the anesthetic management, hemodynamic course and early outcome of a child with severe communicating hydrocephalus, a predicted difficult airway, obstructive ileus, abdominal sepsis and marasmus who underwent emergency exploratory laparotomy under a ketamine-based modified rapid sequence induction with torso-ramped positioning.

2. Methods

2.1. Report design and reporting guideline

This is a single case report of one child. It is reported in accordance with the CARE guideline¹¹. Physiological, hemodynamic, ventilatory and neurological values were recorded at six standardized epochs.

2.2. Setting

Care was delivered in the Department of Anesthesiology and Intensive Care Therapy at Dr. Moewardi Regional General Hospital, in collaboration with the Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Central Java, Indonesia. The hospital is a tertiary academic referral center that handles complex pediatric neurosurgical, neonatal surgical and critical care emergencies from across Central Java. The case reflects the meeting of chronic, neglected congenital neurodevelopmental disease with an acute, life-threatening surgical emergency in a child in a resource-limited setting. The anesthetic plan was individualized and was designed to manage colliding physiological vulnerabilities without causing secondary multi-organ injury.

2.3. Patient information and case selection

The patient was a 5-year-old boy with spastic quadriplegic cerebral palsy and congenital hydrocephalus diagnosed in infancy. Neurosurgical follow-up had lapsed, and no cerebrospinal fluid diversion had ever been performed. He presented with four days of obstipation, progressive abdominal bloating, intractable vomiting and marked oliguria. The case was selected for reporting because severe hydrocephalus with a giant head, obstructive ileus, sepsis and marasmus coincided in one child needing emergency surgery, which set the airway, intracranial and hemodynamic aims against each other. Written informed consent was obtained (section 2.8).

2.4. Case timeline

Table 1 sets out the chronology from the presentation to the handover to intensive care. Epochs T0–T5 are defined in section 2.7.

Table 1. Case timeline in relative time, from presentation to PICU handover.

Time point	Clinical event or intervention	Key findings
Months before admission	Last neurosurgical follow-up; congenital hydrocephalus diagnosed in infancy	No cerebrospinal fluid diversion ever performed; spastic quadriplegic cerebral palsy
Day -4	Onset of obstipation, abdominal bloating, vomiting, oliguria	—
Day 0, T0	Emergency department presentation	GCS E4M4V2; head circumference 81 cm; BP 80/50 mmHg (MAP 60.0), HR 157/min, temperature 38.5 °C, SpO ₂ 97% (room air); lactate 4.2 mmol/L; WBC 18,600/μL; step-ladder air-fluid levels on abdominal radiographs; head MSCT: communicating hydrocephalus, corpus callosum agenesis
Day 0, T0 to T1	Resuscitation and gastric decompression	Ringer's lactate 250 mL, ceftriaxone, metronidazole; nasogastric tube drained 80 mL
Day 0, T1	Transfer to operating table, torso-ramped position	BP 85/55 mmHg (MAP 65.0), HR 118/min; lactate 2.8 mmol/L; SpO ₂ 94% (room air), 99% after pre-oxygenation
Day 0, T2	Modified rapid sequence induction and intubation	Fentanyl 15 mcg, ketamine 7.5 mg, rocuronium 8 mg; Cormack-Lehane grade I; 4.5 mm cuffed tube at 12 cm; MAP 65.3 mmHg; EtCO ₂ 33 mmHg
Day 0, T3	Incision and peritoneal entry	MAP 70.7 mmHg; EtCO ₂ 32 mmHg; lactate 1.8 mmol/L; 150 mL non-purulent peritoneal fluid
Day 0, T4	Band division, decompression and lavage	Adhesive band 15 cm proximal to the ileocecal valve; bowel viable; MAP 67.3 mmHg; EtCO ₂ 34 mmHg; lactate 1.5 mmol/L; operation 2.5 h, blood loss 20 mL
Day 0, T5	Handover to the PICU, intubated	GCS E2V(ett)M1 (sedated); pupils 3 mm, reactive; MAP 65.7 mmHg; lactate 1.3 mmol/L; metamizole analgesia
After T5	Course after PICU admission	Extubation, PICU stay, shunt surgery, discharge and last follow-up are not given in the submitted record

Notes: BP, blood pressure; EtCO₂, end-tidal carbon dioxide; GCS, Glasgow Coma Scale; HR, heart rate; MAP, mean arterial pressure; MSCT, multislice computed tomography; PICU, pediatric intensive care unit; WBC, white blood cell count. Epochs T0–T5 are defined in section 2.7.

2.5. Clinical findings and diagnostic assessment

The diagnoses were established on emergency department admission from clinical, anthropometric, radiological and physiological findings. Severe communicating hydrocephalus was diagnosed on non-contrast multislice computed tomography (MSCT) of the head. The scan showed marked symmetrical dilatation of the whole ventricular system, patent outflow pathways of the fourth ventricle, severe thinning of the cerebral mantle, a persistently patent anterior fontanelle and complete diastasis of the metopic, sagittal and coronal sutures. It also showed agenesis of the corpus callosum. Acute obstructive ileus was confirmed on three-position plain abdominal radiographs. These showed more than three long air-fluid levels in a step-ladder pattern, prominent small-bowel distension and no rectal air. Sepsis of abdominal origin was diagnosed by international pediatric consensus criteria. The child had a core temperature of 38.5 °C, a resting sinus tachycardia of 157 beats/min, hypotension of 80/50 mmHg (mean arterial pressure 60.0 mmHg), a white blood cell count of 18,600 /μL, a lactate of 4.2 mmol/L and a capillary refill time of 3 seconds. Marasmic malnutrition was classified against World Health Organization anthropometric thresholds. Body weight was 15 kg and height 93 cm at 5 years of age, which the authors report as a weight-for-height Z-score below -3 standard deviations. The child had visible muscle wasting and loss of subcutaneous fat, and no nutritional edema.

The patient was monitored continuously. Monitoring included arterial blood pressure, continuous lead II/V5 electrocardiography, pulse oximetry (SpO₂), dual-wavelength side-stream capnography for end-tidal carbon dioxide (EtCO₂), core nasopharyngeal temperature, urine output through an indwelling catheter and serial arterial blood gas analyses. Neurological monitoring consisted of serial Glasgow Coma Scale assessments, bilateral pupillary light reflex testing and palpation of fontanelle tension.

2.6. Therapeutic intervention

Three planning axes guided physiological decisions through the perioperative course. The first weighed defense of cerebral perfusion pressure against support of

systemic vascular resistance. Surgical urgency ruled out direct invasive intracranial pressure monitoring, so clinical and ventilatory surrogates were used instead. Mean arterial pressure was held strictly between 65 and 75 mmHg, and end-tidal carbon dioxide stayed inside a narrow normocapnic window of 30 to 35 mmHg. This was meant to prevent hypercapnia-mediated cerebral hyperemia without causing hypocapnic cerebral ischemia. The second axis was the airway. Torso-elevation ramping was planned to correct the flexion expected in the usual flat supine position, and the Cormack-Lehane glottic view grade and the number of intubation attempts were recorded.¹² The third axis was surgical sequencing, which ranked the pathologies by reversibility. Emergency abdominal exploration came first, to prevent bowel necrosis, bacterial translocation and septic death. Permanent ventriculoperitoneal shunt implantation was deliberately deferred to protect the central nervous system from ascending peritoneal contamination.¹³

Physiological targets were set in advance from pediatric neurocritical care and septic shock guidelines.⁹ Mean arterial pressure was targeted at ≥65 mmHg for the acutely compromised brain of a 5-year-old under general anesthesia.¹⁴ Ventilatory targets were pressure-controlled ventilation with inspiratory pressure ≤15 cmH₂O above a positive end-expiratory pressure of 5 cmH₂O (driving pressure 10 cmH₂O). The fraction of inspired oxygen (FiO₂) was titrated to keep SpO₂ between 96% and 98%. Hyperventilation (EtCO₂ < 30 mmHg) was avoided to prevent marked cerebral vasoconstriction, and hypercapnia (EtCO₂ > 35 mmHg) was actively corrected to prevent intracranial hypertension.¹⁵ Fluid administration followed a balanced strategy. The aim was enough volume to maintain microvascular perfusion without a large positive balance that would worsen cerebral edema or add interstitial pulmonary water in a malnourished child.^{9,16}

The drugs, doses and techniques actually used are described in section 3.3. The airway cart, the induction sequence, the cuffed tube and the ventilator settings were agreed by the anesthesiology, pediatric surgery and pediatric intensive care teams before induction.

