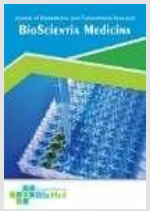
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Point-of-Care Ultrasound in the Sequential Diagnosis of Postoperative Cardiac, Pulmonary, and Vascular Complications Following Thoracoabdominal Aortic Aneurysm Repair: A Case Report and Pathophysiological Review

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ARTICLE INFO

Keywords:
Acute limb ischemia
Critical care echocardiography
Perioperative complications
Point-of-care ultrasound
Thoracoabdominal aortic aneurysm

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v9i9.1387

A B S T R A C T

Background: Open repair of a thoracoabdominal aortic aneurysm (TAAA) is a formidable surgical undertaking associated with profound physiological stress and a high risk of life-threatening postoperative complications. We present a case where a sequential, multi-system point-of-care ultrasound (POCUS) protocol was instrumental in unraveling a cascade of distinct but interconnected postoperative complications. **Case presentation:** A 67-year-old male with a Crawford Type II TAA underwent an elective open repair. His postoperative course in the Intensive Care Unit (ICU) was complicated by a cascade of events. On postoperative day one, he developed hypotensive shock. Bedside cardiac ultrasound revealed new-onset, severe left ventricular systolic dysfunction (ejection fraction ~20-25%), indicative of profound myocardial stunning. By day three, this was followed by progressive hypoxemic respiratory failure. Lung ultrasound identified a large, compressive left-sided pleural effusion, which was contributing to his respiratory decline. On day four, the patient developed signs of acute left lower limb ischemia. Vascular ultrasound confirmed a complete occlusive thrombus in the popliteal artery. This rapid series of diagnoses, all made at the bedside with POCUS, facilitated targeted interventions including the initiation of inotropic support, goal-directed diuretic therapy, and emergency surgical thrombectomy, leading to a successful patient outcome. **Conclusion:** This case highlights the unique diagnostic power of a structured, serial POCUS examination in the complex post-TAAA patient. It demonstrates how this non-invasive modality can effectively diagnose a "triple threat" of interconnected cardiac, pulmonary, and vascular complications, guiding real-time clinical decision-making and facilitating timely, life-saving interventions in the critical care setting.

1. Introduction

The surgical management of a thoracoabdominal aortic aneurysm (TAAA) represents one of the most significant challenges in modern vascular surgery and perioperative medicine.[1] These aneurysms involve both the thoracic and abdominal aorta, placing multiple vital organ systems at risk during their repair. Open surgical repair, while considered the gold standard for many patients, remains a procedure of immense magnitude and physiological impact, with reported mortality rates that vary widely, largely dependent on the extent of the aneurysm and patient comorbidities.[2]

The anatomical extent of the aneurysm, most commonly classified by the Crawford classification system, is a primary determinant of surgical complexity and perioperative risk.[3] Crawford Type II aneurysms, extending from the left subclavian artery to the infrarenal aorta, are particularly notorious for their high rates of morbidity and mortality due to the extensive nature of the repair and the prolonged ischemia times required for visceral and spinal

arteries.[4] The surgical approach necessitates a large thoracoabdominal incision and often requires single-lung ventilation, aortic cross-clamping above the celiac and renal arteries, and reimplantation of critical intercostal and visceral vessels.[5] This sequence of events unleashes a torrent of physiological insults that the anesthesiologist and intensivist must expertly manage. These challenges include: Profound Hemodynamic Derangements: Aortic cross-clamping induces acute, severe proximal hypertension and a dramatic increase in left ventricular afterload, straining the myocardium, while simultaneously causing distal hypotension and organ hypoperfusion.[6] Unclamping can precipitate precipitous vasodilatory shock, massive inflammatory mediator release, and washout of acidotic metabolites; Major Pulmonary Insult**:** Single-lung ventilation, a requisite for thoracic exposure, creates a significant ventilation/perfusion (V/Q) mismatch.[7] The thoracotomy itself induces a substantial systemic inflammatory response, pain, and diaphragmatic dysfunction, predisposing the patient to atelectasis, pneumonia, and pleural effusions. Postoperative pulmonary complications are common and represent a leading cause of morbidity; Risk of Organ Malperfusion: The brain, spinal cord, kidneys, and visceral organs are all vulnerable to ischemic injury during the cross-clamp period. Spinal cord ischemia, leading to paraplegia or paraparesis, is one of the most devastating complications. Acute kidney injury is also common and is a powerful independent predictor of mortality; Thromboembolic Events: The aneurysm sac is often lined with friable thrombus and atherosclerotic debris. Surgical manipulation carries an inherent risk of dislodging this material, resulting in distal embolization to the limbs or visceral organs.[8]

