Case Report

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Anesthetic Management for Laparotomy of Necrotizing Enterocolitis in a Preterm Infant with Tetralogy of Fallot and Patent Ductus Arteriosus: A Case Report

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Abstract:

Anesthetic management of preterm infants with Tetralogy of Fallot (TOF) and patent ductus arteriosus (PDA), complicated by necrotizing enterocolitis (NEC), poses significant challenges. This case report describes a 1540-g male infant, born at 34 weeks and 4 days of gestation, diagnosed with TOF and PDA. Lipo-prostaglandin E1 (lipo-PGE1) was administered immediately to maintain PDA patency. At 18 days old, the infant developed lethargy, sepsis, and NEC stage IIIB, requiring an emergency exploratory laparotomy. Anesthetic management involved maintaining heart rate, contractility and preload, avoiding increases in the pulmonary vascular resistance (PVR) to the systemic vascular resistance (SVR) ratio, preventing PDA closure, and promptly addressing hypercyanotic spells. Surgical findings included extensive intestinal necrosis, necessitating multiple resections and the creation of an ileostomy and a jejunostomy. Postoperative care included meticulous cardiovascular and respiratory support, careful use of vasopressors, and precise lipo-PGE1 adjustments. The case highlights the need to maintain PDA patency in TOF, perform early surgical intervention for NEC, and provide precise postoperative care. The successful outcome was due to timely diagnosis, effective anesthetic care and surgery, and careful management, underscoring the importance of a coordinated, multidisciplinary approach.

Keywords: Anesthetic management, infant, necrotizing enterocolitis, patent ductus arteriosus, preterm, Tetralogy of Fallot

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Introduction

Tetralogy of Fallot (TOF) is the most common form of cyanotic heart disease and has an incidence of 10% of all congenital heart defects[1]. It is characterized by a ventricular septal defect (VSD), an overriding aorta, right ventricular hypertrophy, and pulmonic stenosis, which can be infundibular or subvalvular, valvular, supravalvular, or a combination thereof[1]. The patent ductus arteriosus (PDA) enables a significant left-to-right shunt, resulting in increased flow through the pulmonary circulation and decreased perfusion of the systemic circulation. In the TOF with pulmonary valve stenosis, the PDA is an important key to preserving pulmonary circulation[1]. The survival and management of neonates with TOF with PDA can be particularly challenging, especially when additional complications such as necrotizing enterocolitis (NEC) occur. This case report describes the clinical progression and management of a preterm infant, born at 34 weeks and 4 days of gestation, who was prenatally diagnosed with TOF and PDA, and subsequently developed NEC stage IIIB[2]. The objective is to highlight the challenges encountered and the strategies implemented in the care of this critically ill neonate.

Case report

The patient's parents provided informed consent for this publication in accordance with Tokyo Metropolitan Children's Medical Center protocols. A 1540-g, 34-week-and-4-day gestation preterm boy was delivered by spontaneous vaginal delivery. The infant was born to a 31-year-old primigravida mother with a prenatal diagnosis of TOF. His Apgar score was 8 at 1 minute and 8 at 5 minutes, with a heart rate (HR) of 156 beats per minute, blood pressure (BP) of 48/22 mmHg with a mean arterial pressure (MAP) of 31 mmHg, respiratory rate (RR) of 60 breaths per minute, and oxygen saturation (SpO_2) of 80%. He was admitted to the Neonatal Intensive Care Unit (NICU). Transthoracic echocardiogram (TTE) detected TOF and PDA. It consisted of VSD Kirklin I muscular outlet type, overriding aorta, right aortic arch, right ventricular outflow tract obstruction (RVOTO), pulmonary valve stenosis, and right ventricular hypertrophy with a PDA diameter of 4.6 mm. He was started on Lipo-prostaglandin E1 (lipo-PGE1) at a dose of 3 ng/kg/min immediately after birth due to the fetal diagnosis of TOF to prevent PDA closure. He required supplemental oxygen: initially via continuous positive airway pressure (CPAP), later by nasal cannula, and finally through endotracheal intubation (uncuffed tube size 3.0 with a depth of 7.5 cm) on a mechanical ventilator set to synchronized intermittent mandatory ventilation (SIMV) with pressure control ventilation (PCV) (fraction of inspired oxygen (FiO_2) 21%, peak inspiratory pressure (PIP) 14-16 cmH_2O, positive end-expiratory pressure (PEEP) 6-8 cmH_2O, and RR 20 breaths per minute), which were necessary to control pulmonary vascular resistance (PVR).

