

CASE REPORT

Hemodynamic-Protective Anesthesia for Emergency Rigid Esophagoscopy in an Adult with Uncorrected Pulmonary Atresia with Ventricular Septal Defect, Aortopulmonary Collaterals, and Pneumonia: A Case Study

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ABSTRACT

Background. Adult survivors of uncorrected pulmonary atresia with ventricular septal defect (PA-VSD) depend on aortopulmonary collaterals, and a fall in systemic vascular resistance can increase right-to-left shunting. Emergency shared-airway esophagoscopy adds risks of aspiration, bleeding, and anticoagulation.

Objective. To describe the anesthetic management, hemodynamic course, and outcome of an adult with uncorrected PA-VSD, active pneumonia, and warfarin anticoagulation who needed emergency rigid esophagoscopy for an impacted denture.

Methods. This single case report follows the CARE guideline. A 51-year-old man (56 kg; ASA IV-E) presented four hours after swallowing a partial denture lodged at C4–C5. His cardiac index was 1.86 L/min/m², and *Klebsiella pneumoniae pneumonia* was present. Over four hours, fresh frozen plasma (10 mL/kg) and vitamin K lowered the INR from 2.60 to 1.42. Anesthesia used modified rapid sequence induction (etomidate, fentanyl, rocuronium), sevoflurane, a phenylephrine infusion, lung-protective ventilation, and invasive monitoring.

Results. At +30 minutes, mean arterial pressure (MAP) fell to 58 mmHg with SpO₂ 88%, and recovered to 68 mmHg within 90 seconds of a phenylephrine bolus (100 mcg). Thereafter MAP was 68–76 mmHg and SpO₂ 92–94%. The denture was removed en bloc after 22 minutes of manipulation, with 50 mL blood loss and no mucosal perforation. The patient was extubated on postoperative day 1 and discharged on day 8.

Conclusion. In this patient, early INR correction, etomidate-based induction, phenylephrine support, and normocapnic lung-protective ventilation accompanied an uncomplicated course. One case cannot show causation; larger series are needed.

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1. Introduction

Esophageal foreign bodies are a frequent emergency, and the proximal esophagus is the most common site of impaction.^{1,2} Ingested dentures are a recognized hazard because of their rigid, irregular shape, and delayed removal can cause serious complications.³ The European Society of Gastrointestinal Endoscopy recommends emergent endoscopy for sharp-pointing objects and urgent endoscopy within 24 hours for other impacted objects.⁴ Rigid esophagoscopy under general anesthesia remains effective, particularly when flexible removal fails, but it is not free of harm: in a series of 162 rigid esophagoscopies, perforation occurred in 8% and mortality was 2.5%.¹

The anesthesiologist shares the airway with the endoscopist, and secretions pooled above the impaction raise the risk of aspiration at induction. When the emergency occurs in an adult with congenital heart disease, the risk increases further. In a matched national cohort, adults with congenital heart disease had higher inpatient mortality (4.1% versus 3.6%) and perioperative

morbidity (21.4% versus 16.0%) after noncardiac surgery than matched controls.⁵ Reviews identify hemodynamic instability, arrhythmia, thromboembolism, and bleeding as the main concerns, with the highest risk in cyanotic and complex lesions.⁶⁻⁸

In pulmonary atresia with ventricular septal defect (PA-VSD), pulmonary blood flow is supplied by a patent ductus arteriosus and major aortopulmonary collateral arteries (MAPCAs).^{9,10} Few patients reach adulthood without intervention, and adult presentation is rare.^{11,12} Because both ventricles eject across a large septal defect, the balance between systemic and pulmonary flow depends on the ratio of systemic to pulmonary vascular resistance (SVR and PVR), and a fall in SVR increases right-to-left shunting and hypoxemia. In adults with Eisenmenger syndrome or similar right-to-left shunts, hypotension occurred in 26% of anesthetics and desaturation in 17%, and a vasopressor given at induction reduced hypotension.¹³ Chronic cyanosis is also associated with altered hemostasis and a higher risk of bleeding or thrombosis,¹⁴ and anticoagulation adds to the bleeding risk of rigid instrumentation. Reports of noncardiac surgery in

adults with unrepaired PA-VSD were isolated cases.¹⁵⁻¹⁷ The present report presents the case in the CARE format, with a timeline in relative days, serial laboratory and blood-gas values at six time points, and a physiologic interpretation of the intraoperative events. The objective of this report is to describe the anesthetic management, hemodynamic course, and outcome of this patient.

2. Methods

2.1. Report Design and Reporting Guideline

This is a single case report prepared in accordance with the CARE guideline.¹⁸ The data were taken from the clinical record. The patient was managed in 2024, and the report was prepared and approved for publication in 2026.

2.2. Setting

The patient was managed at Dr. Moewardi General Hospital and the Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia, by the departments of

anesthesiology and intensive care, otorhinolaryngology, cardiology, and pulmonology.

2.3. Patient Information and Case Selection

A 51-year-old man (56 kg, 162 cm) with long-standing uncorrected cyanotic congenital heart disease (PA-VSD, MAPCAs, patent ductus arteriosus, and moderate aortic regurgitation) and secondary polycythemia was receiving long-term warfarin. He presented four hours after accidentally swallowing a maxillary partial acrylic denture. The case was selected because an impacted cervical foreign body, uncorrected cyanotic heart disease, active pneumonia, and therapeutic anticoagulation coincided in one patient.

2.4. Case Timeline

The chronology is given in relative time in Table 1. Day 0 is the day of ingestion and presentation; POD is postoperative day.

Table 1. Clinical timeline of the case in relative time.

Time point	Clinical event	Investigation and result	Intervention
Day -6	Chest radiograph (reviewed at admission)	Cardiothoracic ratio 75%; perihilar haziness; right lower lobe consolidation	—
Day -1	Transthoracic echocardiography	PA-VSD, MAPCAs, PDA, moderate AR; CO 2.90 L/min; CI 1.86 L/min/m ² ; LVEF 52%; RVSP 78 mmHg	—
Day 0, hour 0	Accidental ingestion of a partial denture	—	—
Day 0, hour 4	Emergency department presentation with retrosternal and cervical pain	SpO ₂ 94% (room air); BP 105/60 mmHg; cervical radiograph: foreign body at C4–C5; INR 2.60	Ceftriaxone 2 g IV (continued from admission)
Day 0, hours 4–8	Four-hour preoperative optimization	INR 1.42; PT 15.2 s; CVP 8–10 mmHg	FFP 10 mL/kg (560 mL); vitamin K ₁ 5 mg IV; furosemide 20 mg IV; nebulized bronchodilators
Day 0, +0 to +62 min (operating room)	Rigid esophagoscopy under general anesthesia	MAP 58 mmHg and SpO ₂ 88% at +30 min; MAP 68–76 mmHg thereafter; blood loss 50 mL	Modified rapid sequence induction; phenylephrine infusion and 100 mcg bolus; sugammadex 4 mg/kg at +65 min
Day 0, +75 min	Cardiac intensive care unit admission, intubated	PaO ₂ 102 mmHg (FiO ₂ 0.50)	Mechanical ventilation; fentanyl 50 mcg/h
POD 1	Extubation to high-flow nasal oxygen	No stridor or desaturation	Sedation weaned
POD 3	Repeat endoscopy	Mucosal healing; no extravasation	Soft diet started
POD 7	Warfarin re-titration	INR 2.10	Warfarin resumed
POD 8	Discharge	WBC 7.40 × 10 ⁹ /L; neutrophils 58.0%	Outpatient cardiology and otorhinolaryngology follow-up arranged

