RESEARCH PAPER

Combined Spinal-Epidural Anaesthesia for Bipolar Hemiarthroplasty in an Emaciated Octogenarian with a Giant Ascending Aortic Aneurysm and Multisystem Comorbidities: A Case Report

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ABSTRACT

Background: Unrepaired ascending aortic aneurysms presenting for urgent non-cardiac surgery represent one of the most formidable challenges in perioperative medicine. The paramount concern is prevention of catastrophic aortic dissection or rupture triggered by acute haemodynamic instability. When compounded by multisystem organ compromise, such cases are frequently declined by tertiary referral centres.

Case Presentation: An 86-year-old female (weight 35 kg; BMI 15.2 kg/m²; NYHA Class III; ASA Physical Status IV) presented with a two-week-old femoral neck fracture requiring bipolar hemiarthroplasty. Key pre-operative parameters included: aortic root diameter 5.4 cm on echocardiography; maximum ascending aortic diameter 8.3 cm on CT aortogram; severe concentric left ventricular hypertrophy; Ejection fraction 45%; moderate aortic regurgitation (AR); bilateral pleural effusions; serum creatinine 1.8 mg/dL (eGFR 28 mL/min/1.73m²); acute kidney injury (AKI); Anemia and hypothyroidism. Following a comprehensive multidisciplinary team (MDT) evaluation — including Cardiology, Pulmonology, Vascular Surgery, and Cardiothoracic Surgery (providing cardiopulmonary bypass [CPB] standby) — a combined spinal-epidural (CSE) technique was selected. At L3-L4, a low-dose subarachnoid block (1.5 mL 0.5% hyperbaric bupivacaine with fentanyl 10 mcg) was administered; an epidural catheter advanced 4 cm from L2-L3 provided titratable extension with an intraoperative 0.25% ropivacaine infusion (4–6 mL/hr). Haemodynamic stability was achieved with titrated noradrenaline (0.04–0.12 mcg/kg/min, MAP maintained 74–88 mmHg) and a zero-crystalloid, packed red blood cell (PRBC)-only fluid strategy (2 units transfused).

Outcome: The procedure was completed without haemodynamic crisis or CPB activation. Post-operative creatinine remained stable at 1.9 mg/dL (baseline 1.8 mg/dL). The patient was discharged from the CT-ICU on post-operative day 3, mobilised from day 7, and discharged home on day 14.

Conclusion: A titratable CSE technique with vasopressor-guided haemodynamic management, restrictive fluid strategy, and CPB standby constitutes a viable anaesthetic framework for patients with giant ascending aortic aneurysms and multisystem compromise presenting for urgent non-cardiac surgery. Prospective registry data are needed to validate these principles across similar presentations.

Keywords: Ascending aortic aneurysm; combined spinal-epidural anaesthesia; bipolar hemiarthroplasty; haemodynamic stability; noradrenaline; restrictive fluid strategy; neuraxial anaesthesia; multidisciplinary team

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1. INTRODUCTION

Ascending aortic aneurysms with maximum diameters exceeding 5.0–5.5 cm carry a substantial annual risk of dissection or rupture; diameters above 6 cm are associated with event rates approaching 6–8% per year, with in-hospital mortality exceeding 25% following acute dissection [1,2]. When such patients require urgent non-cardiac surgery, the perioperative team confronts a compound dilemma: the surgical pathology demands timely intervention, yet the procedure itself may

precipitate aortic catastrophe through haemodynamic instability.

Femoral neck fractures in the elderly carry a 30-day mortality of 5–10%, rising steeply with each additional comorbidity and each week of delay [3]. Prolonged immobility perpetuates a cycle of severe pain, haematological compromise, pulmonary deterioration, and nutritional decline — all of which compound pre-existing organ dysfunction and increase operative risk with time. The optimal anaesthetic strategy must therefore achieve two simultaneous objectives: haemodynamic stability sufficient to protect a pathologically fragile aortic wall, and a quality of surgical anaesthesia sufficient to allow timely orthopaedic intervention.

General anaesthesia (GA) in aortic aneurysmal disease carries well-defined hazards. Direct laryngoscopy and tracheal intubation produce a sympathoadrenal surge with transient but potentially fatal elevations in systolic blood pressure (20–40%) and heart rate (15–25 bpm) that directly increases aortic wall stress in proportion to dP/dt [4]. Volatile anaesthetic agents exert negative inotropy deleterious in a ventricle already compromised by concentric hypertrophy and a volume-loading regurgitant lesion [5]. Positive-pressure ventilation (PPV) impairs venous return and may exacerbate pre-existing pleural effusions, increasing pulmonary vascular resistance and worsening right ventricular afterload [5]. Combined spinal-epidural (CSE) anaesthesia, by contrast, provides dense, reliable surgical block through progressive, controllable sympatholysis while eliminating each of the above hazards [6,7].

We present a case report, prepared in full accordance with the CARE (CAse REport) guidelines [8], describing the successful perioperative management of an 86-year-old, 35 kg, ASA Physical Status IV patient with a giant ascending aortic aneurysm (maximum diameter 8.3 cm) and multisystem organ compromise, in whom a titratable CSE technique with vasopressor-guided haemodynamic management enabled safe completion of bipolar hemiarthroplasty after the case had been declined by multiple tertiary centres.

2. CASE REPORT

2.1 Patient Information, Presenting Concerns, and Clinical Perspective

An 86-year-old female (weight 35 kg, height 148 cm, BMI 15.2 kg/m²) with a known history of aortic aneurysmal disease, hypothyroidism, and chronic anticoagulant therapy (warfarin) presented to the emergency department two weeks following a fall at home. The fracture had rendered her completely bedridden, causing severe pain and progressive functional deterioration.