At 18 days of age, he presented with lethargy and sepsis. His SpO_2 ranged from 90% to 94%, HR ranged from 150 to 180 beats per minute, MAP ranged from 38 to 48 mmHg, and RR ranged from 40 to 60 breaths per minute with mechanical ventilation using an FiO_2 of 21%. The examination showed systolic murmurs (Levine grading scale II), marked abdominal distention, poor abdominal sounds, and he presented with bloody stool. Initial complete blood counts showed a white cell count of 11.36×10^3/μL, hemoglobin (Hb) of 13.7 g/dL, hematocrit (Hct) of 37.3%, platelets of 242×10^3/μL, and neutrophils at 70.4%. From a capillary sample, pH was 7.390, partial pressure of oxygen (PaO_2) was 62.8 mmHg, partial pressure of carbon dioxide ($PaCO_2$) was 40.4 mmHg, base excess (BE) -0.5, sodium 123 mmol/L, potassium 3.7 mmol/L, bicarbonate 23.9 mmol/L, chloride 86 mmol/L, calcium 0.99 mmol/L, Hct 41.9%, Hb 13.7 g/dL, glucose 134 mg/dL, and lactate 1.9 mmol/L. The C-reactive protein was 10.39 mg/L. An abdominal radiograph showed prominent bowel loops,

loss of liver haziness, and pneumoperitoneum (Figure 1). He was diagnosed with NEC stage IIIB according to modified Bell's Staging[2]. Medical management steps are often taken to stabilize the patient and prepare them for the operating room, including discontinuation of enteral feeding, nasogastric decompression, administration of broad-spectrum antibiotics, and mechanical ventilation. He underwent a septic workup and had invasive pressure monitoring, along with a peripherally inserted central catheter (PICC) line. It was challenging to reduce the dose of lipo-PGE1 in order to increase systolic circulation. However, the PDA was important for the survival of this patient. We followed up with a TTE to monitor the PDA and adjust the dose of lipo-PGE1 in order to prevent PDA closure and maintain SpO_2 at 90-95%. By including these pre-operative management steps, the patient's condition could be optimized before proceeding to the operating room for emergency exploratory laparotomy under general anesthesia.

For preparation, the operating room temperature was set to 28 °C, and an underbody forced-air warmer, radiant heater, and blankets were used. A neonatal circuit with a heater and humidifier was also employed. Intravenous fluids, blood, and blood products were warmed to prevent

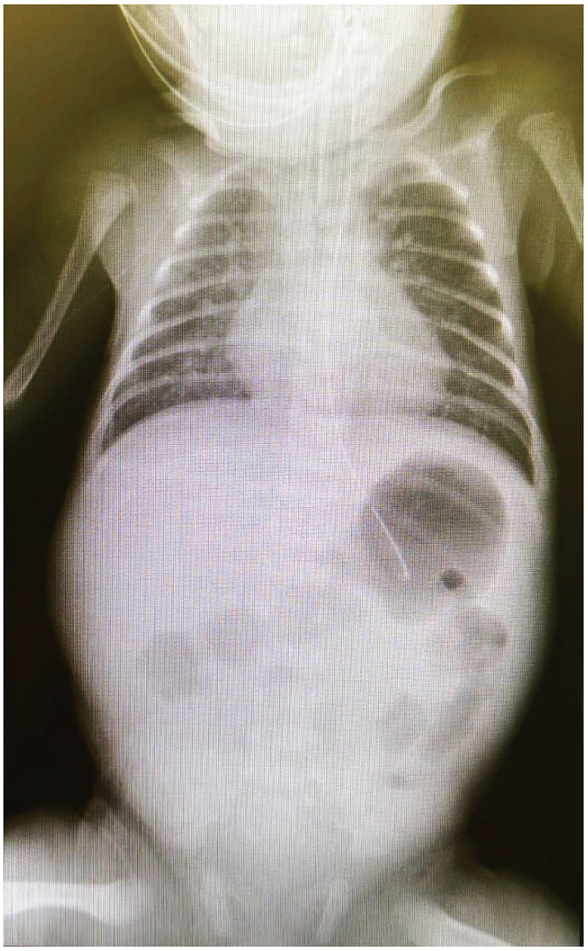

Figure 1 The abdominal radiograph demonstrating prominent bowel loops, loss of liver haziness, and pneumoperitoneum

hypothermia. Additionally, the anesthetic machine, ventilator, breathing circuits, infusion pumps, endotracheal tube, and laryngoscopes were prepared and checked.