Note: Abbreviations: AR, aortic regurgitation; BP, blood pressure; CI, cardiac index; CO, cardiac output; CVP, central venous pressure; FFP, fresh frozen plasma; INR, international normalized ratio; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; MAPCA, major aortopulmonary collateral arteries; PA-VSD, pulmonary atresia with ventricular septal defect; PDA, patent ductus arteriosus; POD, postoperative day; PT, prothrombin time; RVSP, right ventricular systolic pressure.

2.5. Clinical Findings and Diagnostic Assessment

The diagnosis was based on examination, cervical and chest radiography, transthoracic Doppler echocardiography, sputum and blood cultures, arterial blood gas analysis, and a coagulation profile. A right radial arterial catheter (20 G) and a right internal jugular triple-lumen central venous catheter (7 Fr) were placed under ultrasound guidance on arrival in the operating room. Monitoring also included five-lead electrocardiography with ST-segment analysis, pulse oximetry at two sites, and side-stream capnography. Body surface area was calculated by the Mosteller formula, MAP as diastolic pressure plus one third of pulse pressure, and cardiac index as cardiac output divided by body surface area. Systemic and pulmonary vascular resistances and shunt fraction were not measured, and a pulmonary artery catheter was not used because there was no native

pulmonary trunk and collateral vessels could be perforated.

2.6. Therapeutic Intervention

The multidisciplinary plan comprised four hours of preoperative optimization (warfarin reversal, diuresis, antibiotics, and bronchodilators), modified rapid sequence induction with a cuffed endotracheal tube, phenylephrine support, lung-protective ventilation, rigid esophagoscopy by the otorhinolaryngology team, sugammadex reversal, and postoperative intensive care. The regimens are listed in Table 4 and the intraoperative sequence in Table 5.

2.7. Outcome Measures and Follow-Up

Laboratory and blood-gas values were recorded at six time points: T0, admission; T1, after optimization and before induction; T2, +30 minutes (rescue vasopressor); T3, +44 minutes (denture extraction); T4, intensive care unit

(arterial gas at arrival, +75 minutes); and T5, discharge on POD 8. Intraoperative hemodynamics were recorded at the 11 time points of Table 5. Predefined events were right-to-left shunt escalation (SpO₂ below 85% or PaO₂ below 50 mmHg), hemodynamic collapse (MAP fall of more than 25% from baseline or need for an epinephrine infusion), esophageal perforation, and blood loss above 100 mL. Percentage change was calculated as $\Delta\% = [(T_n - T_0) / T_0] \times 100$. No inferential statistics were applied. Follow-up covered the hospital stay to discharge.

2.8. Ethics and Consent

The report was approved by the CMHC Research Center Ethics Committee, Indonesia (reference number 2026/4182) and conforms to the Declaration of Helsinki. Written informed consent for the recording of clinical data and for publication was obtained from the patient and his next of kin. All images were checked to remove identifiers.

3. Results

3.1. Presentation and Baseline Findings

The patient presented four hours after ingestion with sharp retrosternal and lower cervical pain that worsened on swallowing saliva. He denied choking, stridor, and hemoptysis. He was alert, with central and peripheral

cyanosis and digital clubbing. Heart rate was 63 beats/min, blood pressure 105/60 mmHg (MAP 75 mmHg), respiratory rate 20 breaths/min, temperature 37.1°C, and SpO₂ 94% on room air. Weight was 56 kg and height 162 cm (body mass index 21.34 kg/m²; body surface area 1.59 m² by the Mosteller formula). A continuous machinery murmur was heard at the apex and left upper sternal border, with a soft early diastolic murmur along the left sternal border. The jugular venous pulse was 4 cm above the sternal angle, with tender hepatomegaly and bilateral 2+ pretibial edema. Coarse bilateral crackles and right-sided rhonchi were present. The ASA physical status was IV-E and the NYHA class III. Baseline findings are in Table 2.

Echocardiography (day -1) showed pulmonary atresia, a large unrestrictive malalignment ventricular septal defect (22 mm), multiple MAPCAs arising from the descending thoracic aorta, a bidirectional patent ductus arteriosus, and moderate aortic regurgitation. Cardiac output was 2.90 L/min and the reported cardiac index 1.86 L/min/m², which is 25.6% below the lower reference limit of 2.50 L/min/m² (recalculation with the Mosteller body surface area gives 1.83 L/min/m²). The estimated right ventricular systolic pressure was 78 mmHg, below the systemic systolic pressure of 105 mmHg, and the left ventricular ejection fraction was 52%.

Table 2. Baseline demographic, cardiovascular, and anatomic-hemodynamic profile of the patient.