The patient and her family expressed a clear and consistent priority for surgical intervention to relieve her pain and restore some degree of functional independence. They were counselled explicitly — jointly by the anaesthesiology and orthopedic surgery teams — regarding the significant perioperative risks including intraoperative aortic rupture or dissection, cardiac arrest, renal failure, stroke, and death. The consent process was conducted over two sessions and was video-documented with the patient's and family's informed agreement.

The complete CARE clinical timeline from symptom onset to post-operative discharge is presented in **Table 1.**

Table 1. *CARE guideline clinical timeline — symptom onset to post-operative discharge.*

Time Point	Event / Clinical Finding	Action Taken
Pre-Admission		
~2 weeks prior	Fall at home. Femoral neck fracture sustained. Patient rendered completely bedridden with severe pain.	Conservative management; fracture immobilised. Analgesia commenced.
Admission (Day 0)		
Day of presentation	Emergency department presentation. Severe pain; complete immobility. Weight 35 kg (BMI 15.2 kg/m²). NYHA Class III. On warfarin — INR supratherapeutic.	Emergency assessment. IV access. Orthopaedic and anaesthesia referrals made. Analgesics administered.
Pre-operative Workup — Day 1		

Time Point	Event / Clinical Finding	Action Taken
Day 1 — morning	ECG: sinus rhythm with LVH pattern. Echocardiography: EF 45%; severe concentric LVH; moderate AR; aortic root 5.4 cm (sinuses of Valsalva); elevated PASP.	Cardiology MDT consultation. Case classified ASA Physical Status IV. Cardiology written opinion obtained.
Day 1 — afternoon	CT aortogram: max. ascending aortic diameter 8.3 cm; arch 4.5 cm; descending 3.8 cm; diffuse intramural atheromatous calcification; branch vessel dilatation. CT thorax (lung windows): bilateral pleural effusions (R ~250–270 mL, L ~360–380 mL); left lower lobe collapse with fibro-atelectasis; mediastinal shift. L-S spine & pelvis X-ray: femoral neck fracture confirmed; severe lumbar osteophytes noted.	Pulmonology consultation. Vascular Surgery and CTVS consultations initiated. MDT imaging review completed.
Day 1 — evening	Bloods: Hb 9.2 g/dL; serum creatinine 1.8 mg/dL (eGFR 28 mL/min/1.73m²); INR supratherapeutic; platelets 1.98×10^5/μL; Na 136 mEq/L; K 4.1 mEq/L. Hypothyroidism — on replacement therapy; TSH reviewed.	Anticoagulation reversal: Vitamin K + FFP. Blood cross-match: 4 units PRBC. Thyroid replacement therapy reviewed and continued.
Pre-operative Preparation — Day 2		
Day 2	INR post-reversal: 1.1 (meets ASRA 2018 neuraxial criteria). Detailed informed video consent obtained — patient and family counselled regarding risk of intraoperative aortic rupture/dissection, cardiac arrest, renal failure, stroke, and perioperative death. Patient and family expressed clear wish to proceed.	ASRA neuraxial safety criteria confirmed met. Anaesthetic and surgical plan finalised. CPB equipment and CTVS team confirmed present in operative suite. Noradrenaline infusion prepared (4 mg/50 mL).
Intraoperative — Day 3		
Pre-block (baseline)	MAP 88 mmHg; HR 72 bpm; SpO2 99%. Patient positioned in left lateral decubitus. Invasive radial arterial line (right wrist) established. 5-lead ECG, pulse oximetry, capnography connected.	All monitoring confirmed functional prior to neuraxial intervention. Noradrenaline infusion prepared and primed.

Time Point	Event / Clinical Finding	Action Taken
Block placement	L3-L4: needle-through-needle CSE. Intrathecal: 1.5 mL 0.5% hyperbaric bupivacaine (7.5 mg) + fentanyl 10 mcg over 30 seconds. Epidural catheter advanced 4 cm from L2-L3 (tip ~L1-L2). T10 bilateral sensory level confirmed by ice test.	Epidural 0.25% ropivacaine infusion commenced at 4 mL/hr. Noradrenaline infusion commenced at 0.04 mcg/kg/min prophylactically.
0–30 min (block onset)	MAP fell to 74 mmHg at 10 min (sympatholysis onset); HR 68 bpm. SpO2 99%. No MAP <60 mmHg recorded.	Noradrenaline titrated upward; MAP restored to 78 mmHg by 30 min. Ropivacaine infusion maintained at 4 mL/hr.
30–90 min (maintenance)	MAP 78–81 mmHg; HR 64–66 bpm; SpO2 99–100%. Haemodynamically stable. Surgeon reports satisfactory operating conditions.	Noradrenaline 0.04–0.08 mcg/kg/min. 2 units PRBC transfusion commenced. Zero crystalloid/colloid administered.
90–150 min (completion)	MAP 79–83 mmHg; HR 64–65 bpm; SpO2 100%. Bipolar hemiarthroplasty completed. Total duration 2.5 hours. CPB standby not activated.	Noradrenaline 0.06–0.12 mcg/kg/min. Ropivacaine infusion titrated to 5 mL/hr. PRBC transfusion completed (2 units total). Patient transferred to CT-ICU.
Post-operative Course		
POD 0–1 (CT-ICU)	Haemodynamically stable on monitoring (Figure 5): HR 65 bpm; invasive BP 149/51 mmHg (MAP 83 mmHg); SpO2 100%. Epidural analgesia effective — NRS pain score ≤3/10 at rest. Creatinine at 48 hrs: 1.9 mg/dL (no clinically significant deterioration from baseline 1.8 mg/dL).	Noradrenaline weaned; discontinued at 18 hours post-op. Epidural 0.1% ropivacaine infusion maintained for post-operative analgesia.
POD 3	Haemodynamically stable without vasopressor support. Pain well-controlled (NRS ≤3). Epidural catheter removed.	Discharged from CT-ICU to high-dependency orthopaedic ward. Oral analgesia commenced.
POD 7–10	Mobilisation commenced with physiotherapy support. Creatinine returned to baseline. Patient communicating and reporting satisfaction with pain relief — expressed improvement over pre-admission functional state.	Discharge planning initiated. Outpatient cardiac, renal, and orthopaedic follow-up arranged.