2.7. Outcome measures and follow-up

The patient was assessed at six standardized perioperative epochs. Epoch T0 was emergency department presentation before resuscitation. Epoch T1 was the end of initial volume resuscitation and transfer to the operating table in the torso-ramped position. Epoch T2 was the period just after modified rapid sequence induction and tracheal intubation. Epoch T3 was surgical incision and peritoneal entry during exploratory laparotomy. Epoch T4 was decompression of the obstructed small bowel, release of internal adhesions and extensive warm peritoneal lavage. Epoch T5 was handover and initial stabilization on invasive mechanical ventilation in the Pediatric Intensive Care Unit (PICU). Outcomes were hemodynamic stability, first-attempt intubation, glottic view, end-tidal carbon dioxide, lactate, laboratory trends and neurological signs.

Because this is a single patient ($n = 1$), inferential testing (p-values, t-tests, analysis of variance, chi-square) was not used, as it would be invalid for one observational trajectory. The analysis was descriptive and longitudinal. Serial percentage changes between epochs were calculated as $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$, where T0 is baseline admission and Tn is a later timepoint; absolute changes are given for temperature. The trajectories were read against age-specific physiological and laboratory reference intervals from the clinical pathology laboratory of Dr. Moewardi Regional General Hospital. No causality scale was applied.

2.8. Ethics and consent

The clinical protocol, the observation of the patient and the publication of anonymized patient data were reviewed by CMHC Research Center Ethics Committee, Indonesia, which granted ethical clearance (Protocol Approval Number: 2026/5194). Review followed the institutional standards of Dr. Moewardi Regional General Hospital and the World Medical Association Declaration of Helsinki (amended Fortaleza, Brazil, 2013). Before anesthesia and surgery, the patient's legal guardians (parents) gave written informed consent after counseling. The discussion covered the anticipated difficult airway, the very high perioperative mortality risk of septic shock with untreated hydrocephalus, the pharmacologic reasons for choosing

off-label ketamine, the decision to put exploratory laparotomy ahead of shunt surgery, and the planned postoperative intensive care admission. The parents also gave explicit written authorization for the scientific analysis and dissemination of anonymized clinical data, serial laboratory results, radiological images and intraoperative photographs. The eyes were permanently masked in all visual material so that the patient could not be identified, in line with International Committee of Medical Journal Editors guidelines.

3. Results

3.1. Presentation and baseline findings

The patient was a 5-year-old boy who came to the emergency department with four days of obstipation, progressive abdominal bloating, intractable vomiting and marked oliguria. He had a documented history of spastic quadriplegic cerebral palsy and congenital hydrocephalus diagnosed in infancy. No cerebrospinal fluid diversion had ever been performed. At admission (Epoch T0) his metabolic and neurological state was poor, with a baseline Glasgow Coma Scale of E4M4V2 (total 10/15). Head circumference was 81 cm, above the +3 standard deviation threshold for age. He had prominent frontal bossing, widened cranial sutures, a tense anterior fontanelle and a bilateral sunset-eye sign, which indicates chronically elevated intracranial pressure. The vital signs showed decompensated septic physiology. Blood pressure was 80/50 mmHg (mean arterial pressure 60.0 mmHg), heart rate 157 beats/min, respiratory rate 28 breaths/min, core temperature 38.5 °C and peripheral oxygen saturation 97% on room air. The extremities were cool. Capillary refill time was prolonged at 3 seconds. The abdomen was severely distended and tympanic, with hypoactive bowel sounds and shifting dullness. Body weight was 15 kg and length 93 cm. These findings, together with loss of buccal and mesenteric adipose tissue, were taken to indicate severe acute malnutrition of the marasmic subtype. Table 2 gives the baseline characteristics and risk stratification, and Table 1 gives the timeline. Figure 1 shows the disproportion between the macrocephalic head, the wasted thorax and the grossly distended abdomen.

Table 2. Baseline clinical, anthropometric, laboratory and radiological characteristics at admission.

Parameter category	Clinical variable	Admission value or finding	Reference interval	Interpretation and risk impact
Demographics	Age / Biological Sex	5 years / Male	Pediatric	Vulnerable young child with multi-organ physiological fragility
Anthropometry	Weight / Height	15 kg / 93 cm	Expected: 18–20 kg / 105–110 cm	Reported as marasmic severe acute malnutrition
Baseline Neurology	Congenital Hydrocephalus	Severe communicating hydrocephalus	Intact CSF dynamics	Untreated, non-shunted; massive ventricular enlargement & open sutures
Baseline Neurology	Structural Brain Anomalies	Agenesis of the corpus callosum	Intact commissure	Confirms congenital chronicity; absent signs of acute brain herniation
Neurological State	Glasgow Coma Scale (GCS)	E4M4V2 (Total: 10/15)	15/15	Impaired sensorium (septic encephalopathy and raised ICP suspected; ICP not measured)
Cranial Morphology	Head Circumference	81 cm (Z-score > +3 SD; Figure 1 legend: > +9.8)	49–52 cm (5 years)	Severe macrocephaly; prominent occiput forces extreme cervical flexion
Ocular Signs	Sunset-Eye Sign	Bilateral positive	Absent	Sign of raised intracranial pressure
Hemodynamics	Blood Pressure (BP)	80/50 mmHg (MAP 60.0 mmHg)	95–110 / 55–70 mmHg	Hypotension; septic vasoplegia compounded by severe dehydration
Hemodynamics	Heart Rate (HR) / Rhythm	157 beats/min / Sinus tachycardia	70–110 beats/min	Compensatory tachycardia in response to hypovolemia and sepsis
Respiration	Respiratory Rate / SpO ₂	28 breaths/min / 97% (room air)	18–24 / ≥95%	Tachypneic; diaphragmatic splinting from gross abdominal distension
Thermoregulation	Body Temperature	38.5 °C (Febrile)	36.5–37.5 °C	Systemic inflammatory response syndrome (SIRS) of abdominal origin
Peripheral Perfusion	Capillary Refill Time (CRT)	3 s; cool extremities	< 2 s; warm extremities	Delayed peripheral perfusion indicative of septic hypovolemia
Gastrointestinal	Abdominal Distension / Bowel	Grossly distended; hypoactive sounds	Soft, non-distended	Low small-bowel obstruction with third-space intraluminal fluid accumulation
Gastrointestinal	Obstipation / Emesis	4-day obstipation; recurrent vomiting	Daily normal bowel habits	Obstructive ileus; extreme aspiration risk mandating full-stomach RSI
Hydration Status	Clinical Dehydration	Severe dehydration; oliguria	Euhydrated; normal turgor	Sluggish skin turgor, dry mucous membranes, scant urine output
Hematology	White Blood Cell (WBC) Count	18,600 /μL (1.86×10^4 /μL)	5,000–14,500 /μL	Marked leukocytosis with neutrophilic shift; systemic bacterial sepsis
Hematology	Hemoglobin / Hematocrit	11.2 g/dL / 33.8%	11.5–15.5 g/dL / 34–40%	Possible relative hemoconcentration with chronic nutritional anemia
Hematology	Platelet Count	320,000 /μL (3.20×10^5 /μL)	150,000–450,000 /μL	Preserved thrombocyte count; adequate for surgical hemostasis
Metabolic Profile	Serum Lactate	4.2 mmol/L	0.5–2.0 mmol/L	Lactic acidosis reflecting tissue hypoperfusion and anaerobic metabolism
Metabolic Profile	Random Blood Glucose	68 mg/dL (3.77 mmol/L)	70–100 mg/dL	Borderline low glucose; depleted glycogen stores suspected
Renal Function	Blood Urea Nitrogen / Creatinine	28 mg/dL / 0.62 mg/dL	5–18 / 0.3–0.7 mg/dL	Prerenal azotemia with elevated BUN:creatinine ratio secondary to dehydration
Coagulation Profile	PT / INR / aPTT	13.4 s / 1.12 / 32.8 s	11–14 s / 0.8–1.2 / 25–35 s	Normal coagulation reserve; absence of overt consumption coagulopathy
Abdominal Imaging	Abdominal Radiography (3 views)	Multiple long air-fluid levels (>3)	Normal bowel gas pattern	Typical step-ladder pattern; low small-bowel obstruction without perforation
Neuroimaging	Non-contrast Head MSCT	Severe communicating hydrocephalus	Normal ventricles	Massive ventricular dilatation, open anterior fontanelle, metopic/sagittal sutures open
Anesthetic Risk	ASA Physical Status Classification	Class III-E (Emergency)	Class I–VI	Reported as ASA III-E; severe systemic disease with neuro-septic emergencies

Notes: Reference intervals are those of the Dr. Moewardi Regional General Hospital pediatric laboratory and radiology service. ASA, American Society of Anesthesiologists; BUN, blood urea nitrogen; CRT, capillary refill time; GCS, Glasgow Coma Scale; ICP, intracranial pressure; MAP, mean arterial pressure; MSCT, multislice computed tomography; PT, prothrombin time; aPTT, activated partial thromboplastin time; RSI, rapid sequence induction; SpO₂, peripheral oxygen saturation.