Domain	Patient finding	Reference value	Interpretation
Age and sex	51 years; male	—	Adult survivor of uncorrected cyanotic heart disease
Anthropometrics	56 kg; 162 cm; BMI 21.34 kg/m ² ; BSA 1.59 m ² (Mosteller)	BMI 18.5–24.9 kg/m ²	Normal body mass index
Presentation	Retrosternal pain, dysphagia, neck fullness	—	Onset 4 hours after swallowing a partial acrylic denture
Cardiac anatomy	PA with large malalignment VSD	Intact septum; patent pulmonary artery	Absent right ventricular to pulmonary artery continuity
Pulmonary blood supply	Descending aortic MAPCAs and PDA	Native pulmonary trunk	Pulmonary flow depends on systemic collaterals and PDA
Valves and shunt	Moderate AR; bidirectional PDA shunt	Competent valves	Diastolic run-off; fragile shunt balance
Cardiac output and index	CO 2.90 L/min; CI 1.86 L/min/m ²	CI 2.50–4.00 L/min/m ²	Reduced resting cardiac reserve
Ventricular function	LVEF 52%; RVSP 78 mmHg; RV hypertrophy	RVSP < 30 mmHg; LVEF > 55%	Elevated RV pressure, below systemic systolic pressure (105 mmHg)
Hemodynamics	HR 63 beats/min; BP 105/60 mmHg; MAP 75 mmHg	MAP 70–100 mmHg	Compensated resting blood pressure
Pulmonary and infectious status	K. pneumoniae bronchopneumonia	Clear lung fields	CTR 75%; perihilar haziness; right lower zone consolidation
Erythrocytosis	Hb 18.4 g/dL; Hct 56.0%; RBC 6.30 × 10 ¹² /L	Hb 13.5–17.5 g/dL	Secondary polycythemia from chronic hypoxemia
Oxygenation (room air)	pH 7.41; PaCO ₂ 36.0 mmHg; PaO ₂ 58.0 mmHg; SpO ₂ 94%	PaO ₂ 80–100 mmHg	Chronic hypoxemia
Anticoagulation	Warfarin; PT 26.4 s; INR 2.60; aPTT 38.0 s	INR 0.90–1.10	High bleeding risk for rigid instrumentation
Risk scores	ASA IV-E; NYHA III	ASA I–II; NYHA I	Emergency in a patient with limited reserve

Note: Values obtained at emergency department admission (T₀) unless stated. AR, aortic regurgitation; ASA, American Society of Anesthesiologists; BMI, body mass index; BP, blood pressure; BSA, body surface area; CI, cardiac index; CO, cardiac output; CTR, cardiothoracic ratio; Hb, hemoglobin; Hct, hematocrit; HR, heart rate; INR, international normalized ratio; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; MAPCA, major aortopulmonary collateral arteries; NYHA, New York Heart Association; PA, pulmonary atresia; PDA, patent ductus arteriosus; PT, prothrombin time; RBC, red blood cell count; RV, right ventricular; RVSP, right ventricular systolic pressure; VSD, ventricular septal defect.

3.2. Diagnostic Findings

A cervical radiograph showed an irregular radiopaque foreign body at C4–C5, at the level of the upper esophageal sphincter. The chest radiograph showed massive cardiomegaly (cardiothoracic ratio 75%), bilateral perihilar haziness, and dense right lower lobe consolidation (Figure 1). Sputum and peripheral blood cultures grew *Klebsiella pneumoniae* susceptible in vitro to

third-generation cephalosporins, piperacillin-tazobactam, and fluoroquinolones. Room-air blood gas values were pH 7.41, PaCO₂ 36.0 mmHg, PaO₂ 58.0 mmHg, bicarbonate 22.0 mmol/L, and base excess -1.40 mmol/L. Hemoglobin was 18.4 g/dL, leukocytes 14.6 × 10⁹/L (neutrophils 82.0%), platelets 246 × 10⁹/L, prothrombin time 26.4 s, INR 2.60, and aPTT 38.0 s. Serial laboratory values are given in Table 3.

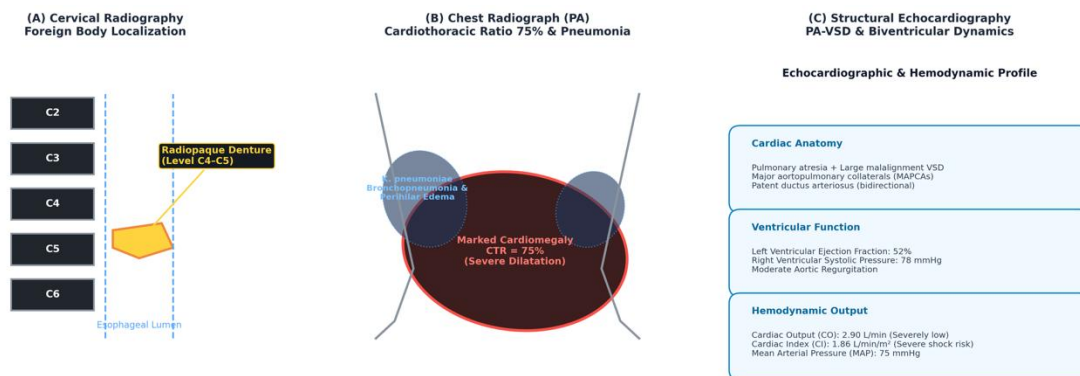


Figure 1. Schematic summary of the diagnostic findings. (A) Cervical radiograph: radiopaque foreign body at C4–C5. (B) Chest radiograph: cardiothoracic ratio 75% with perihilar haziness and right lower lobe pneumonia. (C) Echocardiographic findings: PA-VSD, MAPCAs, patent ductus arteriosus, moderate aortic regurgitation, cardiac output 2.90 L/min, cardiac index 1.86 L/min/m². CTR, cardiothoracic ratio; MAP, mean arterial pressure; MAPCA, major aortopulmonary collateral arteries; PA-VSD, pulmonary atresia with ventricular septal defect.

Table 3. Serial laboratory, arterial blood gas, and coagulation values with percentage change from baseline ($\Delta\%$).