Time Point	Event / Clinical Finding	Action Taken
POD 14	Patient discharged home. Ambulatory with walking aid. Family confirmed improvement in function and quality of life compared to bedridden pre-operative state.	Discharge summary issued. Cardiology, nephrology, and orthopaedic outpatient follow-up booked. GP letter sent.

2.2 Pre-operative Assessment and Investigations

Cardiovascular evaluation (echocardiography and CT aortogram) identified: aortic root diameter 5.4 cm at the sinuses of Valsalva on echocardiography; maximum tubular ascending aortic diameter 8.3 cm on axial CT imaging; aortic arch 4.5 cm; descending thoracic aorta 3.8 cm — with diffuse intramural atheromatous calcification and significant dilatation of arch branch vessels at their origins (Figure 4, mediastinal windows). These two aortic measurements reflect anatomically distinct levels: the 5.4 cm is the echocardiographic root dimension at the sinuses of Valsalva, while 8.3 cm is the maximum diameter of the tubular ascending aorta on CT — both are clinically relevant and are reported separately to avoid confusion [1,2].

Echocardiography confirmed ejection fraction 45%, severe concentric left ventricular hypertrophy (LVH), moderate aortic regurgitation (AR), and elevated estimated pulmonary artery systolic pressure. CT thorax in lung windows (Figures 3 and 4) demonstrated bilateral moderate pleural effusions with passive subsegmental atelectasis, collapse with fibro-atelectasis of the left lower lobe, mediastinal shift to the left, and bilateral sub-pleural fibrotic strands. Plain radiograph of the lumbo-sacral spine and pelvis **(Figure 1)** confirmed the femoral neck fracture and demonstrated severe exaggerated dorsal kyphosis with anterior marginal and bridging osteophytes — findings directly relevant to neuraxial access planning.

All relevant pre-operative laboratory values and clinical parameters are summarized in **Table 2.**

Table 2. *Pre-operative investigations and clinical parameters with reference ranges and clinical significance.*

Parameter	Value	Reference Range	Clinical Significance
Patient Demographics			
Age / Sex	86 years / Female	—	Advanced age; elevated perioperative baseline risk
Weight / Height	35 kg / 148 cm	—	Markedly underweight; reduced drug dosing required
BMI	15.2 kg/m²	18.5–24.9 kg/m²	Severe malnutrition; impaired wound healing and immunity
NYHA Functional Class	Class III	Class I	Symptomatic on mild exertion; limited functional reserve
ASA Physical Status	IV	—	Life-threatening systemic disease
Cardiovascular Assessment			
Aortic Root Diameter (Echo)	5.4 cm	<4.0 cm	Dilated at sinuses of Valsalva level
Max. Ascending Aorta (CT)	8.3 cm	<4.0 cm	Giant aneurysm — surgical threshold 5.5 cm; high rupture risk

Parameter	Value	Reference Range	Clinical Significance
Aortic Arch Diameter (CT)	4.5 cm	<3.0 cm	Dilated; branch vessel involvement noted
Descending Thoracic Aorta (CT)	3.8 cm	<3.0 cm	Diffuse panoptic aortic disease
Ejection Fraction (Echo)	45%	≥55%	Mildly reduced LV systolic function; impaired cardiac output
LV Wall Morphology	Severe concentric LVH	Normal	Reduced compliance; increased myocardial oxygen demand
Aortic Valve	Moderate AR	Normal — competent	Volume overload on LV; afterload reduction beneficial
ECG	Sinus rhythm; LVH pattern	Normal	No acute ischaemic changes identified
Respiratory Assessment			
Pleural Effusion — Right	~250–270 mL	None	Passive subsegmental atelectasis of adjacent lung
Pleural Effusion — Left	~360–380 mL	None	Left lower lobe collapse; fibro-atelectasis; mediastinal shift
Lung Parenchyma (CT)	Bilateral sub-pleural fibrotic strands	Normal	Reduced lung compliance; positive-pressure ventilation to be avoided
Gross Cardiomegaly	Present (LV configuration)	Normal	Further evidence of chronic cardiac volume overload
Haematology and Biochemistry			
Haemoglobin	9.2 g/dL	12.0–16.0 g/dL	Moderate anaemia; impaired O_2 delivery; PRBC transfusion indicated
Platelet Count	1.98 × 10^5/µL	1.5–4.0 × 10^5/µL	Within normal range; adequate for neuraxial procedure
INR (pre-reversal)	Supratherapeutic	<1.5 (neuraxial)	Contraindication to neuraxial until reversed

