

Perioperative Anesthetic Management During Endovascular Repair of Stanford Type B Aortic Dissection: A Case Report And Narrative Literature Review Case Report and Review

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ABSTRACT

Introduction: Thoracic endovascular aortic repair (TEVAR) is the treatment of choice for complicated Stanford type B aortic dissection; however, procedural success depends on appropriate perioperative anesthetic management. We present a clinical case and a narrative review of the literature, emphasizing an anesthetic strategy focused on maintaining four key physiological objectives throughout the perioperative period.

Case Presentation: A 49-year-old man with a history of systemic arterial hypertension presented with an extensive Stanford type B aortic dissection associated with visceral malperfusion and underwent TEVAR. The procedure was performed under balanced general anesthesia with continuous hemodynamic and metabolic monitoring, intraoperative transthoracic echocardiography, and strict maintenance of mean arterial pressure between 70 and 80 mmHg using a norepinephrine infusion. During the intervention, an iatrogenic left femoral artery injury occurred, requiring immediate surgical repair and blood product transfusion. Despite this complication, hemodynamic stability, adequate tissue perfusion, and the absence of metabolic acidosis or hyperlactatemia were maintained throughout the procedure. The patient was successfully extubated in the operating room without neurological deficits and experienced an uneventful postoperative recovery, with no cardiovascular, neurological, or renal complications.

Discussion: This case highlights the importance of a structured anesthetic strategy based on physiological goals to minimize aortic wall stress, optimize perfusion of vital organs, and facilitate the early detection and management of intraoperative complications during TEVAR. The literature supports multimodal monitoring and individualized hemodynamic management as key components for improving perioperative outcomes in these patients.

Conclusion: Goal-directed anesthetic management represents an essential component of the endovascular treatment of Stanford type B aortic dissection. The proposed strategy provided sustained hemodynamic stability and a favorable clinical outcome despite an intraoperative vascular complication, suggesting that this reproducible approach may enhance perioperative safety in patients undergoing TEVAR.

KEYWORDS: Aortic Dissection; Endovascular Procedures; Anesthesia, General; Perioperative Care; Transesophageal Echocardiography.

ARTICLE DETAILS

Published On:
29 September 2026

Available on:
<https://ijmscrs.com>

INTRODUCTION

Acute aortic dissection (AAD) is one of the cardiovascular emergencies associated with the highest mortality, requiring

prompt diagnosis and immediate treatment. It is defined as the separation of the layers of the aortic wall secondary to an intimal tear that allows blood to enter the medial layer,

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creating a true lumen and a false lumen that compromise the structural integrity of the vessel. A dissection is considered acute when it occurs within the first two weeks after symptom onset (1). Although it is an uncommon condition, its estimated incidence ranges from 5 to 30 cases per million population per year, with an increasing trend worldwide (2). It predominantly affects men in the fifth to seventh decades of life, and approximately three out of every 1,000 patients presenting to the emergency department with chest, back, or abdominal pain are diagnosed with this disease (3). Despite advances in diagnostic and therapeutic strategies, mortality remains high, and a substantial proportion of patients die before reaching the hospital (4).

The Stanford classification remains the standard system for guiding the management of acute aortic dissection. Type A dissection involves the ascending aorta and constitutes a surgical emergency because of the high risk of life-threatening complications, including cardiac tamponade, hemopericardium, and acute aortic regurgitation (5). In contrast, Type B dissection originates distal to the origin of the left subclavian artery and is confined to the descending aorta, accounting for approximately 35–36% of all cases (6). In these patients, initial management consists of strict blood pressure and heart rate control, whereas endovascular or surgical repair is reserved for complicated cases, such as those with persistent pain, refractory hypertension, impending rupture, or malperfusion syndrome (7).