For the first operation, the patient was classified as American Society of Anesthesiologists Physical Status (ASA-PS) III (emergency). Before proceeding, the patient's vital signs were as follows: BP was 71/33 mmHg, MAP was 49 mmHg, HR was 170 beats per minute, SpO_2 was 95%, and RR ranged from 30 to 60 breaths per minute on SIMV with PCV. He continued on lipo-PGE1 at a dose of 1–3 ng/kg/min, adjusted based on the patient's hemodynamic status and clinical condition. Monitoring included electrocardiogram (ECG), capnography, BP, HR, SpO_2, intra-arterial pressure, and intraesophageal temperature. During anesthetic management, an FiO_2 of 21% was used to maintain the preterm infant's SpO_2 between 90% and 95%, effectively preventing hemodynamic collapse.

Anesthesia induction included sevoflurane 0.4% in oxygen and air (FiO_2 21%), fentanyl 2 mcg/kg IV, remifentanil 0.08 mcg/kg/min infusion, and rocuronium 1.2 mg/kg IV. Maintenance included sevoflurane 0.4%, remifentanil 0.08 mcg/kg/min infusion, titrated fentanyl 1 mcg/kg/dose IV, and rocuronium 1 mg/kg/hr infusion. The operation revealed multiple segments of intestinal necrosis along the small intestine (Figure 2). An exploratory laparotomy with resection of necrotic small intestine and application of vacuum-assisted closure (VAC®) was performed. The total anesthetic time was 155 minutes, the operation time was 89 minutes, with minimal blood loss and a loss of 35 mL of ascitic fluid. Arterial blood gas (ABG) results showed a change from: pH 7.317, PaO_2 50.7 mmHg, $PaCO_2$ 45.2 mmHg, BE -3.1, sodium 126 mmol/L, potassium 3.3 mmol/L, bicarbonate 22.5 mmol/L, chloride 97 mmol/L, calcium 1.12 mmol/L, Hct 32.7%, Hb 10.6 g/

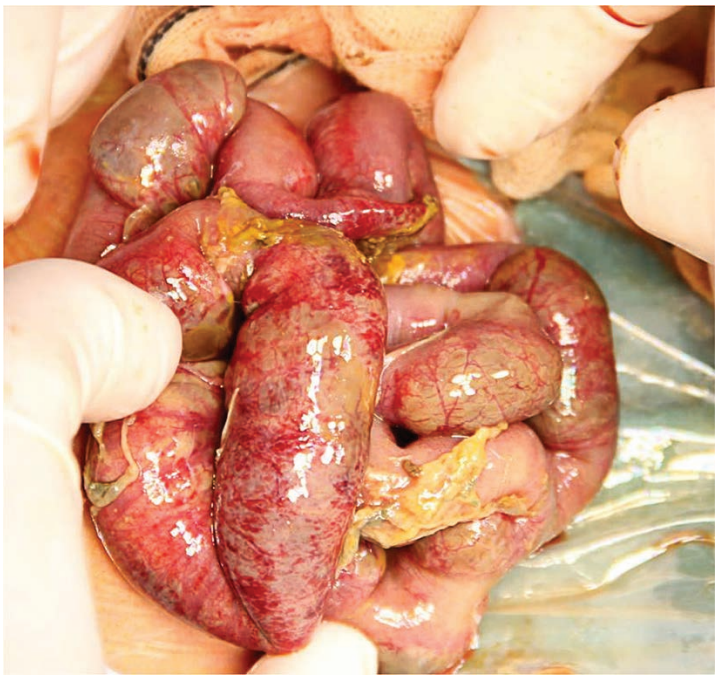

Figure 2 Necrotizing enterocolitis at the time of laparotomy. Note the multiple segments of intestinal necrosis along the small intestine.