In the postoperative period, patients recovering from TAAA repair present a significant diagnostic challenge in the Intensive Care Unit (ICU). They are often mechanically ventilated, hemodynamically unstable, and tethered to multiple life-sustaining devices, making transportation for conventional imaging modalities like computed tomography (CT) both logistically difficult and inherently risky.[9] A patient's sudden deterioration can stem from a multitude of cardiac, pulmonary, vascular, or neurological causes, demanding a rapid and accurate diagnostic tool at the bedside.

Point-of-care ultrasound (POCUS) has emerged as a transformative technology in this environment. It is a non-invasive, radiation-free, and repeatable imaging modality that allows the clinician to directly visualize organ function and pathology in real-time. Its application in critical care is well-established, encompassing goal-directed echocardiography, lung ultrasonography, and the evaluation for deep vein thrombosis. Despite its widespread use, the literature often describes its utility in diagnosing isolated problems.[10]

The primary novelty of this report lies in its detailed illustration of a sequential, multi-system POCUS protocol used to unravel a complex cascade of three distinct, yet pathophysiologically linked, life-threatening complications in a single patient after TAAA repair. We aim to demonstrate how an integrated POCUS approach, moving from cardiac to pulmonary to vascular assessment, provided the diagnostic clarity necessary to navigate a rapidly evolving and multifaceted clinical crisis. This case serves as a powerful example of the indispensable role of POCUS in the hands of the modern anesthesiologist-intensivist for managing the formidable challenges of post-TAAA care.

2. Case Presentation

Informed consent for the publication of this case report was obtained from the patient. A 67-year-old male (height 170 cm, weight 75 kg) presented with a known, enlarging thoracoabdominal aortic aneurysm. His medical history was significant for long-standing, well-controlled hypertension treated with amlodipine, and chronic obstructive pulmonary disease (COPD), GOLD stage II, for which he used an ipratropium inhaler as needed. He was a former smoker with a 30-pack-year history but had quit 10 years prior. His functional capacity was good, and he was able to walk

several blocks without significant dyspnea (Metabolic Equivalent of Task [METs] > 4). He was classified as American Society of Anesthesiologists (ASA) physical status IV E due to the nature of the scheduled emergency surgery (Table 1).

Pre-operative investigations included a CT angiogram, which confirmed a Crawford Type II TAAA, extending from the distal thoracic aorta (just distal to the left subclavian artery) to the infrarenal abdominal aorta, with a maximum diameter of 7.2 cm. The aneurysm sac contained a significant burden of mural thrombus. Laboratory results were largely unremarkable, with a hemoglobin of 12.8 g/dL, a serum creatinine of 1.1 mg/dL (eGFR 68 mL/min/1.73m²), and normal coagulation studies. Pulmonary function tests showed a moderate obstructive pattern consistent with his COPD diagnosis (FEV1/FVC ratio 0.65; FEV1 70% of predicted). A transthoracic echocardiogram (TTE) demonstrated normal left ventricular size and function with an estimated ejection fraction (EF) of 60%, no significant valvular abnormalities, and Grade I (mild) diastolic dysfunction.