Parameter (units)	Reference interval	T0	T1	T2	T3	T4	T5	$\Delta\%$ T0 to T5
Hemoglobin (g/dL)	13.5–17.5	18.4	17.6 (–4.3%)	17.4 (–5.4%)	17.5 (–4.9%)	17.8 (–3.3%)	18.1 (–1.6%)	–1.6%
Hematocrit (%)	40.0–50.0	56.0	53.5 (–4.5%)	52.8 (–5.7%)	53.2 (–5.0%)	54.0 (–3.6%)	55.2 (–1.4%)	–1.4%
Erythrocytes ($\times 10^{12}$ /L)	4.50–5.90	6.30	6.00 (–4.8%)	5.90 (–6.3%)	6.00 (–4.8%)	6.10 (–3.2%)	6.20 (–1.6%)	–1.6%
Leukocytes ($\times 10^9$ /L)	4.00–11.0	14.6	14.2 (–2.7%)	14.0 (–4.1%)	13.8 (–5.5%)	11.2 (–23.3%)	7.4 (–49.3%)	–49.3%
Neutrophils (%)	40.0–70.0	82.0	81.0 (–1.2%)	80.5 (–1.8%)	80.0 (–2.4%)	72.0 (–12.2%)	58.0 (–29.3%)	–29.3%
Platelets ($\times 10^9$ /L)	150–450	246	240 (–2.4%)	238 (–3.3%)	242 (–1.6%)	236 (–4.1%)	252 (+2.4%)	+2.4%
INR	0.90–1.10	2.60	1.42 (–45.4%)	1.40 (–46.2%)	1.38 (–46.9%)	1.40 (–46.2%)	2.10 (–19.2%)	–19.2%
Prothrombin time (s)	11.0–13.5	26.4	15.2 (–42.4%)	15.0 (–43.2%)	14.8 (–43.9%)	15.0 (–43.2%)	22.0 (–16.7%)	–16.7%
aPTT (s)	25.0–35.0	38.0	33.5 (–11.8%)	33.0 (–13.2%)	32.8 (–13.7%)	31.5 (–17.1%)	33.0 (–13.2%)	–13.2%
Arterial pH	7.35–7.45	7.41	7.42	7.38	7.40	7.39	7.42	—
PaCO ₂ (mmHg)	35.0–45.0	36.0	36.5	38.5	37.0	38.0	36.0	—
PaO ₂ (mmHg)	80.0–100.0	58.0 (RA)	72.0 (O ₂ 3 L/min)	62.0 (FiO ₂ 0.60)	65.0 (FiO ₂ 0.60)	102.0 (FiO ₂ 0.50)	60.0 (RA)	—
Bicarbonate (mmol/L)	22.0–26.0	22.0	22.4	22.1	22.3	22.0	23.0	—
Base excess (mmol/L)	–2.0 to +2.0	–1.40	–1.10	–1.80	–1.30	–1.40	–0.80	—
Sodium (mmol/L)	135–145	134	136 (+1.5%)	136 (+1.5%)	137 (+2.2%)	138 (+3.0%)	139 (+3.7%)	+3.7%
Potassium (mmol/L)	3.50–5.00	4.60	4.40 (–4.3%)	4.30 (–6.5%)	4.30 (–6.5%)	4.20 (–8.7%)	4.40 (–4.3%)	–4.3%
Urea nitrogen (mg/dL)	10.0–50.0	38.0	36.0 (–5.3%)	35.0 (–7.9%)	35.5 (–6.6%)	32.0 (–15.8%)	26.0 (–31.6%)	–31.6%
Creatinine (mg/dL)	0.60–1.10	1.10	1.08 (–1.8%)	1.05 (–4.5%)	1.06 (–3.6%)	1.02 (–7.3%)	0.95 (–13.6%)	–13.6%
Random glucose (mg/dL)	70.0–140.0	128	122 (–4.7%)	134 (+4.7%)	130 (+1.6%)	118 (–7.8%)	104 (–18.8%)	–18.8%

Note: Timepoints: T0, admission; T1, after optimization before induction; T2, +30 min; T3, +44 min (denture extraction); T4, intensive care unit (arterial gas obtained at arrival, +75 min, on FiO₂ 0.50 during mechanical ventilation); T5, discharge on POD 8. Percentage change ($\Delta\%$) is relative to T0. Arterial gas values were obtained from the radial arterial catheter. RA, room air.

3.3. Treatment Course and Outcome

During the four-hour optimization, the patient received fresh frozen plasma 10 mL/kg (560 mL) and vitamin K₁ 5 mg intravenously, and the INR fell from 2.60 to 1.42 ($\Delta\%$ –45.4%) with a prothrombin time of 15.2 s ($\Delta\%$ –42.4%). Furosemide 20 mg intravenously was given with central

venous pressure targeted at 8–10 mmHg and maintenance crystalloid restricted to less than 500 mL, in addition to the plasma. Ceftriaxone was continued, with a booster dose 30 minutes before induction, and nebulized ipratropium 0.5 mg and salbutamol 2.5 mg were given with chest physiotherapy (Table 4).

Table 4. Preoperative optimization and anesthetic plan with regimens and rationale.

Domain	Intervention and regimen	Target	Rationale
Infection	Ceftriaxone 2 g IV every 24 h (continued from admission)	Eradication of <i>K. pneumoniae</i>	Time-dependent bactericidal activity (fT>MIC)

Domain	Intervention and regimen	Target	Rationale
Endocarditis prophylaxis	Ceftriaxone 2 g IV, single booster dose 30 min before induction	Less bacteremia during instrumentation	Unrepaired cyanotic heart disease is a high-risk lesion; the booster was a team decision
Coagulation	FFP 10 mL/kg (560 mL) and vitamin K ₁ 5 mg slow IV	INR < 1.50 before esophagoscopy (achieved 1.42)	Replaces vitamin K-dependent factors II, VII, IX, and X
Preload and edema	Furosemide 20 mg slow IV; maintenance crystalloid < 500 mL/24 h, in addition to 560 mL plasma	CVP 8–10 mmHg	Reduces perihilar congestion without hypovolemia
Airway reactivity	Nebulized ipratropium 0.5 mg and salbutamol 2.5 mg every 6 h; chest physiotherapy	Lower airway resistance; secretion clearance	Bronchodilation lowers airway pressures
Heart rate	Oral bisoprolol 2.5 mg daily, taken with sips of water	HR 60–65 beats/min	Avoids tachycardia and preserves diastolic filling
Blood products	Packed red cells 2 units crossmatched; platelets on standby	Available in the operating room	Anticipates mucosal hemorrhage in a patient with erythrocytosis
Induction	Fentanyl 1.5 mcg/kg (85 mcg), etomidate 0.2 mg/kg (11.2 mg), rocuronium 0.9 mg/kg (50 mg); cricoid pressure	Intubation within 60 s; no fall in SVR	Etomidate has little effect on vascular tone; rocuronium avoids succinylcholine; cricoid pressure limits regurgitation
Vasopressor support	Phenylephrine infusion 0.1–0.5 mcg/kg/min; boluses of 50–100 mcg available	MAP 65–80 mmHg	α 1-agonist raises SVR relative to PVR
Ventilation	VT 330 mL (5.6 mL/kg predicted body weight); PEEP 5 cmH ₂ O; FiO ₂ 0.60; Pplat $\leq$ 22 cmH ₂ O	ETCO ₂ 35–40 mmHg; PaO ₂ > 60 mmHg	Avoids overdistension and hypercapnia that raise PVR
Postoperative care	Cardiac intensive care; sedated ventilation; planned weaning; high-flow nasal oxygen	No re-bleeding	Continuous invasive monitoring; delayed extubation

Note: The plan was agreed by anesthesiology, adult congenital cardiology, pulmonology, otorhinolaryngology, and intensive care. CVP, central venous pressure; ETCO₂, end-tidal carbon dioxide; FFP, fresh frozen plasma; HR, heart rate; INR, international normalized ratio; MAP, mean arterial pressure; PEEP, positive end-expiratory pressure; Pplat, plateau pressure; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance; VT, tidal volume.