The management of Stanford Type B aortic dissection has evolved considerably over the past two decades. While optimal medical therapy remains the initial treatment strategy for uncomplicated cases, thoracic endovascular aortic repair (TEVAR) has become the treatment of choice for patients with complicated dissections by excluding the primary intimal tear, restoring true lumen perfusion, and promoting favorable aortic remodeling (8). Compared with open surgical repair, TEVAR is associated with lower perioperative morbidity and mortality, as well as faster recovery. Nevertheless, it remains a technically demanding procedure with the potential for serious complications,

including spinal cord ischemia, stroke, acute kidney injury, endoleaks, and retrograde Type A dissection, underscoring the need for highly specialized perioperative management (9). Anesthetic management is a critical component of successful TEVAR, as the hemodynamic fluctuations that occur during anesthetic induction, endograft deployment, and endovascular manipulation may increase aortic wall stress, thereby promoting dissection progression or even aortic rupture (10). Accordingly, the primary anesthetic objective is to maintain effective anti-impulse therapy through adequate anesthetic depth, sufficient analgesia, and stable cardiovascular conditions, while avoiding abrupt changes in blood pressure and heart rate throughout all phases of the procedure (11).

In addition to hemodynamic control, the anesthesiologist must ensure adequate cerebral, spinal cord, and visceral perfusion by maintaining an individualized mean arterial pressure, supported by invasive and multimodal monitoring. The use of intraoperative echocardiography, continuous arterial pressure monitoring, and serial assessment of metabolic parameters allows real-time evaluation of cardiovascular function, early detection of endograft-related complications, and timely recognition of tissue hypoperfusion, thereby facilitating prompt intervention when necessary (11).

Based on the available evidence and the physiological principles underlying the perioperative management of these patients, we propose an anesthetic strategy structured around four fundamental objectives (Figure 1): (1) prevent abrupt increases in aortic wall stress during anesthetic induction and endovascular manipulation; (2) maintain an individualized mean arterial pressure between 70 and 80 mmHg to ensure adequate cerebral, spinal cord, and renal perfusion; (3) preserve tissue perfusion through continuous hemodynamic and metabolic monitoring; and (4) facilitate early neurological recovery at the end of the procedure. These principles formed the foundation of the anesthetic management implemented in the present case and constitute the central framework of this narrative review.