Table 1. Comprehensive pre-operative patient assessment.

PARAMETER	FINDING / VALUE	CLINICAL SIGNIFICANCE & PERIOPERATIVE IMPLICATION
Patient Demographics		
Age	67 years	Advanced age is an independent risk factor for perioperative morbidity and mortality in major vascular surgery.
Sex	Male	Male sex is associated with a higher prevalence of aortic aneurysms.
Body Mass Index (BMI)	25.9 kg/m²	Normal to slightly overweight range; does not pose a significant independent risk for this case.
Clinical Status & Risk Assessment		
ASA Physical Status	ASA IV E	Indicates a patient with a severe systemic disease that is a constant threat to life, undergoing an emergency procedure. High risk.
Functional Capacity (METs)	> 4 METs	Good functional capacity is a favorable prognostic indicator, suggesting adequate cardiorespiratory reserve to withstand surgical stress.
Comorbidities		
Hypertension	Well-controlled on Amlodipine	A primary risk factor for aneurysm development. Pre-operative control is crucial for managing intraoperative hemodynamic lability.
COPD	GOLD Stage II	Significantly increases risk for postoperative pulmonary complications (atelectasis, pneumonia) especially after thoracotomy and single-lung ventilation.
Smoking History	30 pack-years (quit 10 years ago)	Contributes to both atherosclerotic disease and COPD. Cessation reduces, but does not eliminate, perioperative risk.
Laboratory & Pulmonary Assessment		
Hemoglobin	12.8 g/dL	Adequate baseline for tolerating anticipated major blood loss.
Serum Creatinine / eGFR	1.1 mg/dL / 68 mL/min/1.73m²	Indicates mild chronic kidney disease. Patient is at high risk for acute kidney injury (AKI) from aortic cross-clamping.
Pulmonary Function Tests	FEV1/FVC: 0.65; FEV1: 70% predicted	Confirms moderate obstructive lung disease. Guides intraoperative ventilation strategy and predicts potential difficulty in postoperative weaning.
Cardiac & Aneurysm Imaging Assessment		
Transthoracic Echo (TTE)	Normal LV size; EF 60%; Grade I diastolic dysfunction	Reassuring baseline cardiac function, but diastolic dysfunction can predispose to fluid intolerance and pulmonary edema with aggressive resuscitation.
Aneurysm Type (CT Angio)	Crawford Type II TAAA	The most extensive type, involving aorta from subclavian to infrarenal arteries. Carries the highest risk of spinal cord ischemia, renal failure, and mortality.
Aneurysm Diameter	7.2 cm (maximum)	Large diameter indicates high rupture risk, justifying the high-risk surgical intervention.
Mural Thrombus	Significant burden noted on CTA	High risk for distal atheroembolism ("trash foot") during surgical manipulation and aortic clamping. Requires meticulous surgical technique.

Given the complexity and anticipated hemodynamic shifts, a comprehensive anesthetic plan involving combined general and thoracic epidural anesthesia was formulated. After establishing standard ASA monitoring, a 20-gauge thoracic epidural catheter was placed at the T8-T9 interspace. Anesthesia was induced with fentanyl 200 mcg, propofol 150 mg, and rocuronium 50 mg. The airway was secured with a 39-Fr left-sided double-lumen endotracheal tube, with placement confirmed by fiberoptic bronchoscopy (Table 2).

Invasive monitoring was established, including a right radial arterial line, a left femoral arterial line (for distal perfusion pressure monitoring), a 9-Fr multi-lumen central venous catheter via the right internal jugular vein, and a pulmonary artery catheter (PAC). A transesophageal echocardiography (TEE) probe was also inserted for intraoperative cardiac assessment. Anesthesia was maintained with a total intravenous anesthesia (TIVA) technique using infusions of propofol (80-120 mcg/kg/min) and remifentanil (0.1-0.2 mcg/kg/min).