dL, glucose 99 mg/dL, lactate 1.1 mmol/L to: pH 7.233, PaO_2 48.8 mmHg, $PaCO_2$ 45.2 mmHg, BE −8.0, sodium 130 mmol/L, potassium 3.2 mmol/L, bicarbonate 18.4 mmol/L, chloride 102 mmol/L, calcium 1.04 mmol/L, Hct 22.4%, Hb 7.2 g/dL, glucose 104 mg/dL, lactate 1.2 mmol/L. Anemia and metabolic acidosis were corrected with intravenous fluids, including Physio140 (1% glucose with acetate Ringer's solution) (36 mL), 5% albumin (162 mL), and leukoreduced irradiated red blood cells (25 mL). Metabolic acidosis was treated with 7% sodium bicarbonate (2 mEq/kg) and 2% calcium chloride (10 mg/kg). In the operating room, dopamine or dobutamine infusion had not yet been started. The patient had no urine output. At the end of the operation, vital signs were as follows: BP 66/30 mmHg, HR 172 beats per minute, SpO_2 90%, end-tidal carbon dioxide ($EtCO_2$) 36 mmHg, and body temperature (BT) 36.2 °C. In the NICU, a post-operative bladder ultrasound showed urine accumulation, with a urine output of 20 mL in 8 hours (1.6 mL/kg/hr). Dopamine and dobutamine (3 mcg/kg/min) were started an hour before the second operation for hemodynamic support.

For the second operation on day 20 of age, anesthesia induction included oxygen with air (FiO_2 21%), midazolam 0.1 mg/kg IV, fentanyl 2 mcg/kg IV, remifentanil 0.2 mcg/kg/min infusion, and rocuronium 1.2 mg/kg IV. Maintenance involved remifentanil 0.2 mcg/kg/min infusion, titrated fentanyl 1 mcg/kg/dose IV, and rocuronium 1 mg/kg/dose IV, along with continuous dopamine and dobutamine infusions at 3 mcg/kg/min each, and lipo-PGE1 at 1–3 ng/kg/min, continued from the NICU. An exploratory laparotomy with resection of necrotic small intestine and drainage using VAC® was performed. The total anesthetic time was 152 minutes, with an operation time of 74 minutes and minimal blood loss. ABG results indicated anemia (Hb 10.7 g/dL, Hct 32.9%). This was corrected with a newborn cocktail (consisting of 20% albumin (10 mL) with Physio 140 (40 mL) up to 50 mL) (110 mL), and leukoreduced irradiated red blood cells (70 mL). Urine output was 28 mL (7.2 mL/kg/hr). At the end of the operation, vital signs were as follows: BP 65/32 mmHg, HR 168 beats per minute, SpO_2 90%, $EtCO_2$ 32 mmHg, and BT 37 °C.

For the third operation on day 22 of age, anesthesia induction included oxygen with air (FiO_2 21%), midazolam 0.2 mg/kg IV, fentanyl 2 mcg/kg IV, and rocuronium 1.2 mg/kg IV. Maintenance involved titrated fentanyl 2 mcg/kg/dose IV and rocuronium 1 mg/kg/dose IV, and continued lipo-PGE1 at 1–3 ng/kg/min, without dopamine or dobutamine infusions. An exploratory laparotomy and drainage using VAC® were performed. The total anesthetic time was 154 minutes, and the operation time was 63 minutes, with a blood loss of 2 mL. ABG results were within normal range. Fluid replacement included a newborn cocktail (consisting of 20% albumin (10 mL) with Physio 140 (40 mL) up to 50 mL) for a total of 65 mL. Urine output was 32 mL (8 mL/kg/hr). At the end of the operation, vital signs were as follows: BP 72/36 mmHg, HR 138 beats per minute, SpO_2 96%, $EtCO_2$ 30 mmHg, and BT 36 °C.