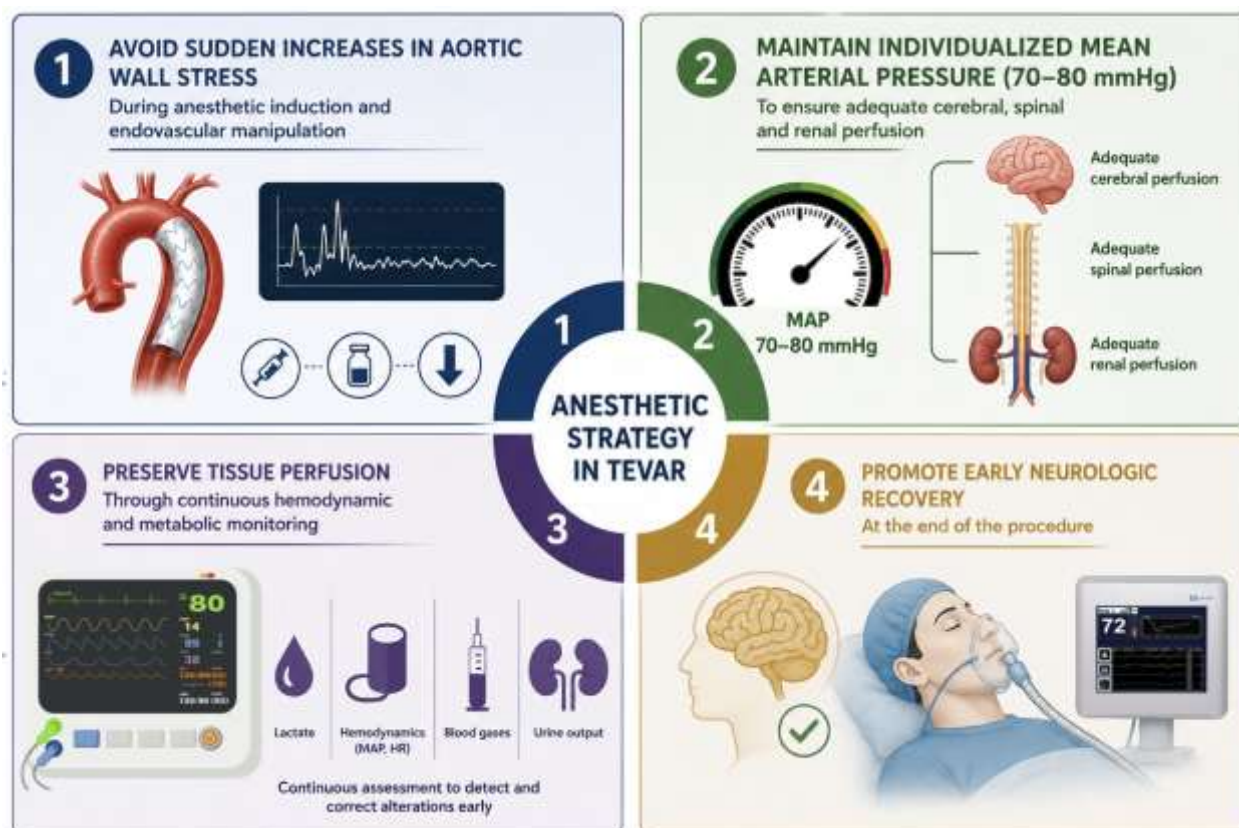


Figure 1. Proposed perioperative anesthetic strategy for thoracic endovascular aortic repair (TEVAR) in Stanford Type B aortic dissection. The figure illustrates the four fundamental objectives of anesthetic management: control of aortic wall stress, maintenance of an individualized mean arterial pressure, preservation of tissue perfusion, and promotion of early neurological recovery.

CASE REPORT

We present the case of a 49-year-old mestizo man, employed as a merchant, with a seven-year history of hypertension treated with valsartan 80 mg twice daily, metoprolol 50 mg once daily, and aspirin 100 mg daily. His cardiovascular history was notable for two previous episodes of unstable angina occurring seven and two years before presentation. He had no history of prior surgery, hereditary disorders, or a family history of aortic disease. He presented to the emergency department with sudden-onset epigastric pain radiating to the anterior chest and interscapular region. The pain was described as oppressive, with a severity of 9/10 on the visual analog scale, persisted despite rest, and was associated with severe hypertension (blood pressure, 200/110 mmHg). The initial electrocardiogram demonstrated ischemic changes in the anterior leads, leading to a presumptive diagnosis of high-risk acute coronary syndrome. Medical management was initiated with dual antiplatelet therapy, anticoagulation, statins, beta-blockade, and analgesia. Due to persistent chest pain despite initial treatment and the need for evaluation by interventional cardiology to determine a pharmacoinvasive strategy or

perform diagnostic cardiac catheterization, the patient was referred to a tertiary care hospital.

A patient was evaluated at a tertiary care hospital and was neurologically intact and hemodynamically stable, although he had persistent severe arterial hypertension (220/110 mmHg). Physical examination revealed no chest pain, with regular heart sounds of normal tone and intensity, without murmurs or other added sounds. Due to poor blood pressure control despite intravenous antihypertensive therapy, hospital admission was decided for strict blood pressure monitoring and evaluation for secondary hypertension. A broad spectrum of potentially life-threatening differential diagnoses was considered, including acute coronary syndrome, acute aortic dissection, pulmonary thromboembolism, hypertensive emergencies with target-organ damage, acute pericarditis, cardiac tamponade, and spontaneous pneumothorax.

As part of the diagnostic workup, contrast-enhanced computed tomography angiography of the chest was performed, revealing a Stanford Type B (DeBakey IIIb) aortic dissection, with an intimal flap originating immediately distal to the origin of the left subclavian artery and extending throughout the descending thoracic aorta (Figure 2).



Figure 2. Sagittal computed tomography angiography image demonstrating a Stanford Type B (DeBakey IIIb) aortic dissection, with an intimal flap originating immediately distal to the origin of the left subclavian artery and extending throughout the descending thoracic aorta.

Subsequent computed tomography angiography of the chest, abdomen, and pelvis confirmed progression of the dissection into both common iliac arteries, with patency of both the true and false lumens, involvement of the celiac trunk, and imaging findings suggestive of splenic infarctions, without evidence of false lumen thrombosis. The patient was admitted to the intensive care unit for anti-impulse therapy, allowing gradual discontinuation of the nitroglycerin infusion and transition to oral antihypertensive agents with adequate blood pressure control. Following multidisciplinary evaluation by the vascular surgery team, thoracic endovascular aortic repair (TEVAR) was indicated because of the extensive nature of the dissection and evidence of visceral malperfusion.