Table 2. Detailed intraoperative management.

PHASE / PARAMETER	DETAILS / VALUE	ANESTHETIC RATIONALE & MANAGEMENT
Anesthetic Management		
Anesthetic Technique	Combined General Anesthesia (TIVA) + Thoracic Epidural Anesthesia (TEA)	TIVA provides stable hemodynamics. TEA offers superior postoperative analgesia, reduces surgical stress response, and may improve gut motility.
Induction Agents	Fentanyl 200 mcg, Propofol 150 mg, Rocuronium 50 mg	Standard balanced induction aimed at ensuring hemodynamic stability and facilitating smooth airway instrumentation.
Maintenance	Propofol (80-120 mcg/kg/min) + Remifentanil (0.1-0.2 mcg/kg/min)	TIVA with short-acting agents allows for precise titration, rapid emergence, and neurological assessment post-procedure.
Epidural Analgesia	T8-T9 placement; Bupivacaine 0.1% with Fentanyl 2 mcg/mL	Provides excellent somatic and visceral analgesia for the thoracoabdominal incision, reducing opioid requirements.
Invasive Monitoring & Airway		
Arterial Lines	Right Radial (proximal) & Left Femoral (distal)	Dual lines are essential to monitor proximal (afterload) and distal (organ perfusion) pressures during aortic cross-clamping.
Central Access	Right IJ 9-Fr CVC; Pulmonary Artery Catheter (PAC)	Large-bore CVC for rapid volume resuscitation. PAC for monitoring cardiac output, filling pressures, and guiding fluid/inotropic therapy.
Airway Management	39-Fr Left Double-Lumen Endotracheal Tube	Required for single-lung ventilation of the right lung to facilitate surgical exposure of the thoracic aorta.
Cardiac Monitoring	Transesophageal Echocardiography (TEE)	Provides real-time assessment of ventricular function, volume status, and valvular integrity, especially during hemodynamic stress.
Surgical & Hemodynamic Events		
Total Surgical Time	7 hours, 45 minutes	Prolonged duration increases risks of hypothermia, coagulopathy, and fluid shifts.
Aortic Cross-Clamp Time	82 minutes	The most critical phase. Longer duration directly correlates with higher risk of spinal cord, renal, and visceral ischemia.
Cross-Clamp Hemodynamics	Proximal BP: 180/100 mmHg; Distal BP: 35 mmHg	Classic presentation. Proximal hypertension managed with esmolol to reduce LV afterload. Distal hypotension tolerated as an obligate part of the procedure.
Post-Unclamping	Severe Hypotension (MAP < 55 mmHg)	Declamping shock due to central hypovolemia, reperfusion acidosis, and inflammatory mediator release. Managed with vasopressors and volume.
Vasopressor Requirement	Norepinephrine up to 0.2 mcg/kg/min	Required to counteract profound vasodilation and maintain organ perfusion pressure after clamp release.
Fluid & Blood Product Resuscitation		
Estimated Blood Loss (EBL)	4.0 Liters	Represents approximately one full blood volume loss, necessitating massive transfusion.
Crystalloid / Colloid	3.5 L / 1.0 L	Used for initial resuscitation and maintenance, but limited to prevent excessive edema and coagulopathy.
Blood Products	6 units PRBCs, 4 units FFP, 1 unit Platelets	Balanced transfusion guided by ROTEM to correct coagulopathy and restore oxygen-carrying capacity. A ratio approaching 1:1:1 (RBC:FFP:Plt) is targeted.

The patient was positioned in the right lateral decubitus position. The surgery commenced via a long thoracoabdominal incision through the 8th intercostal space. Single-lung ventilation of the right lung was initiated. The total surgical duration was 7 hours and 45 minutes. The aorta was cross-clamped proximally between the left carotid and subclavian arteries and distally at the infrarenal aorta. The total aortic cross-clamp time was 82 minutes. During this period, critical intercostal arteries (T8-T12) and the celiac, superior mesenteric, and right renal arteries were successfully reimplanted onto the 24 mm Dacron graft.