Figure 1. Bedside photograph of the patient at presentation, showing the disproportion between the enlarged neurocranium (head circumference 81 cm), the wasted thorax and the distended abdomen. The eyes are masked to prevent identification.

Admission laboratory tests were consistent with systemic bacterial sepsis, metabolic decompensation and prerenal azotemia. The complete blood count showed severe leukocytosis. The white blood cell count was 18,600 / μ L (1.86×10^4 / μ L), with a prominent neutrophilic left shift (82% absolute neutrophils). Hemoglobin was 11.2 g/dL and hematocrit 33.8%, which suggested relative hemoconcentration on top of chronic nutritional anemia. Platelets were preserved at 320,000 / μ L (3.20×10^5 / μ L). Arterial blood gas analysis on room air showed uncompensated metabolic acidosis: pH 7.28, PaCO₂ 31.2 mmHg, PaO₂ 82.5 mmHg, serum bicarbonate 14.8 mEq/L and base excess -9.4 mEq/L. Serum lactate was clearly elevated at 4.2 mmol/L (reference interval 0.5–2.0 mmol/L), which indicated systemic microvascular hypoperfusion and anaerobic glycolysis. Renal function showed acute prerenal azotemia, with blood urea nitrogen 28 mg/dL and serum creatinine 0.62 mg/dL (BUN:creatinine ratio >40). Serum sodium was 134 mEq/L, potassium 4.1 mEq/L, chloride 98 mEq/L and ionized calcium 1.12 mmol/L. Random blood glucose was borderline low at 68 mg/dL (3.77 mmol/L), which was attributed to exhausted hepatic glycogen stores. Baseline coagulation parameters were normal (prothrombin time 13.4 seconds, INR 1.12, activated partial thromboplastin time 32.8 seconds).

3.2. Diagnostic findings

Imaging showed the anatomical basis of both emergencies. Non-contrast multislice computed tomography of the head

showed massive, symmetrical dilatation of the lateral and third ventricles. They communicated freely through the aqueduct of Sylvius with an expanded fourth ventricle, which confirmed severe communicating hydrocephalus. The cortical mantle was compressed to a thin peripheral rim of less than 4 mm over the frontoparietal convexities. The corpus callosum was completely absent, with no normal commissural fibers and a parallel orientation of the lateral ventricular bodies. The calvarium was greatly enlarged. The anterior fontanelle was widely patent (5.2 cm $\times$ 4.8 cm), and the metopic, sagittal and coronal sutures were unossified. The lateral scout view in Figure 2 showed extreme craniofacial disproportion, with a cranial vault volume exceeding facial and mandibular dimensions by a ratio greater than 4:1. This large occipital overhang meant that a conventional supine position would force the cervical spine into more than 45° of involuntary flexion. The larynx would sit beneath the thoracic inlet, and line-of-sight laryngoscopy would be expected to be impossible. Abdominal plain radiographs were taken supine, erect and in left lateral decubitus. They showed multiple long air-fluid levels, more than three in number, in a classic step-ladder pattern across the central and lower abdomen. This confirmed low small-bowel obstruction with massive intraluminal fluid and gas sequestration. No subdiaphragmatic pneumoperitoneum was seen.

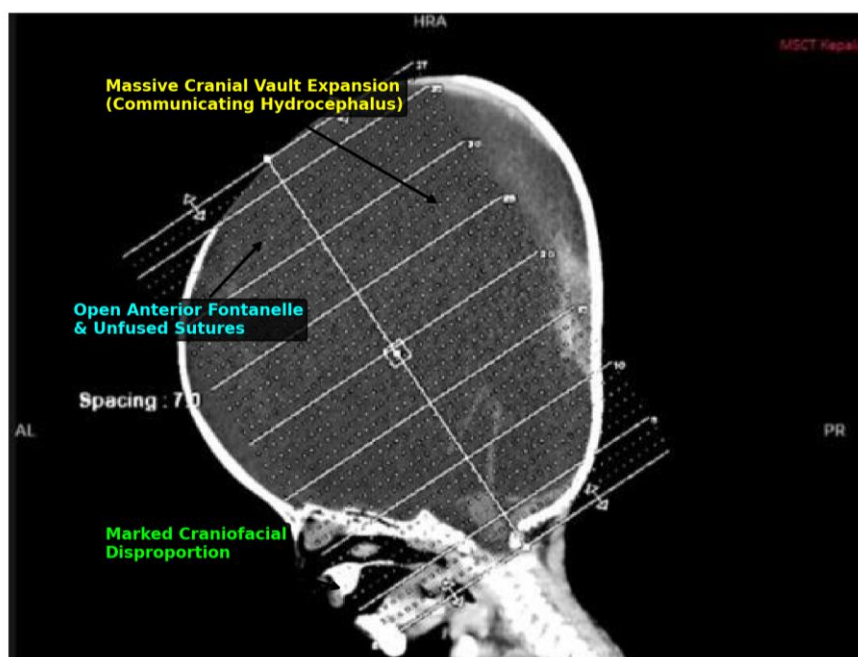


Figure 2. Lateral scout view from the head CT, showing the enlarged cranial vault and craniofacial disproportion.

3.3. Treatment course and outcome

A multidisciplinary team of pediatric intensivists, pediatric surgeons and anesthesiologists began stabilization in the emergency department. Intravenous access was placed. Resuscitation used warm balanced crystalloid (Ringer's lactate) in titrated aliquots of 10 mL/kg over 60 minutes, for a total pre-induction resuscitation volume of 250 mL. Broad-spectrum antimicrobial therapy started with intravenous ceftriaxone (50 mg/kg) and metronidazole (7.5 mg/kg). A 10 French nasogastric tube was inserted and passively drained 80 mL of dark, bilious, foul-smelling gastric contents, after which continuous low-pressure suction was applied. After volume expansion (Epoch T1) the hemodynamic indices improved. Blood pressure rose to 85/55 mmHg (mean arterial pressure 65.0 mmHg, a +8.3% increase from T0), and heart rate fell from 157 to 118 beats/min (a -24.8% reduction). The extremities became warm, capillary refill time improved to 2.5 seconds, and serum lactate fell to 2.8 mmol/L (a -33.3%

clearance). Room air SpO₂ was 94% and rose to 99% with pre-oxygenation with 100% oxygen through a tight-fitting face mask.

After transfer to the operating table, the predicted difficult airway was handled through positioning. Figure 3 shows the setup. On a flat horizontal table the 81 cm occiput would have been expected to push the head into acute cervical flexion. Instead, folded towels and silicone ramps were built up beneath the thoracic cage and shoulders, and the torso was raised by approximately 8 cm until the anterior plane of the chest lay flush with the enlarged occipital prominence. This restored a neutral alignment of the oral, pharyngeal and laryngeal axes. On external check, the external auditory meatus lay horizontal with the anterior clavicular notch, and the submandibular space was open. A difficult airway cart was placed at the bedside. It held a pediatric video laryngoscope (blade sizes 1.5 and 2), a flexible fiberoptic bronchoscope, pediatric bougies and second-generation supraglottic airway rescue devices.



Figure 3. Intraoperative torso-elevation ramp with the cuffed endotracheal tube secured. Folded towels and silicone ramps beneath the shoulders and upper back raise the torso relative to the occiput. The eyes are masked to prevent identification.

General anesthesia was induced with a modified rapid sequence regimen chosen for this child. After three minutes of pre-oxygenation with 100% oxygen and repeat aspiration of the nasogastric tube, the drugs were given in sequence. Fentanyl 15 mcg (1 mcg/kg) blunted the sympathetic hypertensive and intracranial pressor response to airway instrumentation. Dexamethasone 1.5 mg (0.1 mg/kg) and ondansetron 1.5 mg (0.1 mg/kg) were given as antiemetic and anti-edema prophylaxis. Ketamine 7.5 mg (0.5 mg/kg) was the primary induction agent, chosen to stimulate central sympathetic tone and defend systemic vascular resistance. Rocuronium 8 mg ($\approx$ 0.53 mg/kg) was chosen for neuromuscular blockade to avoid the hyperkalemic risk attributed to succinylcholine in cerebral palsy. Complete apnea was not enforced. Gentle low-pressure mask ventilation was given instead (peak airway pressure strictly limited to <12 cmH₂O). Its purpose was to prevent arterial desaturation in this malnourished, hypermetabolic child without insufflating the stomach. Laryngoscopy was performed 60 seconds after rocuronium with a pediatric Macintosh blade. Torso

ramping gave a Cormack-Lehane Grade I view. A 4.5 mm internal-diameter cuffed endotracheal tube was passed atraumatically at the first attempt. It was secured at a depth of 12 cm at the alveolar ridge, with the cuff inflated to 18 cmH₂O. Bilateral breath sounds were equal. Capnography tracings were clean.