Parameter	Value	Reference Range	Clinical Significance
INR (post-reversal)	1.1	<1.5 (neuraxial)	Safe for neuraxial per ASRA 2018 guidelines [9]
Serum Creatinine	1.8 mg/dL	0.5–1.1 mg/dL	AKI — avoid nephrotoxins; zero-crystalloid strategy adopted
eGFR	28 mL/min/1.73m²	≥60 mL/min/1.73m²	CKD Stage 4; fragile renal reserve; liberal fluids contraindicated
Sodium / Potassium	136 / 4.1 mEq/L	135–145 / 3.5–5.0	Electrolytes within normal range
Endocrine / Other			
Thyroid Function	Hypothyroid	Euthyroid	On levothyroxine replacement; TSH reviewed perioperatively
Anticoagulation	Warfarin (chronic)	—	Reversed with Vitamin K + FFP prior to neuraxial procedure
L-S Spine Imaging	Severe osteophytes; exaggerated kyphosis; bridging osteophytes; end-plate sclerosis	Normal	Directly impacts neuraxial technique: L3-L4 selected via careful palpation and landmark triangulation

2.3 Multidisciplinary Team Decision and Consent

A formal MDT meeting was convened with Cardiology, Pulmonology, Vascular Surgery, and Cardiothoracic and Vascular Surgery (CTVS). The collective clinical judgement was that definitive aortic aneurysm repair prior to orthopedic intervention was not feasible given the patient's decompensated physiological state, nutritional failure, and the ongoing fracture-related deterioration. The consensus recommendation was to proceed with bipolar hemiarthroplasty under neuraxial anaesthesia, subject to four mandatory conditions: (i) CPB equipment and CTVS team physically present within the operative suite complex throughout the procedure; (ii) invasive beat-to-beat arterial blood pressure monitoring established prior to neuraxial intervention; (iii) avoidance of GA, PPV, and laryngoscopy under all circumstances; and (iv) pre-defined haemodynamic targets with vasopressor-driven management from block onset.

The CPB standby requirement was not precautionary excess but represented the minimum ethical standard for a procedure in which acute aortic decompensation carried a non-trivial probability and in which no intervention short of emergent CPB-supported surgical repair would offer any realistic survival chance [2]. The team acknowledged the extremely high mortality of emergency CPB activation in this context, but accepted that its omission would have been clinically and ethically indefensible.

2.4 Anaesthetic Management

Warfarin anticoagulation was reversed with Vitamin K and fresh frozen plasma. INR was confirmed at 1.1 on the morning of surgery, satisfying the threshold for neuraxial instrumentation per ASRA 2018 guidelines [9]. Standard monitoring was established and confirmed functional prior to any neuraxial intervention: continuous 5-lead ECG, pulse oximetry, capnography, and an invasive radial arterial line (right wrist) for beat-to-beat blood pressure monitoring.

With the patient positioned in the left lateral decubitus position, the L3-L4 interspace was identified by careful surface landmark palpation and triangulation, accounting for the severe lumbar osteophytic changes documented on pre-operative imaging. Loss-of-resistance to saline confirmed epidural space identification at L3-L4 via an 18-gauge Tuohy needle. Using the needle-through-needle CSE technique, a 27-gauge Whitacre pencil-point spinal needle was introduced through the Tuohy needle;

intrathecal injection comprised 1.5 mL of 0.5% hyperbaric bupivacaine (7.5 mg) with fentanyl 10 mcg (0.2 mL), administered slowly over 30 seconds to attenuate cardiovascular impact. An epidural catheter was subsequently advanced 4 cm cephalad from the L2-L3 interspace — one level above the spinal entry — positioning the catheter tip at approximately L1-L2. A T10 sensory level was confirmed bilaterally by ice test prior to surgical draping.

The epidural catheter was advanced from L2-L3 and provided coverage to T10 dermatomes through cephalad migration of the local anaesthetic solution — a well-described property of lumbar epidural blockade at adequate volumes [6,7]. An epidural infusion of 0.25% ropivacaine was commenced immediately at 4 mL/hr and titrated to 4–6 mL/hr to maintain the T10 sensory level throughout the procedure. Ropivacaine was selected over bupivacaine for its superior cardiac safety profile at equivalent analgesic concentrations and its lesser degree of motor blockade [10]. A T10 level is the minimum requirement for hip arthroplasty, providing anaesthesia of the hip capsule (L1-L2), femoral shaft (L2-L3), and sacral nerve roots (S1-S3) via hyperbaric bupivacaine pooling in the dependent sacral canal in the lateral position — confirmed in this patient and maintained throughout by epidural supplementation [7].

Noradrenaline was commenced prophylactically at 0.04 mcg/kg/min at the time of spinal injection and titrated to 0.04–0.12 mcg/kg/min throughout the procedure to maintain MAP ≥65 mmHg. No crystalloid or colloid fluids were administered. Two units of packed red blood cells (PRBCs, approximately 500 mL total) were transfused over the course of the procedure to maintain haemoglobin ≥9 g/dL and optimise oxygen delivery in the context of impaired cardiac output and surgical blood loss [17]. The patient maintained spontaneous ventilation throughout, receiving supplemental oxygen at 4 L/min via nasal cannula (SpO2 maintained ≥98%). Intraoperative syringe pump configuration is shown in **Figure 2.**

2.5 Intraoperative Haemodynamic Trend and Post-operative Course

Intraoperative haemodynamic data are documented in Table 1. In summary: MAP ranged 74–88 mmHg throughout the procedure (no episode of MAP <60 mmHg or systolic blood pressure >150 mmHg was recorded); HR was maintained 64–72 bpm. The procedure was completed in 2.5 hours without haemodynamic crisis, cardiac arrhythmia, or the need to activate CPB standby.