In the operating room the patient was placed in 15° reverse Trendelenburg and preoxygenated for five minutes (heart rate 64 beats/min, blood pressure 104/59 mmHg, SpO₂ 98%). Induction used fentanyl, etomidate (over 30 seconds), and rocuronium with cricoid pressure. Laryngoscopy gave a Cormack–Lehane grade I view, and an 8.0-mm cuffed tube was placed at 23 cm at the lip (cuff pressure 25 cmH₂O; ETCO₂ 37 mmHg). Ventilation was volume controlled with a tidal volume of 330 mL (5.6 mL/kg predicted body weight of 58.7 kg), a rate of 12–14 breaths/min, PEEP of 5 cmH₂O, FiO₂ of 0.60, and a plateau pressure of 22 cmH₂O or less. Anesthesia was maintained with sevoflurane at 1.0–1.5 MAC, and a phenylephrine infusion was started at 0.1 mcg/kg/min when the scope was inserted at +22 minutes.

At +30 minutes, with the scope advancing to C4–C5, blood pressure fell to 88/43 mmHg (MAP 58 mmHg; Δ % –22.7% from baseline) and SpO₂ to 88%, with PaO₂ 62 mmHg. A phenylephrine bolus of 100 mcg was given through the central line and the infusion was increased to 0.5 mcg/kg/min. MAP reached 68 mmHg within 90 seconds and SpO₂ returned to 92%. Thereafter MAP was 68–76 mmHg and SpO₂ 92–94% until the end of the procedure (Table 5 and Figure 4). The thresholds for shunt escalation (SpO₂ below 85% or PaO₂ below 50 mmHg) and for hemodynamic collapse (MAP fall above 25%) were not reached, and no adverse drug reaction was observed.

Table 5. Intraoperative procedural, hemodynamic, and pharmacological timeline.

Time (min)	Phase and maneuver	Clinical action and monitoring	Hemodynamics	Ventilation	Pharmacotherapy and resuscitation
0	Operating room entry and baseline monitoring	Transfer to table; 5-lead ECG, pulse oximeter, noninvasive BP cuff. Ultrasound-guided radial arterial line and internal jugular CVC placed.	HR 63 beats/min; BP 105/60 mmHg (MAP 75 mmHg); SpO ₂ 94% (room air); CVP 9 mmHg	Spontaneous ventilation; room air	Phenylephrine primed on a syringe driver (20 mcg/mL)
+10	Preoxygenation	15° reverse Trendelenburg; 100% O ₂ at 10 L/min by tight mask for 5 min	HR 64; BP 104/59 (MAP 74); SpO ₂ 98%; CVP 9	Spontaneous breathing; 100% O ₂	Airway and thoracotomy trays on standby
+15	Induction	Modified rapid sequence: fentanyl 1.5 mcg/kg (85 mcg), etomidate 0.2 mg/kg (11.2 mg) over 30 s, rocuronium 0.9 mg/kg (50 mg); cricoid pressure	HR 60; BP 96/54 (MAP 68); SpO ₂ 96%; CVP 9	Apneic oxygenation	Smooth loss of consciousness
+18	Laryngoscopy and intubation	Macintosh 3 blade; Cormack–Lehane grade I; 8.0-mm cuffed ETT at 23 cm	HR 68; BP 102/58 (MAP 73); SpO ₂ 95%; CVP 10	TV 330 mL; rate 14/min; PEEP 5 cmH ₂ O; FiO ₂ 0.60; cuff 25 cmH ₂ O; ETCO ₂ 37 mmHg	—
+22	Rigid esophagoscopy begins	Scope introduced through the right oral commissure, contralateral to the ETT	HR 65; BP 98/56 (MAP 70); SpO ₂ 92%; CVP 9	Sevoflurane 1.0–1.5 MAC in air–O ₂ (FiO ₂ 0.60); ETCO ₂ 37; Pplat 21 cmH ₂ O	Phenylephrine infusion started at 0.1 mcg/kg/min
+30	Fall in blood pressure and rescue vasopressor	Scope at C4–C5 with the denture in view; arterial blood gas sampled	HR 72; BP 88/43 (MAP 58); SpO ₂ 88%; CVP 8	ETCO ₂ 39; Pplat 22; PaO ₂ 62 mmHg	Phenylephrine 100 mcg IV bolus; infusion raised to 0.5 mcg/kg/min; MAP 68 mmHg within 90 s; SpO ₂ 92%

Time (min)	Phase and maneuver	Clinical action and monitoring	Hemodynamics	Ventilation	Pharmacotherapy and resuscitation
+44	Denture disimpaction and extraction	Hydro-dissection with 10 mL saline; denture grasped and removed en bloc	HR 66; BP 106/58 (MAP 74); SpO ₂ 92%; CVP 9	ETCO ₂ 38; Pplat 21; PaO ₂ 65 mmHg	Blood loss 50 mL (mucosal ooze)
+55	Hemostasis check and mucosal survey	Scope reintroduced; superficial abrasions; no perforation or active bleeding	HR 62; BP 105/60 (MAP 75); SpO ₂ 93%; CVP 9	ETCO ₂ 37; Pplat 20	Phenylephrine reduced to 0.3 mcg/kg/min
+62	End of endoscopy	Scope withdrawn; oropharyngeal suction under vision; ETT position confirmed; volatile agent stopped	HR 60; BP 106/60 (MAP 75); SpO ₂ 93%; CVP 9	100% O ₂ ; mechanical ventilation continued	Crystalloid 450 mL; urine output 85 mL
+65	Neuromuscular reversal and sedation	Sugammadex 4 mg/kg (224 mg); TOF ratio 1.0 within 90 s; kept intubated and sedated	HR 60; BP 108/60 (MAP 76); SpO ₂ 93%; CVP 9	SIMV; TV 330 mL; PEEP 5; FiO ₂ 0.50	Fentanyl 50 mcg/h; phenylephrine 0.1 mcg/kg/min
+75	Handover to cardiac intensive care	ICU ventilator; arterial and central lines zeroed; handover	HR 64; BP 108/62 (MAP 77); SpO ₂ 94% (FiO ₂ 0.50); CVP 9	Volume control; TV 330 mL; PEEP 5; FiO ₂ 0.50; ETCO ₂ 38	ABG: pH 7.39, PaCO ₂ 38 mmHg, PaO ₂ 102 mmHg, HCO ₃ ⁻ 22 mmol/L, lactate 1.2 mmol/L

Note: Time is in minutes from entry into the operating room (0 min). The rigid esophagoscope was in place from +22 to +44 min (extraction time 22 min), reintroduced at +55 min, and withdrawn at +62 min (total scope time about 40 min). ABG, arterial blood gas; BP, blood pressure; CVC, central venous catheter; CVP, central venous pressure; ETCO₂, end-tidal carbon dioxide; ETT, endotracheal tube; FRC, functional residual capacity; HR, heart rate; MAP, mean arterial pressure; PEEP, positive end-expiratory pressure; Pplat, plateau pressure; SVR, systemic vascular resistance; TOF, train-of-four; TV, tidal volume.