After tracheal intubation (Epoch T2), hemodynamics were stable. Blood pressure was 84/56 mmHg (mean arterial pressure 65.3 mmHg, a +8.8% increase from baseline), heart rate was 108 beats/min (a -31.2% reduction from baseline) and SpO₂ was 98% on FiO₂ 0.50. No hypotension was recorded after induction, and no emergency bolus of a vasopressor or inotrope was needed. The operation lasted 2.5 hours (Epochs T3 and T4). Mean arterial pressure stayed between 65.0 and 70.7 mmHg. Systolic blood pressure ranged from 84 to 88 mmHg and diastolic blood pressure from 56 to 62 mmHg, and heart rate stayed between 102 and 108 beats/min. Table 3 gives the physiological parameters and percentage changes at every epoch, and Figure 4 shows the trajectories.

Table 3. Physiological, hemodynamic and ventilatory values across the six perioperative epochs.

Parameter	T0 Admission	T1 Pre-induction (ramped)	T2 Post-induction	T3 Incision	T4 Decompression	T5 PICU handover	Change T5 vs T0
Systolic Blood Pressure (mmHg)	80	85 (+6.3%)	84 (+5.0%)	88 (+10.0%)	86 (+7.5%)	85 (+6.3%)	+6.3%
Diastolic Blood Pressure (mmHg)	50	55 (+10.0%)	56 (+12.0%)	62 (+24.0%)	58 (+16.0%)	56 (+12.0%)	+12.0%
Mean Arterial Pressure (MAP, mmHg)	60.0	65.0 (+8.3%)	65.3 (+8.8%)	70.7 (+17.8%)	67.3 (+12.2%)	65.7 (+9.5%)	+9.5%
Heart Rate (beats/min)	157	118 (-24.8%)	108 (-31.2%)	105 (-33.1%)	102 (-35.0%)	104 (-33.8%)	-33.8%
Respiratory Rate (breaths/min)	28	23 (-17.9%)	22 (Controlled)	22 (Controlled)	22 (Controlled)	22 (Controlled)	-21.4%
Oxygen Saturation (SpO ₂ , %)	97 (Room air)	94 (Room air)	98 (FiO ₂ 0.5)	97 (FiO ₂ 0.5)	98 (FiO ₂ 0.5)	98 (FiO ₂ 0.5)	+1.0%; T1 SpO ₂ 94% on room air
End-Tidal CO ₂ (EtCO ₂ , mmHg)	Unmeasured	Unmeasured	33	32	34	32	Target 30–35 mmHg sustained
Inspiratory Pressure (P _{insp} , cmH ₂ O)	Unventilated	Unventilated	15	15	15	8 (Assist-control)	Low driving pressure preserved
Positive End-Expiratory Pressure (cmH ₂ O)	0	0	5	5	5	5	Lung-protective baseline
Glasgow Coma Scale (Total /15)	10 (E4M4V2)	10 (E4M4V2)	Sedated / Paralyzed	Sedated / Paralyzed	Sedated / Paralyzed	E2V(ett)M1	Sedated and paralyzed during surgery
Capillary Refill Time (seconds)	3.0	2.5 (-16.7%)	2.0 (-33.3%)	2.0 (-33.3%)	2.0 (-33.3%)	2.0 (-33.3%)	-33.3%
Core Body Temperature (°C)	38.5	37.8 (-0.7 °C)	37.2 (-1.3 °C)	36.9 (-1.6 °C)	36.8 (-1.7 °C)	36.8 (-1.7 °C)	-1.7 °C
Serum Lactate (mmol/L)	4.2	2.8 (-33.3%)	2.1 (-50.0%)	1.8 (-57.1%)	1.5 (-64.3%)	1.3 (-69.0%)	-69.0%
Random Blood Glucose (mg/dL)	68	88 (+29.4%)	102 (+50.0%)	110 (+61.8%)	105 (+54.4%)	98 (+44.1%)	+44.1%
Cumulative Fluid Balance (mL)	0	+250 (Resuscitation)	+260	+280	+300	+311 (+61 intraop net)	+311 mL (+250 resuscitation, +61 intraoperative as recorded)

Notes: Percent change = $[(T_n - T_0) / T_0] \times 100\%$; absolute change is given for temperature. MAP was derived as $(SBP + 2 \times DBP) / 3$ in all epochs. CPP, cerebral perfusion pressure; EtCO₂, end-tidal carbon dioxide; FiO₂, fraction of inspired oxygen; MAP, mean arterial pressure; PICU, pediatric intensive care unit; SpO₂, peripheral oxygen saturation.

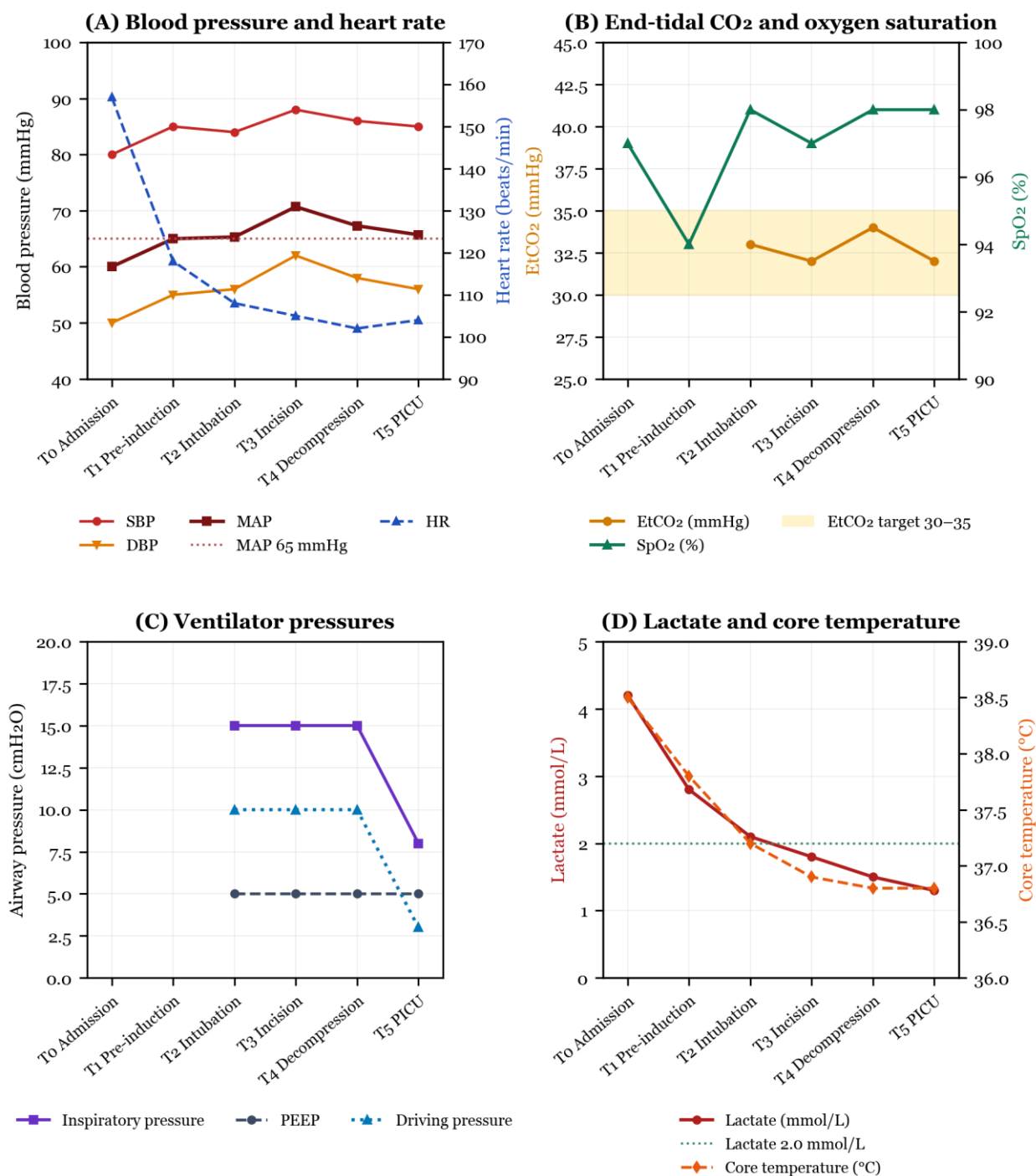


Figure 4. Perioperative trajectories across the six epochs, plotted from Table 3. (A) Systolic, diastolic and mean arterial pressure with the 65 mmHg target, and heart rate. (B) End-tidal CO₂ with the 30–35 mmHg target band, and SpO₂. (C) Inspiratory pressure, PEEP and driving pressure of the ventilator. (D) Serum lactate with the 2.0 mmol/L reference limit, and core temperature. T0, admission; T1, pre-induction (ramped, after resuscitation); T2, intubation; T3, incision; T4, decompression and lavage; T5, PICU handover. EtCO₂, end-tidal carbon dioxide; PEEP, positive end-expiratory pressure; PICU, pediatric intensive care unit.