Post-operative CT-ICU monitoring (Figure 5) demonstrated: HR 65 bpm; invasive arterial BP 149/51 mmHg (MAP 83 mmHg); SpO2 100% — all within pre-defined target ranges. Noradrenaline was weaned and discontinued at 18 hours post-operatively. Serum creatinine at 48 hours post-operatively was 1.9 mg/dL — representing no clinically significant deterioration from the pre-operative baseline of 1.8 mg/dL — confirming the renal safety of the zero-crystalloid fluid strategy. Epidural analgesia (0.1% ropivacaine infusion) provided effective post-operative pain control (NRS ≤3/10 at rest) for 48 hours. The patient was discharged from CT-ICU on post-operative day 3, mobilised with physiotherapy from day 7, and discharged home on day 14 with outpatient Cardiology, Nephrology, and Orthopedics follow-up.

3. DISCUSSION

This case report describes the perioperative management of an 86-year-old, 35 kg, ASA-IV patient with an 8.3 cm ascending aortic aneurysm presenting for bipolar hemiarthroplasty following refusal at multiple tertiary centres. We present this case in full accordance with CARE reporting guidelines [8] and acknowledge explicitly the limitations inherent to single-patient case reports: no causal inferences can be drawn, and individual application of these principles requires independent MDT assessment, institutional capability, and clinical judgement. Prospective registry data are needed to validate this approach.

3.1 Rationale for CSE over General Anaesthesia

The physiological case for neuraxial anaesthesia over GA in aortic aneurysmal disease is well-established [4,5,11,12]. Direct laryngoscopy produces a 20–40% increase in systolic blood pressure and 15–25 bpm increase in heart rate in unpremeditated patients, lasting up to 90 seconds [4]. In an aorta dilated to 8.3 cm with atheromatous, presumably fragile walls, this transient but extreme elevation in transmural wall stress — which, per the law of Laplace, increases proportionally with intraluminal pressure × radius — represents a quantifiable and potentially fatal haemodynamic event. Neuraxial anaesthesia eliminates this stimulus entirely by interrupting afferent and efferent sympathetic signaling, and avoids the negative inotropic effects of volatile agents on an already-compromised ventricle [5].

The existing published literature on neuraxial anaesthesia for non-cardiac surgery in high-risk vascular patients, while limited, is consistent in supporting this approach. Chaikof et al. [11] reported superior haemodynamic stability with epidural compared to GA for endovascular aortic procedures. A systematic analysis by Neary et al. [12] of anaesthetic technique in high-risk vascular patients similarly identified neuraxial approaches as associated with fewer cardiovascular adverse events. Our case extends this experience to the extreme end of the risk spectrum — a patient with a giant aneurysm, multisystem failure, and BMI of 15.2 kg/m².

3.2 Technical Justification of the CSE Technique and T10 Sensory Level

The needle-through-needle CSE technique at L3-L4

(spinal injection) and L2-L3 (epidural catheter advancing ~4 cm to tip at ~L1-L2) is well-described in the regional anaesthesia literature [6]. Placing the epidural catheter one interspace above the spinal needle entry allows independent catheter positioning and avoids the theoretical risk of subarachnoid catheter migration through the dural puncture. The final catheter position at approximately L1-L2 provides adequate dermatomal coverage when supplemented with epidural local anaesthetic [6,7].

A T10 sensory level is the minimum dermatome requirement for hip arthroplasty, encompassing the parietal peritoneum over the hip capsule (L1), proximal femur and shaft (L2-L3), and the sciatic nerve contributions to the posterior thigh and acetabulum (S1-S3) — the sacral components covered by the dependent pooling of hyperbaric bupivacaine in the lateral position [7]. The epidural 0.25% ropivacaine infusion (higher concentration than the 0.1–0.2% maintenance doses commonly used in obstetric and post-operative settings) was deliberately selected to ensure dense, reliable block maintenance: in this patient, breakthrough pain causing sympathoadrenal activation was as physiologically dangerous as haemodynamic depression from excessive blockade [10].

3.3 Vasopressor Selection, Dosing, and Pharmacological Rationale

Noradrenaline was selected as the vasopressor of choice over ephedrine, phenylephrine, or dopamine, based on its pharmacological profile in the context of this patient's specific pathology. Its predominant α1-adrenoceptor-mediated vasoconstriction restores SVR and MAP without the clinically significant chronotropy of ephedrine or dopamine — tachycardia being specifically deleterious in moderate AR, where elevated heart rate shortens diastolic filling time and increases regurgitant volume per unit time, and in a hypertrophied ventricle in which impaired coronary perfusion reserve makes tachycardia poorly tolerated [13,18]. At the doses employed (0.04–0.12 mcg/kg/min), noradrenaline's modest β1 effect provided useful positive inotropy without observed tachycardia, consistent with published vasopressor dosing guidelines for neuraxial-induced hypotension in high-risk surgical patients [13,14].

3.4 Zero-Crystalloid, PRBC-Only Fluid Strategy: Rationale and Evidence

The decision to administer no crystalloid or colloid fluids was driven by the convergence of three distinct pathophysiological imperatives in this patient. First, in decompensated congestive cardiac failure (EF 45%) with bilateral pleural effusions, crystalloid loading of even 250 mL risks elevating pulmonary capillary hydrostatic pressure beyond the oncotic threshold — acutely exacerbating pulmonary oedema in lungs already compromised by effusions and atelectasis [15]. Second, in AKI (eGFR 28 mL/min/1.73m²), liberal fluid administration paradoxically worsens renal perfusion via the 'venous congestion' mechanism: elevated central venous pressure impairs glomerular filtration by reducing the pressure gradient driving renal perfusion, and increases renal interstitial oedema — a pathophysiology now well-supported in the critical care nephrology literature [16]. Third, in severe protein-calorie malnutrition (BMI 15.2 kg/m², likely hypoalbuminaemia), reduced plasma oncotic pressure markedly exaggerates the third-spacing of administered crystalloid.