The denture was seen impacted against the lateral mucosal folds at C4–C5, producing subtotal obstruction with mucosal erythema and edema (Figure 2). After hydro-dissection with 10 mL of saline, it was grasped with forceps and removed en bloc at +44 minutes (Figure 3), so that extraction took 22 minutes. The scope was reintroduced at

+55 minutes for a mucosal survey, which showed superficial erosions and submucosal ecchymosis without perforation or active bleeding, and was withdrawn at +62 minutes (total scope time about 40 minutes). Blood loss was 50 mL, crystalloid 450 mL, and urine output 85 mL.

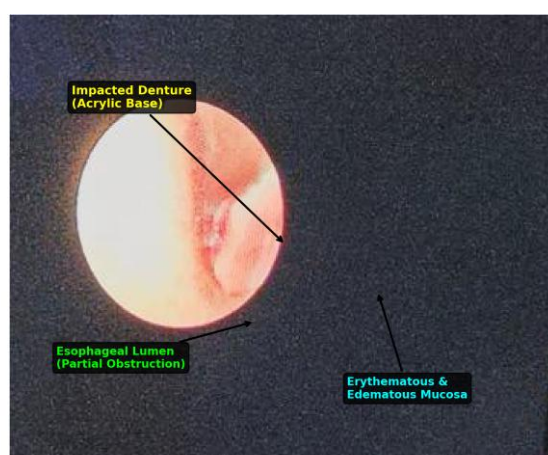


Figure 2. Intraoperative rigid esophagoscopy showing the impacted partial acrylic denture at the upper esophageal sphincter (C4–C5) with surrounding mucosal erythema and edema.



Figure 3. Partial acrylic denture after en-bloc removal by rigid esophagoscopy, displayed on sterile gauze beside the endoscopic instruments (measured 4.2 × 2.8 × 1.1 cm).

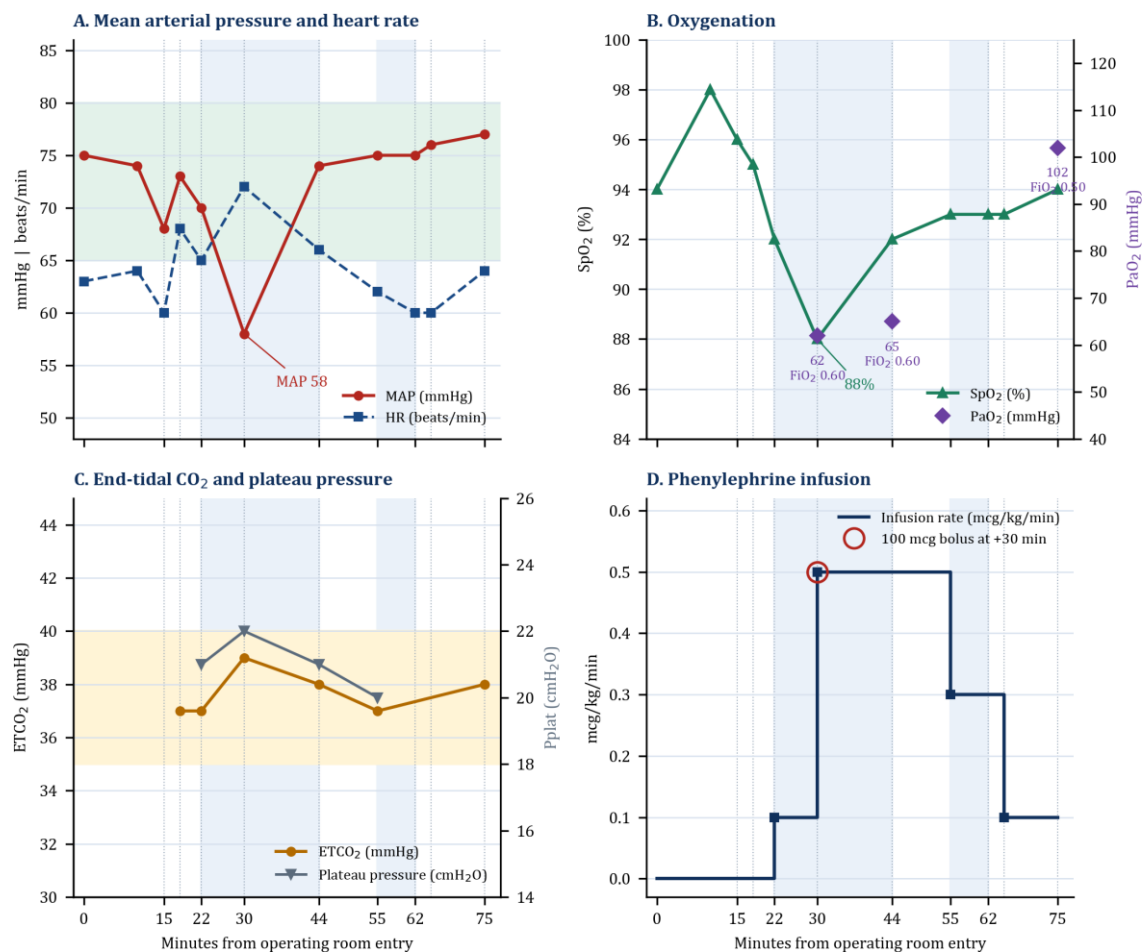


Figure 4. Intraoperative course plotted from Table 5. (A) MAP and heart rate; the green band marks a MAP of 65–80 mmHg. (B) SpO₂ and the PaO₂ values measured at +30, +44, and +75 minutes. (C) ETCO₂ (shaded band 35–40 mmHg) and plateau pressure. (D) Phenylephrine infusion rate (steps hold the last documented rate) and the 100 mcg bolus. Shaded areas in the plots mark the periods with the esophagoscope in place (+22 to +44 and +55 to +62 minutes); dotted lines mark induction (+15), intubation (+18), scope insertion (+22), rescue bolus (+30), extraction (+44), scope withdrawal (+62), sugammadex (+65), and ICU arrival (+75). ETCO₂, end-tidal carbon dioxide; HR, heart rate; MAP, mean arterial pressure; Pplat, plateau pressure.