Mechanical ventilation was pressure-controlled, with an inspiratory pressure of 15 cmH₂O, positive end-expiratory pressure of 5 cmH₂O, a respiratory rate of 22 breaths/min and FiO₂ of 0.50. These settings delivered a tidal volume of 90 to 95 mL (6.0–6.3 mL/kg). Dual-wavelength capnography showed end-tidal carbon dioxide between 32 and 34 mmHg throughout (Epoch T2: 33 mmHg; Epoch T3: 32 mmHg; Epoch T4: 34 mmHg). An arterial blood gas at the intraoperative midpoint (Epoch T3) agreed closely with the capnography. It gave PaCO₂ 33.8 mmHg, PaO₂ 138 mmHg, pH 7.36, serum bicarbonate 20.2 mEq/L, base excess -3.2 mEq/L, and lactate 1.8 mmol/L. The aim of

holding low-normal normocapnia was to avoid both vasodilatory cerebral swelling and ischemic cerebral vasoconstriction.

Emergency exploratory laparotomy was done through a midline abdominal incision. About 150 mL of clear, yellowish, non-purulent reactive peritoneal fluid was drained on entry into the peritoneal cavity. The surgeons found a low small-bowel obstruction in the distal ileum. An acute fibrous adhesive band constricted the terminal ileum approximately 15 cm proximal to the ileocecal valve. The jejunum and ileum above it were markedly distended, and the distal colon was collapsed. The band was divided

sharply, which relieved the mechanical obstruction. The bowel wall was hyperemic and edematous but regained peristalsis and mucosal pinkness at once on decompression. This confirmed full vascular viability, with no transmural necrosis or perforation. The peritoneum was lavaged thoroughly with 500 mL of warm sterile normal saline. The ventriculoperitoneal shunt was deferred.

Total operative time was 2.5 hours. Fluid management was kept restrictive to limit overload and cerebral edema while replacing ongoing losses. Intravenous maintenance and

replacement consisted of 100 mL of 1% dextrose in balanced electrolyte solution and 9.8 mL of drug carrier volume. Intraoperative output was an estimated surgical blood loss of 20 mL, urine output of 15 mL (0.4 mL/kg/h) and nasogastric aspirate of 5 mL. Total intraoperative intake was 109.8 mL against a total output of 40 mL, which left a net positive balance of +69.8 mL (recorded as +61 mL in Table 3). Blood glucose rose from 68 mg/dL at admission to 88 mg/dL before surgery, 102 mg/dL at intubation, 110 mg/dL during laparotomy and 98 mg/dL at PICU transfer, without hyperglycemia. Table 4 summarizes the serial laboratory trajectories.

Table 4. Serial laboratory values, acid-base status and organ function at four epochs.

Domain	Laboratory parameter	T0 Admission	T1 Pre-induction	T3 Intraoperative midpoint	T5 PICU handover	Comment
Inflammatory	White Blood Cell Count (/μL)	18,600	16,800 (-9.7%)	14,200 (-23.7%)	11,400 (-38.7%)	Leukocytosis, then fall
Inflammatory	Absolute Neutrophil Count (/μL)	15,250 (82%)	13,440 (80%)	10,650 (75%)	7,750 (68%)	Neutrophil count (percentage in parentheses) falls
Inflammatory	C-Reactive Protein (CRP, mg/L)	68.4	66.2 (-3.2%)	58.0 (-15.2%)	34.5 (-49.6%)	-
Acid-Base / Gas	Arterial pH	7.28	7.33	7.36	7.39	-
Acid-Base / Gas	PaCO ₂ (mmHg)	31.2	33.5	33.8	34.2	Low-normal PaCO ₂ at each time point
Acid-Base / Gas	PaO ₂ (mmHg)	82.5 (Room air)	145.0 (FiO ₂ 0.5)	138.0 (FiO ₂ 0.5)	142.0 (FiO ₂ 0.5)	PaO ₂ /FiO ₂ 276–290 on FiO ₂ 0.5
Acid-Base / Gas	Serum Bicarbonate (HCO ₃ ⁻ , mEq/L)	14.8	17.5	20.2	22.4	Rises toward normal
Acid-Base / Gas	Base Excess (BE, mEq/L)	-9.4	-6.1	-3.2	-1.1	Base deficit decreases
Perfusion Metric	Serum Lactate (mmol/L)	4.2	2.8 (-33.3%)	1.8 (-57.1%)	1.3 (-69.0%)	Falls below 2.0 mmol/L by T3
Renal Profile	Blood Urea Nitrogen (mg/dL)	28.0	24.5 (-12.5%)	19.8 (-29.3%)	15.2 (-45.7%)	Falls with resuscitation
Renal Profile	Serum Creatinine (mg/dL)	0.62	0.55 (-11.3%)	0.48 (-22.6%)	0.42 (-32.3%)	Within the reference interval at all times (low muscle mass limits interpretation)
Renal Profile	Urine Output Rate (mL/kg/h)	0.35 (Oliguric)	0.85 (Normal)	1.00 (Normal)	1.40 (Robust)	-
Electrolytes	Serum Sodium (Na ⁺ , mEq/L)	134.0	136.5	137.0	138.0	Normonatremia from T1
Electrolytes	Serum Potassium (K ⁺ , mEq/L)	4.1	4.0	3.9	4.0	Normal at all times
Electrolytes	Serum Chloride (Cl ⁻ , mEq/L)	98.0	100.5	101.0	102.0	Normal at all times
Electrolytes	Ionized Calcium (mmol/L)	1.12	1.18	1.20	1.22	Rises toward the reference interval
Hepatic Profile	Total Bilirubin (mg/dL)	0.8	0.7	0.7	0.6	Normal
Hepatic Profile	AST / ALT (U/L)	32 / 26	30 / 25	28 / 24	26 / 22	Normal
Coagulation	Prothrombin Time / INR	13.4 s / 1.12	13.0 s / 1.08	12.8 s / 1.06	12.4 s / 1.03	Normal

Notes: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BE, base excess; BUN, blood urea nitrogen; CRP, C-reactive protein; FiO₂, fraction of inspired oxygen; INR, international normalized ratio; PICU, pediatric intensive care unit.

After surgery and skin closure, the team chose to keep the child intubated rather than extubate in the operating room. The combined burden of spastic quadriplegic cerebral palsy, massive hydrocephalic macrocephaly, marasmic malnutrition and a laparotomy for abdominal sepsis put him at high risk of central hypoventilation, airway re-obstruction and failed re-intubation. He went straight to the Pediatric Intensive Care Unit with the cuffed 4.5 mm endotracheal tube in place. On PICU admission (Epoch T5) his Glasgow Coma Scale was E2V(ett)M1, with symmetrical 3 mm reactive pupils, soft non-bulging fontanelles and intact brainstem reflexes. Invasive mechanical ventilation was set up in assist-control pressure-controlled mode with inspiratory pressure reduced to 8 cmH₂O, PEEP 5 cmH₂O, respiratory rate 22 breaths/min and FiO₂ 0.50. Postoperative analgesia was intravenous metamizole (150

mg every 8 hours, 10 mg/kg). Continuous opioid infusions were deliberately avoided. The aim was to preserve neurological surveillance. The record given ends at the PICU admission.

4. Discussion

4.1. Principal lesson of the case

The main lesson of this case is that, in a child with a giant hydrocephalic head, a full stomach and septic physiology, a ketamine-based modified rapid sequence induction with torso ramping, normocapnic ventilation and deferral of shunt surgery was followed by stable hemodynamics and uncomplicated first-attempt intubation. The child had four major emergencies at once: severe communicating hydrocephalus with giant macrocephaly, acute low small-

bowel obstruction, abdominal sepsis and marasmic severe acute malnutrition. Clinical guidelines usually treat each in isolation, and their advice can conflict. Neuroanesthesia calls for vasodilatory, low-metabolism agents such as propofol to blunt intracranial pressure. Sepsis care demands immediate support of systemic vascular resistance and volume resuscitation to prevent circulatory collapse. A full stomach from bowel obstruction calls for rapid sequence induction to prevent pulmonary aspiration. Severe malnutrition requires strict metabolic and fluid stewardship to avoid cerebral edema and hypoglycemia. The team set aside the usual defaults and reworked each part of care. Propofol was replaced with a ketamine-fentanyl-rocuronium regimen, and torso ramp elevation was used to overcome the occipital deformity. Ventilation was strictly normocapnic and pressure-controlled, and the surgical steps were re-sequenced. The child stayed hemodynamically stable, no aspiration was recorded, and no clinical sign of raised intracranial pressure was noted at PICU admission.²³

4.2. Comparison with the published literature

Table 5 compares this case with published reports that could be verified. Kumari et al. described a massive hydrocephalus case with severe airway difficulty, prolonged intubation and transient hypoxemia after standard unramped inhalation induction. In our patient the torso ramp was followed by a Cormack-Lehane Grade I view and intubation at the first attempt. Hussain et al. surveyed classical rapid sequence induction and reported the widespread hazard of pediatric desaturation. With our modified technique and gentle low-pressure ventilation, SpO₂ stayed at 97–98% after induction. Our observation is compatible with the neurocritical care evidence of Mazandi et al. on the intracranial safety of ketamine. With strict normocapnic ventilation (EtCO₂ 32–34 mmHg), ketamine was followed by stable systemic hemodynamics and no clinical sign of intracranial hypertension.^{17–22} Finally, our surgical priorities are compatible with acute care surgery guidance. In multi-cavitary emergencies, a life-threatening septic abdominal focus takes precedence over a chronic neurosurgical lesion.²³

Table 5. Comparison of this case with published reports that could be verified.