Intraoperatively, the patient experienced significant hemodynamic lability (Figure 1). During the cross-clamp phase, proximal arterial pressure increased to 180/100 mmHg, managed with an esmolol infusion, while distal femoral pressure dropped to 35 mmHg. Upon clamp release, the patient became severely hypotensive (MAP < 55 mmHg), requiring initiation of a norepinephrine infusion (titrated up to 0.2 mcg/kg/min) and aggressive fluid resuscitation. The TEE assessment during this period showed transient LV hypokinesis but with recovery. Total estimated blood loss was 4.0 liters. Resuscitation consisted of 3.5 liters of crystalloid, 1 liter of colloid, 6 units of packed red blood cells (PRBCs), 4 units of fresh frozen plasma (FFP), and 1 unit of apheresis platelets, guided by PAC-derived cardiac output and rotational thromboelastometry (ROTEM) data. The patient was transferred to the ICU, intubated, sedated, and required continued norepinephrine infusion.

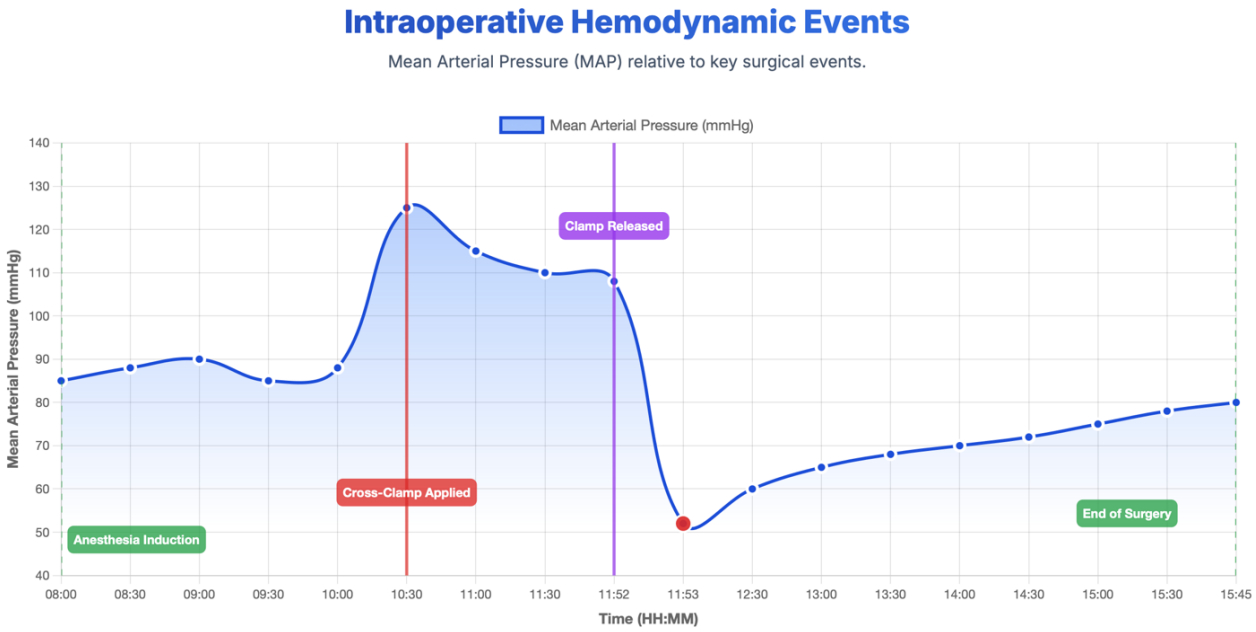

Figure 1. Intraoperative hemodynamic events.