Transfusion of PRBCs served the dual purpose of volume augmentation and haemoglobin optimisation. Maintaining Hb ≥9 g/dL in a patient with impaired cardiac output, a volume-overloaded regurgitant ventricle, and increased perioperative oxygen demand is consistent with AABB 2016 recommendations for patients with active cardiac disease [17]. The observed post-operative creatinine of 1.9 mg/dL — essentially unchanged from the pre-operative baseline of 1.8 mg/dL — provides objective clinical confirmation that the zero-crystalloid strategy did not cause clinically meaningful exacerbation of AKI.

3.5 CPB Standby: Ethical Justification and Institutional Requirements

The requirement for CPB standby with a physically present CTVS team warrants explicit ethical and clinical justification, as it represents a significant logistical and resource commitment. An ascending aorta of 8.3 cm maximum diameter — substantially above the 5.5 cm elective repair threshold — carries an estimated annual rupture or dissection risk of 6–10% under resting conditions; this risk is plausibly higher under the physiological demands of anaesthesia, surgical stress, and positioning [1,2]. In the event of acute aortic decompensation during the procedure, no pharmacological or basic resuscitative intervention could offer meaningful survival benefit; only immediate CPB-supported surgical repair could do so. CPB standby was therefore not precautionary excess but an irreducible minimum of safe care for this procedure.

The team acknowledged transparently that emergency CPB activation in a 35 kg, 86-year-old patient with a 8.3 cm aneurysm, EF 45%, and AKI carries extremely high mortality — but accepted that the alternative of proceeding without this safety mechanism would have been ethically indefensible given the known risk profile, and potentially incompatible with the duty of care owed to the patient and family who had consented on the basis of this protection being available.

4. CONCLUSION

This case report describes the successful perioperative management of an 86-year-old, 35 kg, ASA-IV patient

with an 8.3 cm ascending aortic aneurysm presenting for urgent bipolar hemiarthroplasty, prepared in accordance with CARE guidelines. A titratable CSE technique — comprising a low-dose subarachnoid block supplemented by epidural 0.25% ropivacaine infusion (4–6 mL/hr) — combined with invasive beat-to-beat haemodynamic monitoring, titrated noradrenaline infusion (0.04–0.12 mcg/kg/min), a zero-crystalloid PRBC-only fluid strategy (2 units transfused), and CPB standby, enabled maintained haemodynamic stability (MAP 74–88 mmHg; HR 64–72 bpm) throughout a 2.5-hour procedure. Post-operative creatinine remained stable, confirming renal safety. The patient was discharged from CT-ICU on day 3, mobilised from day 7, and discharged home on day 14.

We propose that a structured neuraxial approach — supported by early MDT engagement, pre-defined haemodynamic targets, vasopressor-driven management, and institutional CPB capability — may constitute a viable and physiologically sound framework for similar presentations. These findings should not be extrapolated beyond their single-case limitations; prospective registry data and multi-centre case series are required to validate and refine the principles described.

Declarations

Ethics Approval and Consent to Participate: Written and video informed consent was obtained from the patient and her family for publication of this case report, all clinical data, and all accompanying imaging prior to submission. The consent process was conducted over two sessions and video-documented. No institutional ethics committee approval was required under local policy for single case report publication; however, all patient identifiers visible on imaging are those of the consented patient.

Patient and Family Perspective: The patient and her family expressed a clear and consistent wish for surgical intervention to relieve severe pain and restore functional independence. They understood the significant perioperative risks and consented to proceed. Post-operatively, the patient reported satisfactory pain control and the family expressed satisfaction with the outcome and the restoration of the patient's ability to communicate and mobilise.

Availability of Data and Materials: All clinical data and imaging are contained within this manuscript. Additional data are available from the corresponding author upon reasonable request.

Competing Interests: None declared.

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Authors' Contributions: [Initials]: Conceived and designed the anaesthetic strategy, performed the procedure, drafted and revised the manuscript. All authors reviewed and approved the final version for submission.

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Figures

All figures are reproduced with written and video informed patient consent. Visible patient identifiers on imaging are those of the consented patient.