During hospitalization, the patient developed a febrile episode that prompted evaluation by the Infectious Diseases service. Following clinical and microbiological assessment, bacteremia was ruled out; however, given the high surgical risk and based on the available antibiogram, levofloxacin therapy was initiated prior to the endovascular procedure, with adequate clinical response and maintenance of an afebrile state. Preoperative evaluation revealed a hemoglobin level of 12.4 g/dL, preserved platelet function, electrolytes within acceptable ranges, and a serum creatinine level of 1.93 mg/dL. The electrocardiogram showed sinus rhythm without changes suggestive of myocardial ischemia. The patient was

classified as ASA IV, with a high thromboembolic risk (Caprini score: 11 points), intermediate risk of pulmonary complications (ARISCAT score: 47 points), EuroSCORE II of 2.9%, and NYHA functional class II. Preoperative transthoracic echocardiography demonstrated preserved left ventricular ejection fraction (61%), anterior wall hypokinesia, and grade I diastolic dysfunction, along with biatrial dilation and preserved right ventricular function (TAPSE: 16.5 mm).

Surgical intervention

Thoracic endovascular aortic repair (TEVAR) was performed under balanced general anesthesia. Anesthetic induction was achieved using a titrated intravenous technique with midazolam (2 mg), fentanyl (250 µg), propofol (200 mg), and rocuronium (50 mg). Orotracheal intubation was performed using video laryngoscopy (Macintosh #3 blade), achieving a glottic view with a POGO score of 50%; a 7.5-mm endotracheal tube was successfully placed without complications. Anesthesia was maintained with balanced general anesthesia and mechanical ventilation in volume-controlled mode (tidal volume: 475 mL, PEEP: 6 cmH₂O, respiratory rate: 30 breaths/min).

Intraoperative monitoring included processed four-channel electroencephalography for assessment of anesthetic depth and transthoracic echocardiography, which confirmed a preserved left ventricular ejection fraction of 61%, an intimal

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flap immediately distal to the origin of the left subclavian artery with high-velocity flow within the false lumen without evidence of thrombosis, and extension of the dissection into the abdominal aorta. During the procedure, 7,000 IU of unfractionated heparin were administered, maintaining an activated clotting time between 250 and 300 seconds.

Hemodynamic stability was achieved through continuous norepinephrine infusion ($0.02\text{--}0.08\text{ }\mu\text{g/kg/min}$), titrated to maintain a mean arterial pressure of 70–80 mmHg throughout the different stages of the procedure. Acid–base balance remained within physiological parameters, with no evidence of metabolic acidosis, stable lactate concentrations ranging from 0.7 to 1.0 mmol/L, and an estimated urine output of

approximately 1.5 mL/kg/h, without signs of renal hypoperfusion.

During the intervention, an injury to the left femoral artery occurred secondary to vascular access, requiring immediate repair by the cardiovascular surgery team. Estimated blood loss was 1,300 mL; therefore, two units of packed red blood cells and fresh frozen plasma were transfused, with hemodynamic stability maintained throughout the event. The surgical time was 225 minutes, and the anesthetic time was 270 minutes. Thoracic endograft implantation was successfully completed (Figures 3 and 4), without device-related complications or major anesthetic adverse events.



Figura 3. Imagen transtoperatoria de hemodinamia previa a la colocación de endoprótesis



Figura 4.. Imagen transtoperatoria de hemodinamia posterior a colocación de endoprótesis

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At the end of the procedure, the patient met extubation criteria in the operating room, recovering an alert mental status without evident neurological deficits. He was subsequently transferred to the intensive care unit for postoperative monitoring. The patient had a favorable clinical course, with no additional neurological, cardiovascular, or renal complications, and was discharged for outpatient follow-up by the cardiology service.

The patient expressed satisfaction with the care received and stated that he understood the severity of his condition, as well as the need for the proposed endovascular treatment. Following the intervention, he reported relief due to the resolution of symptoms and favorable clinical course, emphasizing the information provided by the multidisciplinary team throughout his care. Written informed consent was obtained for the procedure and for the publication of this case report and clinical images, ensuring the confidentiality of his identity at all times. Ethics approval was obtained at the Hospital for the publication of this article under Official Letter No. CI/077/2026, Clinical Case Report Review CC7-2026. The study was conducted in accordance with the applicable Mexican regulations governing health research. The authors declare no conflicts of interest.