Neuromuscular blockade was reversed at +65 minutes with sugammadex 4 mg/kg (224 mg), and the train-of-four ratio reached 1.0 within 90 seconds. The patient was kept intubated and sedated with fentanyl 50 mcg/h and moved to the cardiac intensive care unit at +75 minutes. The blood gas on arrival (FiO₂ 0.50) showed pH 7.39, PaCO₂ 38.0 mmHg, PaO₂ 102.0 mmHg, bicarbonate 22.0 mmol/L, and lactate 1.2 mmol/L. Low-dose phenylephrine (0.1 mcg/kg/min) was weaned off within four hours. Sedation was weaned on POD 1, and the patient was extubated to high-flow nasal oxygen (30 L/min, FiO₂ 0.40) without stridor or desaturation. Over the next 48 hours he changed to low-flow oxygen and then room air, with a room-air SpO₂ of 94%. Oral liquids began on POD 2, and a soft diet followed repeat endoscopy on POD 3 that confirmed mucosal healing. Ceftriaxone was continued for seven days; leukocytes fell to $7.40 \times 10^9/L$ (neutrophils 58.0%) and the chest radiograph cleared. Warfarin was re-titrated to an INR of 2.10 by POD 7, and the patient was discharged on POD 8 with outpatient cardiology and otorhinolaryngology follow-up.

4. Discussion

4.1. Principal Lesson of the Case

An adult with uncorrected PA-VSD and MAPCAs underwent emergency shared-airway rigid esophagoscopy with one brief fall in MAP and SpO₂ that responded to

phenylephrine within 90 seconds. The patient recovered without perforation, major bleeding, or cardiovascular collapse. This is one uncontrolled case, so the findings show an association with the measures used, not that they caused the outcome.

4.2. Comparison with the Published Literature

Noncardiac surgery in adults with unrepaired PA-VSD is reported only as isolated cases, including emergency appendectomy, hysterectomy under combined spinal-epidural anesthesia, and laparoscopic cholecystectomy in a patient with pulmonary atresia (Table 6).^{12,15,16} These reports share the aim of avoiding changes in SVR and PVR, but none involved a shared airway or anticoagulation. In a single-center series of 53 anesthetics in patients with Eisenmenger physiology or similar shunts, hypotension occurred in 26% and desaturation in 17%, and a vasopressor at induction reduced hypotension; etomidate tended to give more stable hemodynamics than other agents.¹³ Our patient had one hypotensive and desaturation episode, but rates cannot be compared at $n = 1$. At the population level, adults with congenital heart disease have higher mortality and morbidity after noncardiac surgery,⁵ and a single survivor cannot be set against cohort rates. For the procedure, rigid esophagoscopy removed foreign bodies in 343 of 400 patients in one series, with one perforation and one fatal aorto-esophageal fistula,¹⁹ and perforation occurred in 8%

of 162 procedures in another, more often when the object lay outside the proximal esophagus.¹ Our impaction was in the proximal esophagus. The position of the collaterals relative to the esophagus was not defined here, but

retroesophageal MAPCAs are described in PA-VSD,²⁰ and unclosed collaterals were associated with more transfusion in children with tetralogy of Fallot,²¹ so vascular injury remains a concern.

Table 6. Published cases and series relevant to the present case.

Reference	Design and sample	Population and procedure	Main findings	Relevance to the present case
Chatzidaki et al., 2009 ¹²	Case report	Adult, unrepaired PA-VSD; emergency appendectomy	Successful outcome; avoid changes in SVR and PVR from acidosis, hypothermia, hypercarbia, and high airway pressure	Closest physiology; no shared airway or anticoagulation
Agarwal et al., 2010 ¹⁶	Case report	Adult with PA-VSD; hysterectomy	Managed with combined spinal-epidural anesthesia	Regional technique not possible for esophagoscopy
Andrews, 1999 ¹⁵	Case report	24-year-old woman, unrepaired tetralogy of Fallot with pulmonary atresia; laparoscopic cholecystectomy	General endotracheal anesthesia; uncomplicated course	Similar principles; elective surgery
Bennett et al., 2014 ¹³	Retrospective; 33 patients, 53 anesthetics	Eisenmenger syndrome or similar shunt; noncardiac surgery	Hypotension 26%; desaturation 17%; vasopressor at induction reduced hypotension; 30-day mortality 3.8%	Supports vasopressor support and etomidate
Maxwell et al., 2013 ⁵	Registry; 10,004 ACHD patients and 37,581 matched controls	Adults with congenital heart disease; noncardiac surgery	Inpatient mortality 4.1% versus 3.6%; morbidity 21.4% versus 16.0%	Population-level risk
Athanassiadi et al., 2002 ¹⁹	Retrospective; 400 patients	Esophageal foreign bodies	Rigid esophagoscopy in 343 (85.7%); one perforation; one fatal aortoesophageal fistula	Procedural context
Alexandre et al., 2024 ¹	Retrospective; 162 rigid esophagoscopies	Foreign bodies; 80% in the proximal esophagus	Perforation 8%; mortality 2.5%	Proximal impaction as in this case
Present case	Single case report	PA-VSD, MAPCAs, pneumonia, warfarin; rigid esophagoscopy	MAP 58 mmHg and SpO ₂ 88% at +30 min, reversed in 90 s; no perforation; discharge on POD 8	—

Note: ACHD, adult congenital heart disease; MAP, mean arterial pressure; MAPCA, major aortopulmonary collateral arteries; PA-VSD, pulmonary atresia with ventricular septal defect; POD, postoperative day; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance.

4.3. Pathophysiological Interpretation

With a large ventricular septal defect, the ratio of systemic to pulmonary flow (Q_p/Q_s) approximates SVR/PVR when the pressure gradients across the two circuits are equal. At +30 minutes, MAP fell to 58 mmHg while SpO₂ fell to 88% and PaO₂ to 62 mmHg, which is consistent with increased right-to-left shunting, although shunt fraction was not measured. Phenylephrine, an α_1 -agonist, restored MAP and SpO₂ within 90 seconds, which fits a rise in SVR relative to PVR. Phenylephrine can also raise PVR, and the net effect of a vasopressor on the pulmonary circulation varies between patients,²² so the response here was observed, not predicted. Sevoflurane vasodilation, scope stimulation, and diuresis with fluid restriction may also have contributed to the fall. Propofol lowers SVR and

preload and may depress contractility at high doses, which is why etomidate was chosen; this agrees with the tendency toward greater stability with etomidate in shunt physiology.¹³ A single dose of etomidate suppresses adrenal function, although its effect on mortality in critically ill patients is unclear.²³ Hypoventilation causes hypercapnia and acidosis, which both raise PVR, and hypoxia triggers hypoxic pulmonary vasoconstriction, so normocapnia and a low plateau pressure were targeted (Figure 5). Pneumonia added an intrapulmonary shunt to the cardiac shunt, and ceftriaxone, a time-dependent β -lactam, was chosen for the susceptible isolate in line with guidance on community-acquired pneumonia.²⁴ Reversal of rocuronium with sugammadex avoided the muscarinic effects of neostigmine, but because the patient stayed intubated and sedated, emergence was not tested.