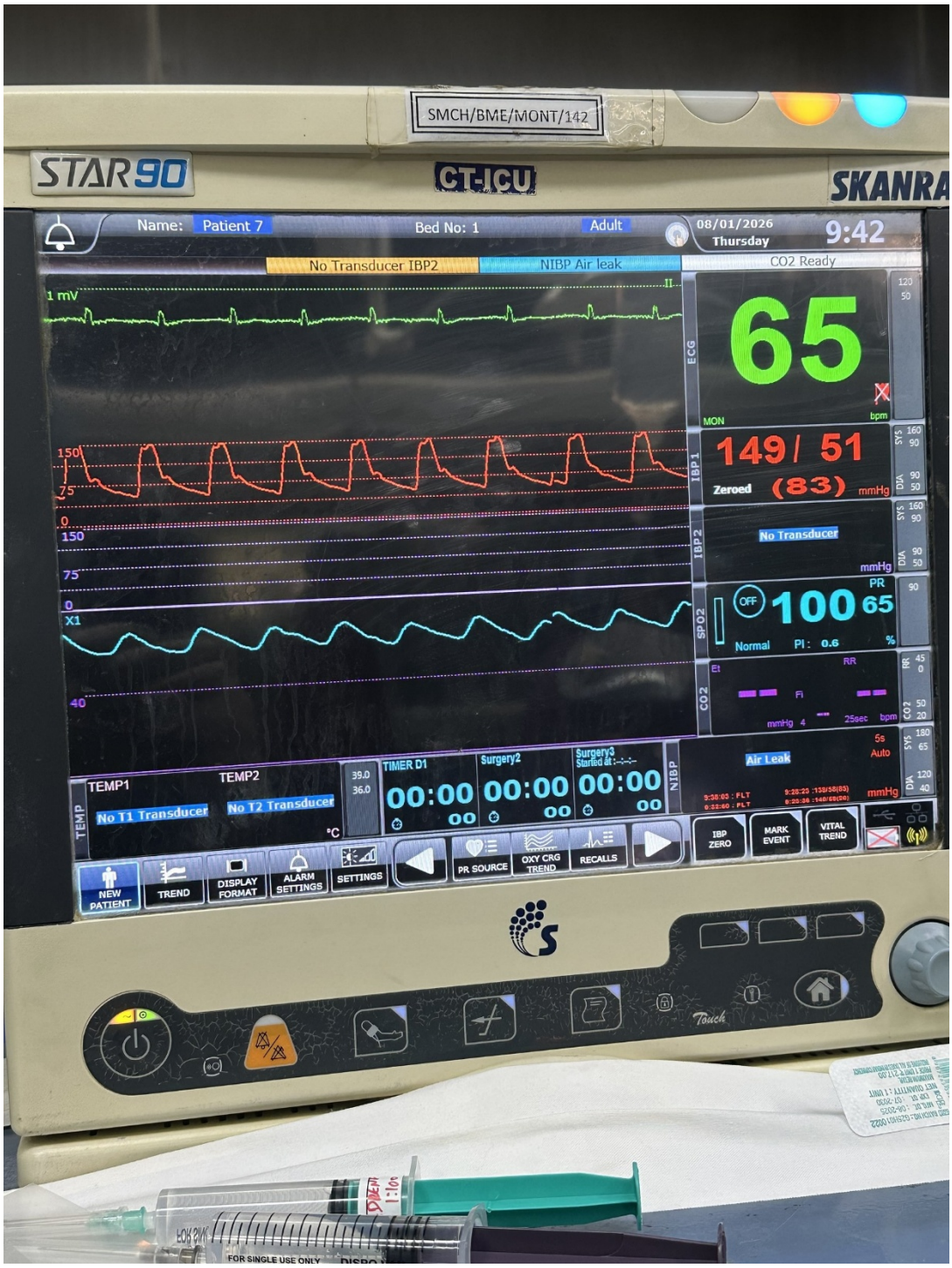

Figure 1. *Intra operative vitals monitoring*

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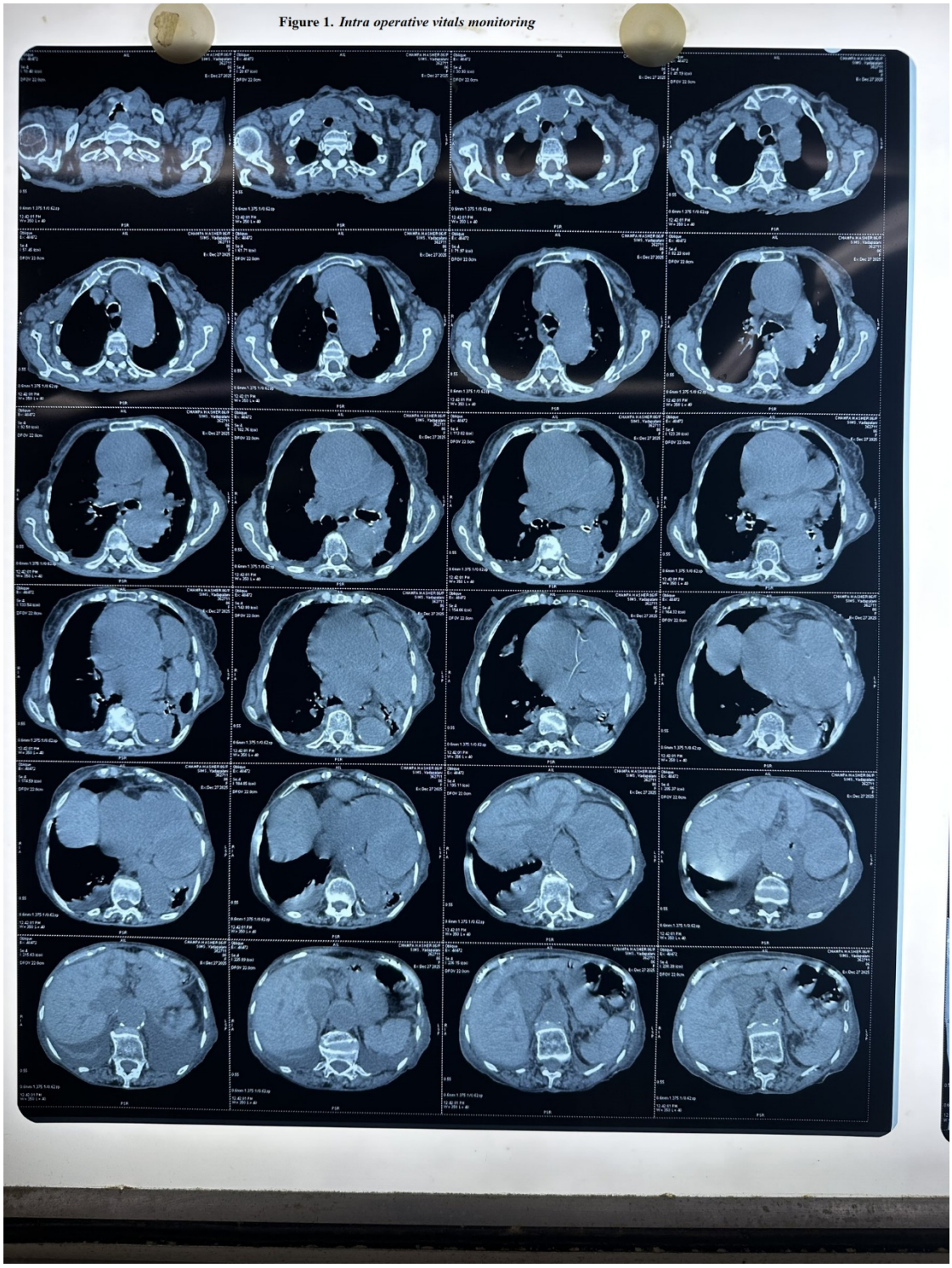