For the fourth operation on day 24 of age, anesthesia induction included sevoflurane 0.6% in oxygen and air (FiO_2 21%), midazolam 0.1 mg/kg IV, fentanyl 2 mcg/kg IV, and rocuronium 0.6 mg/kg IV. Maintenance included sevoflurane 0.6%, titrated fentanyl 1 mcg/kg/dose, rocuronium 0.6 mg/kg/dose IV, along with continuous dopamine infusion at 5 mcg/kg/min, and lipo-PGE1 at 1–3 ng/kg/min. An exploratory laparotomy with resection of necrotic small intestine and creation of an ileostomy and a jejunostomy was performed. The total anesthetic time was 317 minutes, with an operation time of 244 minutes and blood loss of 24 mL. This was corrected with a newborn cocktail (consisting of 20% albumin (10 mL) with Physio 140 (40 mL) up to 50 mL) (44 mL), leukoreduced irradiated red blood cells (36 mL), and fresh frozen plasma (FFP) (31 mL). Urine output was 21 mL (2.6 mL/kg/hr). At the end of the operation, vital signs were as follows: BP 63/24 mmHg, HR 157 beats

per minute, SpO_2 93%, $EtCO_2$ 30 mmHg, and BT 37.4 °C. ABG results were within normal range. The patient was transferred to the NICU and remained on ventilator support. All of the operations were uneventful.

On postoperative day 7 following the last operation, he was active, abdominal signs had improved, he remained euvolemic, the dopamine dose was weaned, and he continued to be on mechanical ventilation. The TTE showed a decrease in the PDA diameter from 4.6 to 1.6 mm, with SpO_2 ranging from 89% to 98% and MAP ranging from 51 to 66 mmHg. The dose of lipo-PGE_1 was adjusted to increase from 1 to 3 ng/kg/min, and hypercyanotic spells (tet spells) were closely observed in the event of PDA closure.

Discussion

This preterm male infant, born with TOF, PDA, and later developing NEC, posed significant anesthetic challenges. Anesthetic management involved maintaining heart rate, contractility, and preload to sustain cardiac output and euvolemia, avoiding increases in the PVR to the systemic vascular resistance (SVR) ratio in order to manage hypoxia (right-to-left shunting), preventing PDA closure, avoiding metabolic acidosis, hypoxemia, and hypercapnia, and promptly addressing hypercyanotic spells, especially given the infant's age[1]. Maintaining PDA patency is crucial for ensuring adequate pulmonary blood flow in TOF patients; therefore, lipo-PGE1 was administered immediately after birth[3].

NEC is a life-threatening condition, a multifactorial disease process caused by gastrointestinal ischemia, leading to inflammation and tissue necrosis[3,4]. Despite advancements in neonatal care, NEC remains a significant cause of mortality and morbidity for premature infants[5,6] with a case fatality rate of 15-30%[6]. NEC is more common in preterm infants, as in this case, with about 90% of cases occurring in this population. About 10% of cases also occur in full-term infants, often linked to congenital heart disease (CHD)[3,7]. The prevalence of NEC in CHD patients is estimated at 3.7%[6]. In preterm infants, the mechanism of NEC is thought to be primarily inflammation rather than hypoxia/ischemia, as observed in full-term infants with CHD[3]. Ductal-dependent CHD may lower diastolic gut perfusion pressures and limit systemic oxygenated blood flow, directly contributing to gastrointestinal hypoperfusion and ischemia and increasing the risk of NEC[4].

The use of lipo-PGE1 supports the maintenance of the medical palliative shunt until an interventional procedure can be carried out[3]. The optimal treatment of NEC remains challenging and requires interdisciplinary management. Decisions regarding the appropriate empiric antibiotic regimen, strict bowel rest with gastric decompression, and the timing of surgery are therefore discussed on a case-by-case basis[6,8].

At 18 days of age, this infant developed symptoms of lethargy, sepsis, and NEC stage IIIB. Surgical intervention is required in 20–40% of all cases of NEC due to insufficient clinical improvement with conservative therapy[6,9]. When pneumoperitoneum or other signs of intestinal perforation are present, surgical intervention is typically indicated and recommended as a last resort[6,9]. However, well-documented risk factors that predispose infants to NEC include being premature or of low birth weight, having CHD, sepsis, or hypotension[10,11]. Early diagnosis can lead to starting therapy sooner, minimizing intestinal injury, and enhancing clinical outcomes[11]. Management of this patient was multifaceted. Initially, the patient received medical management to stabilize and prepare him for the operating room[6,8].