Source	Design and population	Procedure or setting	Main findings	Relevance to this case
Mahajan et al., 2017 ⁵	Retrospective review; 29 children with giant encephalocele (hydrocephalus in 65.5%)	Excision and repair under general anesthesia	Difficult tracheal intubation in 51.7%; direct laryngoscopy usually in the lateral position (82.8%); hemodynamic complications 31.0%, respiratory 17.2%	Difficult airway in children with a large cranial mass; lateral positioning was used instead of torso ramping
Kumari et al., 2024 ³	Case report; one child with massive hydrocephalus and aqueduct stenosis	Ventriculoperitoneal shunt placement	Difficult airway due to macrocephaly and associated anomalies (abstract level)	Same anatomical problem; details of the airway technique are not available at abstract level
Vagyannavar et al., 2017 ²	Case report; child with gross hydrocephalus and Arnold-Chiari type 2 malformation	Emergency ventriculoperitoneal shunt placement	Difficult airway due to the increased head circumference and anomalies (abstract level)	Another report of a giant hydrocephalic head and a difficult airway
Hussain et al., 2023 ⁷	Survey; 192 respondents (response rate 75%)	Practice of pediatric rapid sequence induction in India	Wide variation in practice; succinylcholine the most used relaxant; cricoid pressure used more in older age groups	Context for a modified rapid sequence induction; no data on hydrocephalus or sepsis
Kemper et al., 2020 ⁸	Retrospective audit; 22 classical and 88 controlled rapid sequence inductions in children with bleeding tonsils	Classical (apneic) versus controlled (gentle bag-mask ventilation) induction	Controlled induction: less severe hypoxemia (1 vs 3; $P = .025$), better direct laryngoscopic views ($P = .048$), less hypertension; no excess pulmonary morbidity	Supports gentle mask ventilation during rapid sequence induction in children; not a neurosurgical population
Lee et al., 2018 ²⁴	Randomized study; children aged 1 month to 5 years	Face mask ventilation with and without neuromuscular blockade	Gastric insufflation common; the inspiratory pressure that caused insufflation was about 18 cmH ₂ O	Compatible with limiting mask pressure to <12 cmH ₂ O in this case
Mazandi et al., 2023 ¹⁷	Retrospective observational study; 143 children (70 received ketamine)	Tracheal intubation in children with neurologic conditions at risk of intracranial hypertension	No significant difference in neurologic events ($p = 0.42$), hemodynamic or respiratory events; no 24-h mortality	Compatible with ketamine induction when ventilation is controlled; sepsis and hydrocephalus not studied
Laws et al., 2023 ²⁰	Retrospective observational study; 33 children with severe traumatic brain injury (127 ketamine doses)	Ketamine boluses with intracranial pressure monitoring	No rise in intracranial pressure after ketamine; a fall during pressure crises	Supports ketamine intracranial safety in monitored, ventilated children; our patient had no ICP monitoring
Kang et al., 2022 ²⁵	Randomized study; 144 adults	Videolaryngoscopy in the ramped position at two table heights	Lower (umbilical) table level: total intubation time 28 ± 5 s versus 40 ± 7 s	Adult data on ramped positioning; no pediatric or macrocephaly data
Lotfi et al., 2023 ²⁶	Randomized trial; 112 adults with Mallampati class above 2	Standard position versus modified ramped (RAMP) position	Intubation time 51.25 s versus 88.39 s ($P = 0.019$); better glottic view	Adult data; no pediatric ramped-position study was found
Semler et al., 2017 ²⁷	Randomized trial; critically ill adults	Ramped versus sniffing position for intubation	The ramped position may worsen the glottic view and increase the number of laryngoscopy attempts (grade III or IV view 25.4% versus 11.5%)	Contrary adult evidence; not a pediatric or macrocephaly study
Okada et al., 2021 ²⁸	Systematic review and meta-analysis	Ramped versus sniffing position for tracheal intubation	No favorable aspects of the ramped position compared with the sniffing position	Contrary adult evidence

Source	Design and population	Procedure or setting	Main findings	Relevance to this case
Present case	Single patient (n = 1)	Ketamine-based modified RSI, torso ramping, normocapnic ventilation, shunt deferred	First-attempt intubation (grade I view); MAP 65.0–70.7 mmHg without vasopressors; EtCO ₂ 32–34 mmHg; follow-up ends at PICU handover	Adds a detailed anesthetic record in a child with a giant head, a full stomach and septic physiology

Notes: ICP, intracranial pressure; RSI, rapid sequence induction.

4.3. Pathophysiological or mechanistic interpretation

The sections below separate what was observed from what is inferred. Intracranial pressure was not measured, so statements about intracranial compliance and cerebral perfusion are inferences from clinical and capnographic surrogates.

The principle usually invoked for intracranial dynamics is the Monro-Kellie doctrine, which treats the cranium as a fixed-volume vault holding brain parenchyma, blood and cerebrospinal fluid.²⁹ In a child with open sutures and a patent fontanelle the skull is expandable, so the premise holds only once the vault has reached its limit. In this 5-year-old, long-standing untreated communicating hydrocephalus had enlarged the skull through patent sutures and open fontanelles, up to a head circumference of 81 cm.⁴ Bone expansion is non-linear and finite, though. Once the calvarial volume reached its limit, intracranial compliance is likely to have been exhausted.⁴ On the steep part of the intracranial volume-pressure curve, a small rise in cerebral blood volume produces a steep rise in intracranial pressure.³⁰ Cerebral perfusion pressure is the gradient between mean arterial pressure and intracranial pressure (CPP = MAP - ICP). Severe hydrocephalus raises baseline intracranial pressure, so the perfusion margin is precarious.³¹ If systemic sepsis or anesthetic vasodilators cause mean arterial pressure to fall, cerebral perfusion pressure drops below the autoregulatory threshold (typically 40–50 mmHg in young children). The consequences can be diffuse cerebral ischemia, white matter infarction and permanent neurological damage. Defending mean arterial pressure above 65 mmHg was therefore our main neuroprotective aim.^{14,32}

The choice of induction agent was the main pharmacologic question in this case. Propofol is widely used in neuroanesthesia, because it reliably suppresses cerebral metabolic rate (CMRO₂) and decreases intracranial pressure.⁶ It is also a potent peripheral vasodilator that blunts sympathetic vascular tone and depresses myocardial contractility. In healthy pediatric patients it regularly lowers mean arterial pressure by 20% to 30%. In this child, sepsis had already caused severe vasoplegia through nitric oxide-mediated relaxation of vascular smooth muscle. Third-space hypovolemia from the bowel obstruction added to it. Propofol under these conditions could have worsened hypotension and reduced cerebral perfusion. Ketamine, by contrast, has historically been considered contraindicated in neurosurgery. Early animal and human studies suggested that it raises intracranial pressure through sympathetic stimulation.^{21,33} Contemporary neurocritical care evidence points the other way. The pediatric cohort study of 143 patients by Mazandi et al. and the consensus reviews by Godoy et al. and Robba et al. agree. Ketamine does not increase intracranial pressure or adverse neurological events when ventilation is controlled and normocapnia is maintained.^{17–22} Ketamine acts centrally as a sympathomimetic, and it preserves systemic vascular resistance and diastolic filling. It defends mean arterial pressure.¹⁹ In our patient, ketamine 0.5 mg/kg given with fentanyl (1 mcg/kg) was

followed by a post-induction mean arterial pressure of 65.3 mmHg, with no hypotension recorded.^{34,35}