Figure 2. CT thorax — axial sections in lung window settings . Sequential cross-sections from upper thorax to lung bases demonstrating: bilateral moderate pleural effusions (right ~250–270 mL, left ~360–380 mL); passive subsegmental atelectasis of adjacent lung segments; collapse with fibro-atelectasis of the left lower lobe with mediastinal shift to the left; and bilateral sub-pleural fibrotic strands. These findings supported the decision to avoid positive-pressure ventilation throughout the procedure.

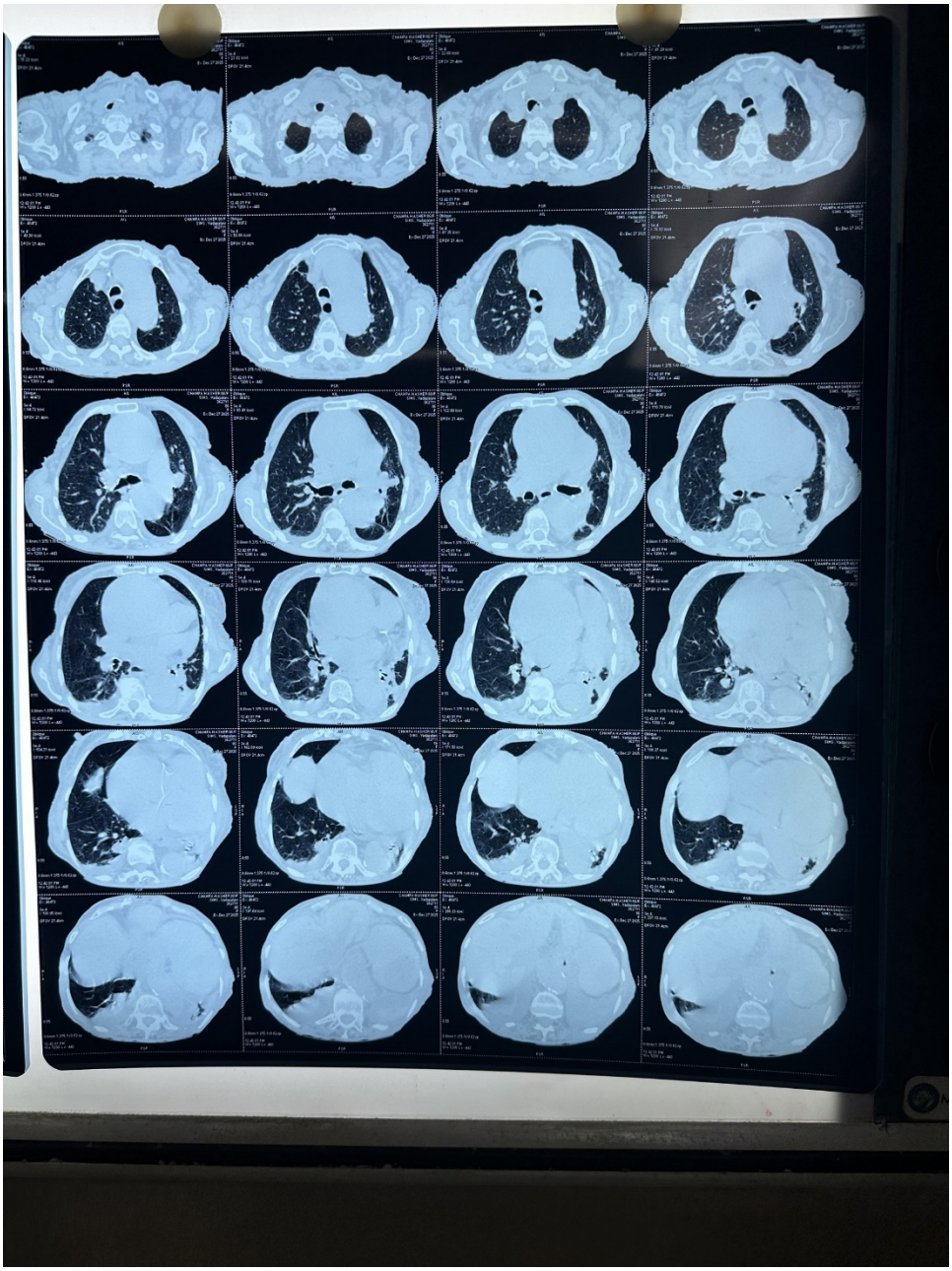

Figure 3. *CT thorax/abdomen — axial sections in mediastinal window settings (27-Dec-2025). Upper rows confirm gross dilatation of the ascending aorta (maximum tubular ascending diameter 8.3 cm), aortic arch (4.5 cm), and descending thoracic aorta (3.8 cm), with diffuse intramural atheromatous calcification and branch vessel dilatation at arch origins. Note: the aortic root at the sinuses of Valsalva measured 5.4 cm on echocardiography — a distinct and more cephalad anatomical level from the 8.3 cm CT measurement. Gross cardiomegaly with LV configuration and bilateral pleural effusions are also demonstrated.*

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