Upon ICU admission, the patient remained mechanically ventilated on Synchronized Intermittent-Mandatory Ventilation with Pressure Control (SIMV-PC), with a set rate of 12 breaths/min, pressure control of 18 cmH_2O, and PEEP of 8 cmH_2O, requiring an FiO_2 of 0.6. His blood pressure was 93/56 mmHg on norepinephrine 0.15 mcg/kg/min. Over the next 12 hours, despite ongoing resuscitation, his hemodynamic status deteriorated, with increasing vasopressor requirements to maintain a MAP > 65 mmHg and development of oliguria (Table 3).

Table 3. Postoperative ICU course and management.

POST-OP DAY	CLINICAL PROBLEM / EVENT	KEY DIAGNOSTIC FINDINGS (POCUS)	MANAGEMENT & OUTCOME
Day 0-1	**Refractory Shock** • Deteriorating hemodynamics despite fluid resuscitation. • Increasing norepinephrine requirement to maintain MAP > 65 mmHg. • Development of oliguria.	**Focused Cardiac Ultrasound (FoCUS)** • Dilated left ventricle. • Severe global systolic dysfunction. • Estimated EF ~20-25% (new finding). • Diagnosis: Postoperative myocardial stunning.	**Intervention** • Diagnosis shifted to cardiogenic shock. • Milrinone infusion initiated for inotropic support. • Norepinephrine continued for vasopressor support. • Outcome: Gradual hemodynamic stabilization.
Day 2-3	**Progressive Hypoxemic Respiratory Failure** • Increased work of breathing and tachypnea. • Worsening oxygenation (FiO_2 increased to 0.8). • PaO_2/FiO_2 ratio dropped to 150.	**Lung Ultrasound (LUS)** • Large anechoic space in left hemithorax. • Compressed, atelectatic left lower lobe. • Estimated effusion volume >1000 mL. • Diagnosis: Large, compressive pleural effusion.	**Intervention** • Aggressive medical management initiated. • Continuous Furosemide infusion (5 mg/hr). • Avoided high-risk invasive thoracentesis. • Outcome: Gradual improvement in respiratory mechanics.
Day 4	**Acute Left Lower Limb Ischemia** • Patient reports severe leg pain. • Exam: Cool, pale foot, non-palpable distal pulses. • Differential SpO_2: 86% (Left) vs 98% (Right).	**Vascular Ultrasound** • Non-compressible, hypoechoic material in popliteal artery. • Absence of color and pulsed-wave Doppler flow. • Diagnosis: Complete occlusive popliteal artery thrombus.	**Intervention** • Immediate vascular surgery consultation. • Emergency open thrombectomy performed. • Outcome: Successful restoration of distal blood flow.
Day 5	**Rapid Clinical Improvement** • Marked improvement following thrombectomy. • Hemodynamic stability achieved. • Improved respiratory status.	**Clinical Reassessment** • No further POCUS indicated at this stage. • Focus on clinical parameters of recovery.	**Management** • Weaning of all vasopressor and inotropic support. • Successful extubation to high-flow nasal cannula. • Outcome: Patient awake, alert, and stable.
Day 6	**ICU Discharge** • Patient meets all criteria for ICU step-down. • No active cardiorespiratory issues. • Full recovery of function in left leg.	**N/A** • -	**Management** • Transfer to the surgical ward for continued recovery. • Ultimate hospital discharge on Day 12.

A focused cardiac ultrasound (FoCUS) was performed at the bedside using a phased array probe to investigate the cause of the refractory shock. The parasternal long-axis and apical four-chamber views revealed a dilated left ventricle with severe, global systolic dysfunction. The visual estimation of the ejection fraction was severely reduced, in the range of 20-25%, a stark contrast to his pre-operative baseline of 60%. There was no evidence of right ventricular failure or significant pericardial effusion. These findings were consistent with profound postoperative myocardial stunning or demand ischemia. Based on these findings, a milrinone infusion was initiated for inotropic support and afterload reduction, and the norepinephrine infusion was continued.