DISCUSSION

This case describes the perioperative management (Table 1) of a patient with complicated Stanford type B aortic dissection and evidence of visceral malperfusion, who was successfully treated with thoracic endovascular aortic repair (TEVAR). Although this procedure has demonstrated a reduction in morbidity and mortality compared with open surgery, it remains associated with potentially severe complications (9). In the patient, the procedure was complicated by a left femoral artery injury during endovascular access, which required immediate repair by the cardiovascular surgery team and transfusion of blood products; however, no major endograft-related events or neurological, cardiovascular, or renal complications occurred during the immediate perioperative period.

We consider that this favorable outcome was achieved through a structured, goal-directed anesthetic strategy focused on specific physiological objectives, which allowed the maintenance of hemodynamic stability and adequate organ perfusion despite the complexity of the procedure. As outlined in the introduction, anesthetic management was based on four key principles: avoiding abrupt increases in aortic wall stress, maintaining an individualized mean arterial pressure between 70 and 80 mmHg, preserving tissue perfusion through continuous hemodynamic and metabolic monitoring, and promoting early neurological recovery.

First, to avoid abrupt increases in aortic wall stress, a titrated induction with fentanyl and propofol was performed, combined with video laryngoscopy to mitigate the adrenergic response. This strategy is supported by the series reported by

Rehman et al., who emphasize that the primary objective is to control sympathetic responses to laryngoscopy and surgical incision in order to prevent aortic rupture (12). Although propofol was used in the present case, Liu et al. reported etomidate as another agent of choice, successfully employed in 93.1% of their patients, and preferentially considered due to its greater cardiovascular stability (13). Nevertheless, both approaches align with the recommendations of Gül and Yılmaz, who highlight that TEVAR requires strict hemodynamic control and airway management safety due to its technical complexity (14).

Similarly, as a second point, maintaining a mean arterial pressure (MAP) between 70 and 80 mmHg through norepinephrine infusion facilitated organ perfusion without exacerbating the dissection. This range is consistent with the “anti-impulse therapy” described by Kumar et al., which is designed to reduce ventricular ejection forces and limit the progression of the lesion (7). Additionally, Liu et al. suggest that although systolic blood pressure may be reduced below 90 mmHg during stent deployment to prevent migration, it is necessary to increase MAP (≥ 80 mmHg) immediately afterward to ensure spinal cord perfusion and prevent spinal cord ischemia (13).

As a third point, preservation of tissue perfusion was ensured through advanced monitoring. The use of electroencephalography in this case exceeds the standard reported by Liu et al., in which bispectral index (BIS) monitoring was performed in only 48.5% of procedures (13). Furthermore, metabolic stability, reflected by lactate levels of 0.7–1.0 mmol/L and adequate urine output, confirmed successful visceral perfusion, overcoming the risk of renal malperfusion described in the literature. Likewise, ACT control between 250 and 300 seconds complied with the recommendation of maintaining values above 200 seconds during endovascular manipulation to prevent thrombotic events (13).

Finally, as a fourth point, early neurological recovery was promoted through extubation in the operating room. This approach, defined by Liu et al. as “fast-track anesthesia,” demonstrated in their cohort a significant reduction in both ICU length of stay (from 24 to 21 hours) and overall hospitalization time (13). Immediate recovery of an alert mental status in this patient allowed for an early neurological examination, supporting the safety and feasibility of this technique for the timely detection of potential postoperative complications. Similarly, Gül and Yılmaz associate rapid recovery strategies with a reduction in prolonged intensive care unit stays (14).

The main limitation of the present study is its single-case report design, which prevents establishing causal relationships or extrapolating the findings to the general population. Additionally, anesthetic decisions were individualized according to the patient’s clinical and anatomical characteristics; therefore, they may not be

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applicable to all cases of Stanford type B aortic dissection undergoing endovascular repair. Another limitation was the absence of intraoperative transesophageal echocardiography (TEE) monitoring; however, transthoracic echocardiography (TTE) provided adequate assessment of ventricular function, aortic anatomy, and flow characteristics, allowing similar clinical objectives to be achieved while avoiding the additional invasiveness associated with TEE. Nevertheless, the value of this work lies in the systematic description of a goal-directed anesthetic strategy based on physiological objectives, complemented by a narrative review of the literature, which may contribute to clinical decision-making in similar scenarios.