The anesthetic management during the exploratory laparotomy aimed to maintain stable SpO_2 levels and prevent hemodynamic collapse. An FiO_2 of 21% was used to maintain SpO_2 between 90% and 95%, ensuring a balance between pulmonary and systemic blood flow[1,12]. Fluid management included glucose-containing fluids, crystalloids, and colloids to ensure adequate hydration

and electrolyte balance[1]. Vasopressors like dopamine and dobutamine were administered to support systemic circulation and maintain blood pressure[1,3]. Noradrenaline was not our first choice due to the risk of excessive peripheral vasoconstriction[1], which could increase pulmonary blood flow and reduce intestinal perfusion. We typically start with low (1–5 mcg/kg/min) to moderate doses of dopamine or dobutamine (5–10 mcg/kg/min). If vital signs remain unstable, adrenaline, noradrenaline, or vasopressin may be considered.

During the first operation, normal anion gap metabolic acidosis was observed, likely due to gastrointestinal losses causing bicarbonate depletion and replacement with chloride[13]. Despite electrolyte correction and fluid management, worsening acidosis and mild lactate elevation suggested reduced blood flow (tissue hypoperfusion) without evidence of widespread obstruction, as seen in volvulus or superior mesenteric artery thrombosis. Although blood loss was minimal, the ABG showed anemia, likely due to hemodilution from intraoperative fluid therapy, and the small amount of blood loss was difficult to measure. For fluid management, acetate Ringer's solution with 1% glucose was used as the perioperative maintenance fluid, while 5% albumin was administered as needed for body fluid losses, such as ascites and intestinal fluids. To correct metabolic acidosis, 7% sodium bicarbonate was used, which may have led to a rapid increase in sodium levels.

Anesthesia was maintained with remifentanil, fentanyl, midazolam, rocuronium, and low-dose sevoflurane to ensure adequate sedation and muscle relaxation while minimizing cardiovascular depression. Fentanyl is used for induction and postoperative pain management, while remifentanil is used for maintenance due to its rapid onset and short duration. Fentanyl's longer action makes it ideal for managing post-surgical pain, whereas remifentanil, which wears off quickly after stopping the infusion, is better suited for controlling pain and stress responses during surgery. In cases of acute pain during surgery, fentanyl is preferred for quick titration via IV bolus, as increasing remifentanil doses through infusion may take several minutes to take effect, especially in preterm infants[14,15].

The reduction of lipo-PGE1 aimed to improve systemic blood flow and NEC. While this adjustment was beneficial, continuous monitoring of PDA with TOF via SpO_2 and TTE remained crucial. A decrease in PVR can cause a significant left-to-right shunt, increasing pulmonary flow and reducing systemic perfusion, leading to oliguria, anuria, and metabolic acidosis. Therefore, regular ABG monitoring during the operation was essential[1].

Postoperatively, the patient required meticulous cardiovascular and respiratory support, including mechanical ventilation and lipo-PGE1 adjustments to maintain PDA patency while managing hypotension[1,8]. Dopamine and dobutamine stabilized hemodynamics, allowing gradual PDA reduction and the cautious weaning of vasopressors[1]. Hypercyanotic (tet) spells were monitored closely in case of PDA closure.

Management of hypercyanotic spells includes 100% oxygen, femoral artery compression, or knee-to-chest positioning. Morphine (0.05–0.1 mg/kg) and crystalloid (15–30 mL/kg) address symptoms, while sodium bicarbonate (1–2 mEq/kg) corrects acidosis. Phenylephrine (5–10 mcg/kg IV bolus or 2–5 mcg/kg/min infusion) and beta-blockers like propranolol (0.1 mg/kg) or esmolol (0.5 mg/kg, then 50–300 mcg/kg/min) may be used. Refractory cases may require extracorporeal membrane oxygenation[1].

The successful perioperative management of this complex case highlights the importance of a multidisciplinary approach, with key factors including vigilant cardiovascular monitoring, judicious vasopressor use, and precise lipo-PGE1 adjustments.

Conclusion

Managing a preterm infant with TOF and PDA complicated by NEC requires maintaining hemodynamic stability, avoiding increases in the PVR to SVR ratio, and reducing NEC risks. This case underscores the importance of maintaining PDA patency with lipo-PGE1, early NEC surgery, and meticulous postoperative care. A successful outcome depends on timely diagnosis, effective anesthetic management, surgery, and coordinated multidisciplinary care.

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Conflict of interest

All authors have no conflict of interest.

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