The patient's hemodynamic status gradually stabilized on combined inotrope and vasopressor support. However, by postoperative day 3, his respiratory status began to decline. His total respiratory rate increased to 26-28 breaths per minute, with increased work of breathing and ventilator dyssynchrony. His oxygenation worsened, requiring an increase in FiO_2 to 0.8 to maintain an SpO_2 of 93-96%. His PaO_2/FiO_2 ratio fell from 250 to 150.

A bedside lung ultrasound was performed to assess the cause of the acute hypoxemic respiratory failure. Examination of the right lung showed a normal "lung sliding" sign with A-lines, indicating good aeration. However, examination of the left posterior-lateral

chest wall revealed a large, anechoic (black) space separating the parietal and visceral pleura. The underlying lung appeared compressed and atelectatic, exhibiting a "sinusoid sign" (inspiratory movement of the lung toward the pleura), confirming the presence of a large liquid pleural effusion. The depth of the effusion was measured at 5.2 cm, corresponding to an estimated volume of over 1000 mL. This large, compressive effusion was deemed a major contributor to his respiratory compromise. A decision was made to pursue aggressive medical management with intravenous furosemide (administered as an infusion of 5 mg/hour) to reduce the effusion size gradually, given the patient's ongoing coagulopathy and hemodynamic fragility, which made invasive thoracentesis a higher-risk option.

On the morning of postoperative day 4, the patient, now more awake, began to complain of severe, constant pain in his left lower leg. A physical examination revealed a cool, pale left foot with diminished capillary refill. The dorsalis pedis and posterior tibial pulses were non-palpable. Sensation to light touch was decreased over the dorsum of the foot. Crucially, pulse oximetry showed a stark difference in saturation between the extremities: 98% on the right foot versus 86% on the left foot. An urgent bedside vascular ultrasound was performed with a high-frequency linear transducer. The examination revealed a patent common femoral artery and superficial femoral artery. However, in the popliteal fossa, the artery was distended with an intraluminal, hypoechoic, and non-compressible mass, consistent with an acute thrombus. Color and pulsed-wave Doppler imaging confirmed the complete absence of arterial blood flow distal to this occlusion.

With a definitive diagnosis of acute limb ischemia secondary to popliteal artery thrombosis, the vascular surgery team was consulted immediately. The patient was taken emergently for a left lower extremity open thrombectomy. A posterior approach to the popliteal artery was used, and a significant amount of acute thrombus was extracted. A completion angiogram confirmed the successful restoration of brisk, three-vessel runoff to the foot.

Following the thrombectomy, the patient's clinical course improved dramatically. He was weaned from vasopressor and inotropic support over the next 24 hours. His respiratory status improved, and he was successfully extubated to a high-flow nasal cannula on postoperative day 5. He was transferred out of the ICU to the surgical ward on day 6 and was ultimately discharged from the hospital on day 12 with full recovery of function in his left leg.

3. Discussion

The postoperative management of a patient following open thoracoabdominal aortic aneurysm (TAAA) repair is a masterclass in vigilance, demanding a profound understanding of complex physiology and the ability to respond to rapidly evolving, multi-system organ failure.[11] The case presented here is a compelling example of this reality, illustrating a "triple threat" of cascading organ system failure—cardiac, pulmonary, and vascular—where each complication likely contributed to the development of the next. The true educational and scientific value of this case lies in its demonstration of how a structured, protocol-driven point-of-care ultrasound (POCUS) examination was the linchpin of the diagnostic process, allowing the ICU team to dissect a complex clinical picture in real-time at the bedside.[12] This discussion will provide an in-depth analysis of the pathophysiological cascade, the critical role of perioperative management, and the integrated diagnostic power of a multi-system POCUS approach.