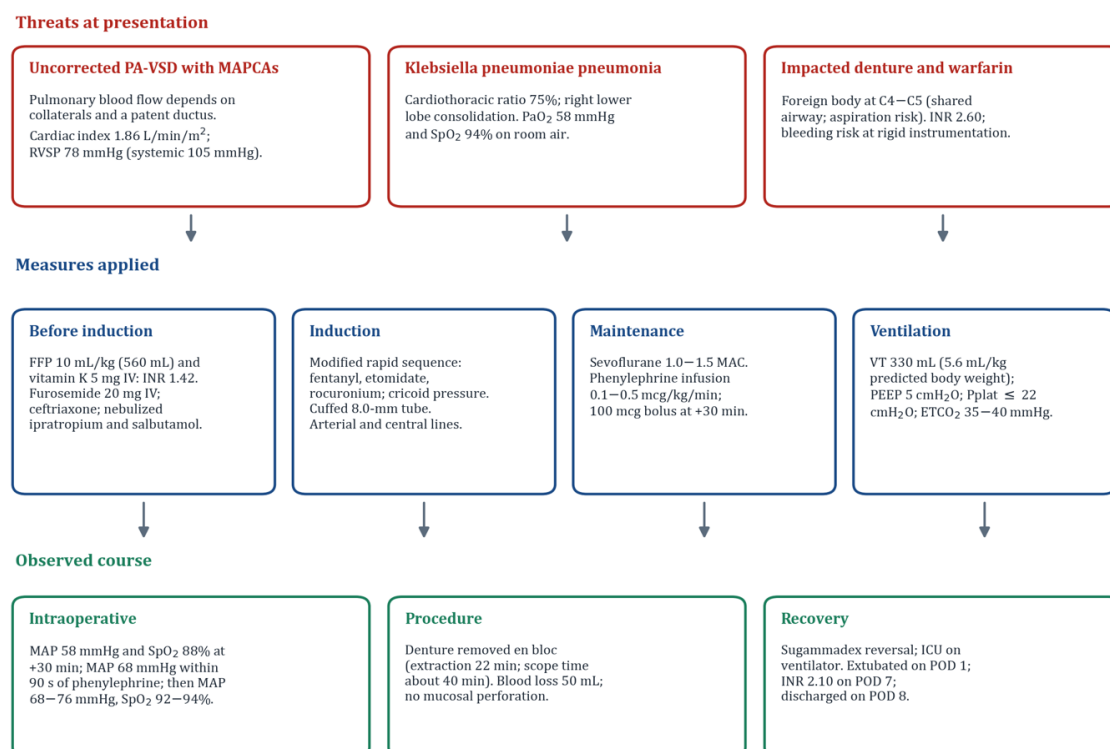


Figure 5. Conceptual summary of the case (schematic). The threats at presentation, the measures applied, and the observed course. FFP, fresh frozen plasma; INR, international normalized ratio; MAP, mean arterial pressure; PA-VSD, pulmonary atresia with ventricular septal defect; MAPCA, major aortopulmonary collateral arteries; PBW, predicted body weight; POD, postoperative day; RVSP, right ventricular systolic pressure; VT, tidal volume.

4.4. Implications for Practice

These observations suggest considerations that may be of use in similar patients, not a protocol. They are early multidisciplinary risk stratification;^{25,26} invasive arterial monitoring before induction; a vasopressor ready or running before induction and scope insertion; avoidance of agents that lower SVR abruptly; normocapnia with a low plateau pressure; and a plan for anticoagulation reversal that accounts for volume and delay. Plasma gave an INR of 1.42 but added 560 mL of volume in a patient with pulmonary congestion and took four hours, so the procedure began about eight hours after ingestion, beyond the emergent window of the guideline. Prothrombin complex concentrate, which corrects the INR faster with less volume overload than plasma,²⁷ was not available. Specialist guidance on bleeding in patients on oral anticoagulants may help in planning.²⁸ Transferability is limited to patients with similar physiology.

4.5. Strengths

Strengths are the serial documentation of hemodynamics, blood gases, and laboratory values, the objective confirmation of the diagnosis by imaging and cultures, invasive monitoring from before induction, and follow-up to discharge.

4.6. Limitations

This is a single case without a control, from one center, and the data were taken from the clinical record. SVR, PVR, and shunt fraction were not measured, and several plotted time points have no blood-gas sampling. A pulmonary artery catheter was not used. Single-dose etomidate was given to a patient with active Gram-negative infection, positive blood cultures, and a low cardiac index; no adrenal function testing is reported, and its contribution to the +30-minute event cannot be judged. Prothrombin complex

concentrate was unavailable, and follow-up ended at discharge.

4.7. Recommendations for Future Work

Multicenter registries or case series of adults with unrepaired cyanotic heart disease undergoing emergency endoscopic procedures are needed, with prospective recording of echocardiographic estimates of vascular resistance, shunt fraction, vasopressor doses, and anticoagulation strategy.

5. Conclusion

Emergency rigid esophagoscopy for an impacted denture was completed without perforation, major bleeding, or cardiovascular collapse in an adult with uncorrected PA-VSD, aortopulmonary collaterals, pneumonia, and warfarin anticoagulation, after INR correction, an etomidate-based induction, phenylephrine support, and normocapnic lung-protective ventilation. A brief fall in MAP and SpO₂ at +30 minutes responded to a phenylephrine bolus within 90 seconds. These observations come from one patient and do not show that the measures caused the outcome.

6. Declarations

Ethics Approval and Consent to Participate: This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under reference number 2026/4182 and conforms to the Declaration of Helsinki.

Consent for Publication: Written informed consent for publication of this case and the accompanying images was obtained from the patient and his next of kin. The images contain no patient identifiers.

Availability of Data and Materials: The de-identified data supporting this report are contained within the article and

are available from the corresponding author on reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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Author contribution statement

ES: Conceptualization, Investigation, Writing – original draft. BA: Investigation, Data curation, Project administration. ASN: Supervision, Formal analysis, Writing – original draft, Writing – review & editing. All authors approved the final version of manuscript.

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

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