The perioperative management of patients undergoing endovascular repair of Stanford type B aortic dissection should be performed through a multidisciplinary approach integrating anesthesiology, vascular surgery, cardiology, intensive care, and cardiovascular imaging. Anesthetic strategies guided by clearly defined physiological goals are recommended, prioritizing anti-impulse therapy, maintenance of an individualized mean arterial pressure to preserve vital organ perfusion, continuous hemodynamic and

metabolic monitoring, and early neurological assessment. Future prospective studies and larger case series will be required to further validate this approach and determine its impact on perioperative outcomes.

CONCLUSION

Thoracic endovascular aortic repair for Stanford type B aortic dissection represents a highly complex procedure in which anesthetic management plays a fundamental role in optimizing clinical outcomes. In the present case, a perioperative strategy structured around four objectives (avoiding increases in aortic wall stress, maintaining an individualized mean arterial pressure, preserving tissue perfusion through continuous monitoring, and promoting early neurological recovery) allowed successful management of a procedure complicated by an intraoperative vascular injury, while maintaining hemodynamic stability and a favorable postoperative course. This case highlights the importance of a protocolized, physiology-based anesthetic approach as a tool to optimize safety and outcomes during endovascular repair of aortic dissection.

Table 1. Table 1. Clinical Timeline of the Patient

Time / Phase	Key Events and Findings	Intervention and Management
Day 1 (06:00 h) Symptom onset and initial care at a secondary-level hospital	• Sudden epigastric/chest pain at rest (VAS 9/10). • Presented to the emergency department with severe arterial hypertension (200/100 mmHg). • ECG showed ischemic changes in the anterior leads. • Suspected secondary hypertension/aortic event.	• Managed as suspected acute coronary syndrome (ACS) with dual antiplatelet therapy, statins, anticoagulation, beta-blocker, and analgesia. • Referred to a tertiary care hospital due to persistent chest pain and hypertension.
Day 1 (20:00 h) Evaluation at a tertiary care hospital	• Persistent severe hypertension (220/110 mmHg). • No chest pain on physical examination.	• Adjustment of antihypertensive therapy and laboratory testing. • Contrast-enhanced CT requested. • Cardiology consultation.
Day 2 Diagnosis and ICU admission	• CT/CT angiography: Stanford type B aortic dissection (DeBakey IIb) extending to the iliac arteries, with involvement of the celiac trunk and splenic infarctions.	• ICU admission. • Anti-impulse therapy with intravenous nitroglycerin, followed by transition to oral antihypertensive therapy. • Vascular Surgery consultation and decision to perform TEVAR.
Days 3–6 Preoperative optimization	• Blood pressure controlled within target range. • Preoperative assessment: ASA IV, EuroSCORE II 2.9%, transthoracic echocardiography showing LVEF 61% and a patent intimal flap.	• Preoperative assessments by Internal Medicine and Anesthesiology. • Elective/priority scheduling of TEVAR.
Days 7–13 Antibiotic management	• Febrile episode documented. • Bacteremia ruled out by Infectious Diseases.	• Surgery postponed due to suspected bacteremia and subsequently rescheduled. • Antibiotic therapy with levofloxacin according to the antibiogram.
Day 14 Surgical procedure (TEVAR)	• Neuro-guided balanced general anesthesia. • Complication: left femoral artery injury with 1,300 mL blood loss. • Successful implantation of the aortic endograft.	• Norepinephrine infusion (target MAP: 70–80 mmHg). • Emergency vascular repair by Cardiovascular Surgery with transfusion of 2 units of packed red blood cells and fresh frozen plasma. • Extubation in the hemodynamic suite at the end of the procedure. • ICU admission for postoperative monitoring.

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Time / Phase	Key Events and Findings	Intervention and Management
Days 20 Postoperative course and discharge	15– • No neurological, metabolic, or renal deficits • Transfer from the ICU 24 hours after surgery with an adequate clinical course. • Favorable clinical course. • Hospital discharge with outpatient follow-up by Cardiology.	

Abbreviations: ACS, acute coronary syndrome; CT, computed tomography; ICU, intensive care unit; TEVAR, thoracic endovascular aortic repair; VAS, visual analog scale; MAP, mean arterial pressure; LVEF, left ventricular ejection fraction; ASA, American Society of Anesthesiologists.

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