The safety of ketamine-based induction in raised intracranial pressure depends on careful ventilatory control.^{18,19} Carbon dioxide is the most potent physiological regulator of cerebral arteriolar caliber. Hypercapnia causes rapid cerebral vasodilation, increases cerebral blood volume and sharply raises intracranial pressure. Hypocapnia causes severe vasoconstriction and threatens cerebral ischemia.¹⁵ In spontaneous breathing models, ketamine raises intracranial pressure mainly because its respiratory depression lets carbon dioxide build up.^{18,19} We paired modified rapid sequence induction with immediate controlled ventilation to an end-tidal carbon dioxide of 30 to 35 mmHg. The aim was to counter the cerebral vasodilation that carbon dioxide retention would cause with ketamine. Pressure-controlled ventilation was also set to limit inspiratory pressure to 15 cmH₂O and PEEP to 5 cmH₂O. High intrathoracic pressure impedes cerebral venous return through the superior vena cava and internal jugular veins, and it transmits raised venous pressure directly into the intracranial venous sinuses and worsens intracranial hypertension. The low-pressure strategy used here was intended to allow cerebral venous drainage while keeping alveolar gas exchange adequate.^{36,37}

Severe hydrocephalus creates anatomical airway problems that conventional positioning may not solve.^{2,3,5} A young child has a relatively larger occiput than an adult, so shoulder elevation is often needed to reach the sniffing position. In this child, an 81 cm head circumference produced an extreme occipital prominence that distorted upper airway kinematics. Supine on a flat surface, the large occiput acts as a fulcrum. It forces the cervical spine into extreme flexion and presses the mandible against the sternum, and this flexion closes the pharyngeal space and collapses the soft tissues of the hypopharynx. It also places the glottic aperture far anterior to the laryngoscopist's visual axis, which would be expected to give a Cormack-Lehane Grade III or IV view. We built a torso elevation ramp from folded towels, and it raised the shoulders and chest by approximately 8 cm and brought the body plane into horizontal alignment with the occiput. This corrected the cervical flexion, opened the submandibular compliance space and lined up the oral, pharyngeal and laryngeal planes into a direct line of sight. The evidence on ramped positioning comes from adults and is mixed. Kang et al. found a shorter intubation time with a lower table height in the ramped position.²⁵ A randomized trial of a modified ramp found a shorter intubation time and a better glottic view.²⁶ Head-elevated positioning was associated with fewer airway complications in a retrospective adult study.³⁸ In contrast, a randomized trial in critically ill adults found that the ramped position may worsen the glottic view,²⁷ and a meta-analysis found no advantage over the sniffing position.²⁸ No pediatric or macrocephaly study was found, so the value of torso ramping here rests on the first-attempt grade I view in this one child.

Obstructive ileus with fecal stasis, vomiting and high intraluminal bowel pressure created a high risk of pulmonary aspiration of acidic gastric contents. Aspiration

in pediatric surgical patients is linked to chemical pneumonitis and fatal acute respiratory distress syndrome.³⁹ Classical rapid sequence induction gives a rapid-onset hypnotic and a neuromuscular blocker, and the child then stays completely apneic until the trachea is intubated with a cuffed tube.⁷ Young children, however, have a high metabolic oxygen consumption rate (6–8 mL/kg/min) and a small functional residual capacity. Arterial desaturation can occur within 30 to 45 seconds of apnea.⁴⁰ Here, sepsis, fever (38.5 °C) and marasmic cachexia sped up oxygen depletion, so classical apneic rapid sequence induction was judged too hazardous.⁴⁰ To combine aspiration prophylaxis with prevention of desaturation, we used a modified rapid sequence induction paradigm.^{7,8,40} First, a 10 French nasogastric tube emptied 80 mL of pooled gastric fluid before induction, which lowered intragastric volume and pressure. Second, after ketamine and rocuronium, gentle low-pressure mask ventilation (peak pressure strictly <12 cmH₂O) was given during the onset of muscle relaxation, and arterial oxygen saturation was 98% and stayed well below the 15 to 20 cmH₂O threshold needed to overcome the lower esophageal sphincter.²⁴ Third, a cuffed 4.5 mm endotracheal tube gave an immediate, impermeable seal that protected the tracheobronchial tree from silent regurgitation.^{41–43}

Rocuronium was chosen over succinylcholine for rapid sequence induction for three reasons.⁴⁴ Succinylcholine acts quickly, but in this setting succinylcholine carries hazards. First, it causes fasciculations that transiently raise intracranial pressure, which could be harmful if intracranial compliance is exhausted. Second, the patient had spastic quadriplegic cerebral palsy with chronic physical immobility. This state is reported to upregulate extrajunctional acetylcholine receptors and may predispose to hyperkalemia after succinylcholine. Third, its duration of action is short, and redosing would have been needed in an anticipated difficult airway. With rocuronium at ≈0.53 mg/kg, intubating conditions were adequate at 60 seconds; serum potassium was not measured after induction. Sugammadex, the selective cyclodextrin encapsulating agent, was also available. It could reverse rocuronium completely within 2 to 3 minutes. That mattered if a 'cannot intubate, cannot oxygenate' emergency developed or an urgent postoperative neurological examination were required.^{45,46}

A central point of this case is the deliberate reordering of surgical priorities. In conventional pediatric neurosurgery, untreated communicating hydrocephalus with macrocephaly calls for definitive cerebrospinal fluid diversion, usually by ventriculoperitoneal shunt.^{1,47} This patient's acute presentation with obstructive ileus and abdominal sepsis changed that order. Placing a ventriculoperitoneal shunt into a distended, obstructed and possibly infected peritoneal cavity is generally avoided. The distal catheter would face high intra-abdominal pressure that impedes cerebrospinal fluid drainage. Insertion risks bowel perforation during trocar placement. Bacterial colonization of the prosthesis could follow, with ascending ventriculitis, shunt infection and intracranial sepsis. The CT findings of corpus callosum agenesis and open cranial sutures showed something else as well. This child's hydrocephalus was a chronic, slowly compensated congenital process. It was not an acute intracranial crisis. There were no clinical signs of impending brainstem herniation, such as bradycardia, irregular respiration or pupillary asymmetry. The acute

surgical abdomen was the immediate, irreversible threat to life.⁴⁸ Prioritizing exploratory laparotomy relieved the mechanical bowel obstruction. The intention was that elective ventriculoperitoneal shunting could be done later, in a decompressed abdomen. The peritoneal fluid was non-purulent and the bowel viable, so the degree of peritoneal contamination at surgery is uncertain, and the shunt had not been placed by the end of the record given.

Marasmic severe acute malnutrition added metabolic and physiological fragility to this operation.^{49,50} Malnourished children may have myocardial atrophy, depleted hepatic glycogen, impaired gluconeogenesis, altered clearance of local anesthetics and muscle relaxants, and defective thermoregulation.^{50,51} The borderline admission blood glucose of 68 mg/dL showed the danger of intraoperative hypoglycemia, which causes irreversible neuronal injury in a brain already compromised by hydrocephalus.⁵² To prevent it, intraoperative maintenance fluid contained 1% dextrose. Glucose was 98–110 mg/dL during surgery, without marked hyperglycemia.⁵³ Glucose had already risen to 88 mg/dL before surgery and 102 mg/dL at intubation, so stress, ketamine and dexamethasone may also have contributed. Fluid balance was handled with equal caution. The volume given was enough to restore effective circulating arterial volume. Lactic acidosis cleared (lactate decreased from 4.2 to 1.3 mmol/L, a 69.0% reduction). The net balance still stayed at a modest +61 mL over 2.5 hours. In malnourished children, serum albumin is low and sepsis raises capillary permeability. Large fluid boluses then leak quickly into the tissues and can cause pulmonary edema and cerebral swelling.^{54,55}

The course up to the PICU handover in this high-risk patient suggests the value of proactive multidisciplinary communication across the perioperative course. From admission, pediatric intensive care, pediatric surgery and anesthesiology worked together. Preoperative fluid resuscitation was completed before induction, and the surgical plan was agreed in advance. The operating room was fully prepared for difficult airway management and hemodynamic stabilization.⁵⁶ The decision to defer extubation and transfer the child intubated to the PICU was a deliberate neuroprotective measure.⁵⁷ Extubation is a time of physiological instability, with coughing, straining, acute hypertensive spikes and transient hypoventilation. Each of these can cause steep rises in intracranial pressure and threaten secondary brain injury.⁵⁸ This child had cerebral palsy, massive macrocephaly, marasmic cachexia and a fresh laparotomy incision. Early extubation risked respiratory failure, hypoxemia and emergency re-intubation of a difficult airway. Transfer to the PICU intubated, with assist-control ventilation and non-opioid metamizole analgesia, allowed neurological surveillance and sustained normocapnia.^{59,60}

Figure 5 is an interpretative schematic of how standard anesthetic practice and the integrated plan used here differ; it shows the authors' reasoning, not measured outcomes. The cascade has four tiers. Tier 1 sets out the baseline multi-system pathology. Tier 2 contrasts the harmful conventional pathway with the integrated protective pathway. The conventional pathway means a propofol vasoplegic crash, flat supine cervical flexion, desaturation with classical apneic RSI and premature shunt inoculation, and the protective pathway means ketamine support of SVR, torso ramp alignment, low-pressure modified RSI and surgical re-sequencing. Tier 3 sets the resulting microvascular outcomes side by side. Ischemic secondary brain injury, pulmonary aspiration

and ventriculitis are contrasted with defended cerebral perfusion pressure, normocapnic vasomotor stability and aspiration prophylaxis. Tier 4 sets the risks of conventional

care, as interpreted by the authors, beside the clinical course observed in this patient up to the PICU handover.

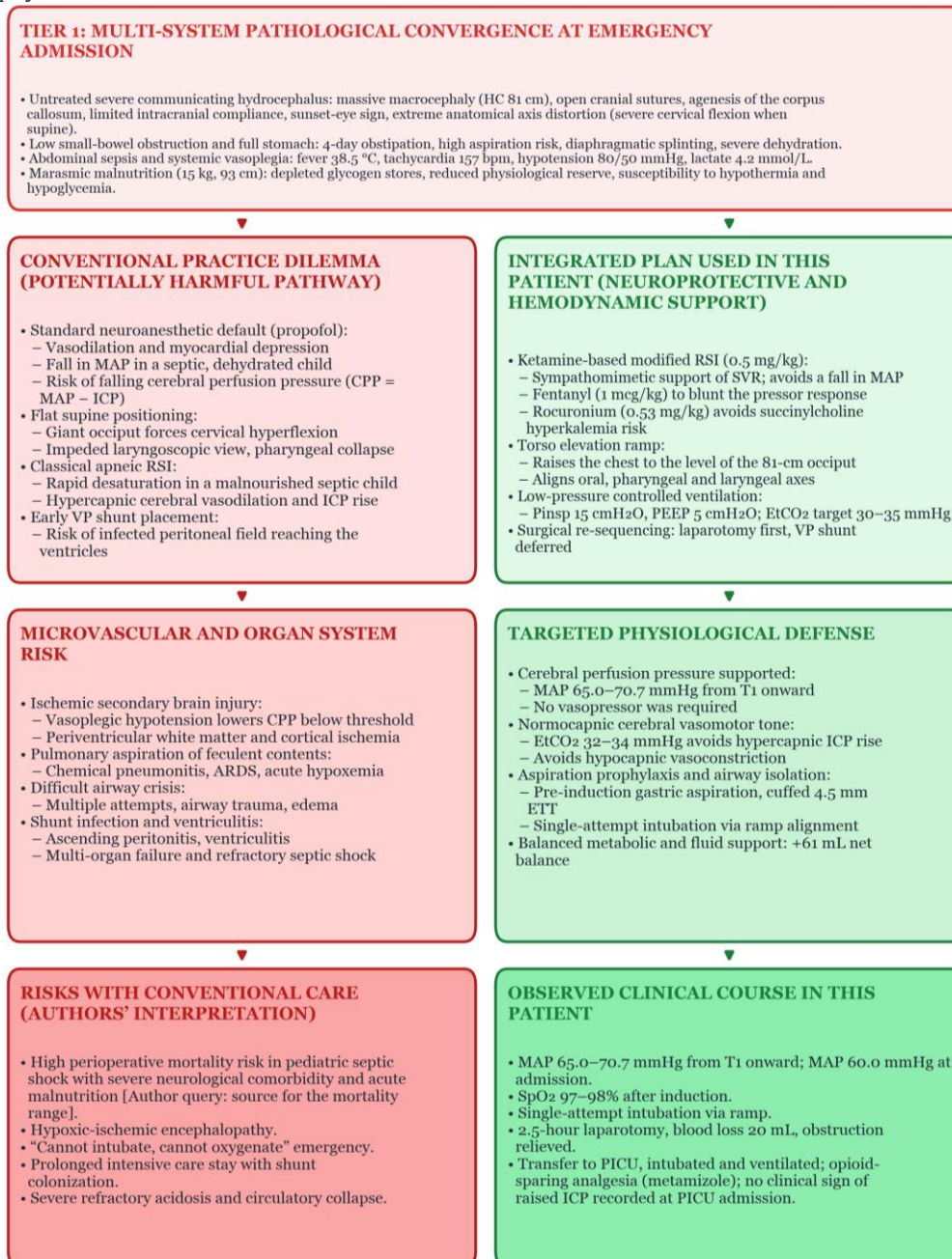


Figure 5. Interpretative schematic of the pathophysiological cascade: the converging emergencies at admission (top), the risks of conventional care (left) and the integrated plan used in this patient with the course observed up to the PICU handover (right). The conventional-care column shows the authors' reasoning and is not based on measured outcomes. ARDS, acute respiratory distress syndrome; CPP, cerebral perfusion pressure; ETT, endotracheal tube; ICP, intracranial pressure; MAP, mean arterial pressure; PICU, pediatric intensive care unit; RSI, rapid sequence induction; SVR, systemic vascular resistance; VP, ventriculoperitoneal.

4.4. Implications for practice

These observations suggest points that may be useful in a similar child, although they are not a protocol. Torso ramping is simple and needs little equipment; it was followed by a grade I view in a child whose flat supine position was expected to give flexion. Gastric decompression before induction, gentle low-pressure mask ventilation and a cuffed tube formed the aspiration strategy while avoiding desaturation. When septic hypotension is present, an induction agent with sympathomimetic action such as ketamine, with controlled normocapnic ventilation, is a reasonable option to consider. Deferring the shunt and keeping the child

intubated and ventilated after a laparotomy were deliberate choices. How far this advice transfers to other children, to other hospitals and to children without cerebral palsy or malnutrition is not known.

4.5. Strengths

The strengths of this report are the detailed record of hemodynamic, ventilatory and laboratory values across six standardized epochs and the explicit reasoning for each anesthetic step. Descriptive statistics only were used. The combination of a giant hydrocephalic head, obstructive ileus, sepsis and marasmus in one child is unusual.

4.6. Limitations

A single case cannot establish causation, prevalence or effect. Intracranial pressure was not measured because surgical urgency precluded it, so intracranial compliance was inferred from clinical and capnographic surrogates. Several measures (volume resuscitation, antibiotics, positioning, induction, ventilation and surgery) were delivered together and cannot be separated. The source of sepsis is unproven (no culture results are given), and the record ends at the PICU admission, without follow-up of neurological outcome, extubation, discharge or shunt surgery. Mean arterial pressure may be a calculated value, and some physiological measures were not recorded (maintenance agent, neuromuscular monitoring). This is a single-center experience in one patient, and the findings may not generalize.

4.7. Recommendations for future work

A prospective case series or registry of children with macrocephaly or giant hydrocephalus who need emergency abdominal surgery would be needed to test whether this approach is reproducible. It should prespecify endpoints (first-attempt intubation, hypotension, desaturation, a surrogate of intracranial pressure and neurological outcome at follow-up) and describe the induction agent, ventilation and positioning used.

5. Conclusion

This case report describes the anesthetic management of a 5-year-old boy with giant communicating hydrocephalus, obstructive ileus, sepsis physiology and marasmic malnutrition who needed emergency laparotomy. A ketamine-based modified rapid sequence induction was used, with gentle low-pressure mask ventilation, torso ramping and a cuffed tube. Ventilation was normocapnic, and the shunt was deferred. Intubation succeeded at the first attempt. Mean arterial pressure stayed at 65.0–70.7 mmHg without vasopressors, and he reached intensive care in stable condition. These are observations from one child. Intracranial pressure was not measured, and follow-up beyond the handover was not recorded, so they cannot show that the measures caused the outcome. Torso ramping is simple and may be considered in a similar child.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under reference number 2026/5194. The study followed the institutional standards of Dr. Moewardi Regional General Hospital and the Declaration of Helsinki (amended Fortaleza, Brazil, 2013). Written informed consent for the procedure and for the use of anonymized clinical data and images was obtained from the patient's parents before surgery. Eyes are masked in all photographs.

Consent for publication. Written informed consent for publication of this case and any accompanying images was obtained from the patient's legal guardians (parents).

Availability of data and materials. The clinical data, operative logs, physiological monitoring trends and radiological examinations supporting this report are available from the corresponding author on reasonable academic request, subject to patient privacy protections and institutional review board compliance.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. A.T.A. conceptualized the report, performed the anesthetic management, collected perioperative data and drafted the manuscript (Conceptualization; Investigation; Data curation; Writing – original draft). A.I. contributed to intraoperative data acquisition, radiological analysis and literature synthesis and revised the manuscript (Investigation; Formal analysis; Resources; Writing – review & editing). J.R.M. supervised the clinical care, designed the neuroanesthetic and hemodynamic plan, revised the manuscript critically, provided institutional oversight and approved the final version (Methodology; Supervision; Project administration; Writing – review & editing). All authors read and approved the final manuscript.

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Conflict of interest (competing interests) statement. The authors declare that they have no competing interests.

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