The first domino to fall, and arguably the engine driving the entire subsequent cascade of complications, was the patient's acute cardiac failure. The development of severe, new-onset left ventricular dysfunction is a well-recognized entity after major non-cardiac surgery, often termed postoperative myocardial injury or stunning.[13] The pathophysiology is multifactorial but is particularly pronounced and mechanistically distinct after TAAA repair (Figure 2).

The primary insult is the aortic cross-clamping itself. The application of a clamp to the thoracic aorta

creates an abrupt and massive increase in systemic vascular resistance, effectively forcing the left ventricle to eject its stroke volume into a severely truncated and high-resistance arterial system.[14] This "hypertensive crisis" dramatically increases myocardial wall stress, which, according to the Law of Laplace, is a primary determinant of myocardial oxygen demand (MVO_2). In this patient, the intraoperative MAP surge to 180/100 mmHg represented a period of extreme afterload mismatch, where MVO_2 likely far outstripped the coronary perfusion capabilities, leading to global subendocardial ischemia even in the absence of anatomically significant coronary artery disease. This is not a focal ischemic event but a global supply-demand mismatch injury.

This initial ischemic insult is then compounded by a "second hit" upon clamp release: ischemia-reperfusion injury. The restoration of blood flow to the previously ischemic distal tissues unleashes a torrent of inflammatory mediators—including tumor necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-6), and reactive oxygen species—into the systemic circulation.[15] These mediators have direct myocardial depressant effects, impairing myocyte contractility and calcium handling, further contributing to the stunned state of the ventricle. The profound vasodilation and acidosis that characterize declamping shock can also impair coronary perfusion pressure, exacerbating the initial injury.

In this patient, the POCUS finding of severe global hypokinesis on day one was the critical first diagnosis. In the undifferentiated shock state, the differential diagnosis is broad, encompassing hypovolemia, distributive shock (from SIRS), and cardiogenic shock.[15] Without imaging, the management might have defaulted to continued aggressive fluid resuscitation, which, in the setting of a failing ventricle, would have been disastrous, leading to pulmonary edema and worsening organ failure. The FoCUS examination immediately narrowed the differential, definitively identifying a cardiogenic component. This shifted the therapeutic paradigm from simple volume replacement to targeted inotropic support. The choice of milrinone, a phosphodiesterase-3 inhibitor, was rational in this context. Its inotropic effects improve contractility, while its vasodilatory properties (inodilatation) help to reduce the pathologically elevated afterload, effectively "uncoupling" the failing ventricle from the high-resistance state.[16] This swift, ultrasound-guided diagnosis and targeted therapy were crucial in preventing a spiral into irreversible organ failure and set the stage for the subsequent events.

The patient's subsequent respiratory failure, manifesting on day three, was driven by the large left-sided pleural effusion. The genesis of this effusion was a perfect storm of hydrostatic and inflammatory pressures, directly linked to both the surgical procedure and the preceding cardiac failure.[16] Firstly, the left thoracoabdominal approach itself is a significant inflammatory trigger. The extensive surgical dissection, retraction of the lung, and irritation of the pleura cause a localized inflammatory response, increasing capillary permeability and favoring the formation of a reactive, exudative pleural effusion. This is compounded by the physiological stress of one-lung ventilation, which can lead to atelectasis and inflammatory changes (biotrauma) in the dependent, surgically manipulated lung.

However, the most significant contributing factor, which likely transformed a small, expected reactive effusion into a large, hemodynamically significant collection, was the preceding cardiac failure. The severe LV dysfunction diagnosed on day one would have led to a substantial increase in left ventricular end-diastolic pressure (LVEDP) and, consequently, elevated left atrial and pulmonary capillary wedge pressures.[17] This increase in hydrostatic pressure pushes fluid from the pulmonary capillaries into the interstitial space and, eventually, across the visceral pleura into the pleural space. This cardiogenic, transudative fluid added volume to the existing inflammatory, exudative effusion, creating a large, mixed-etiology fluid collection.

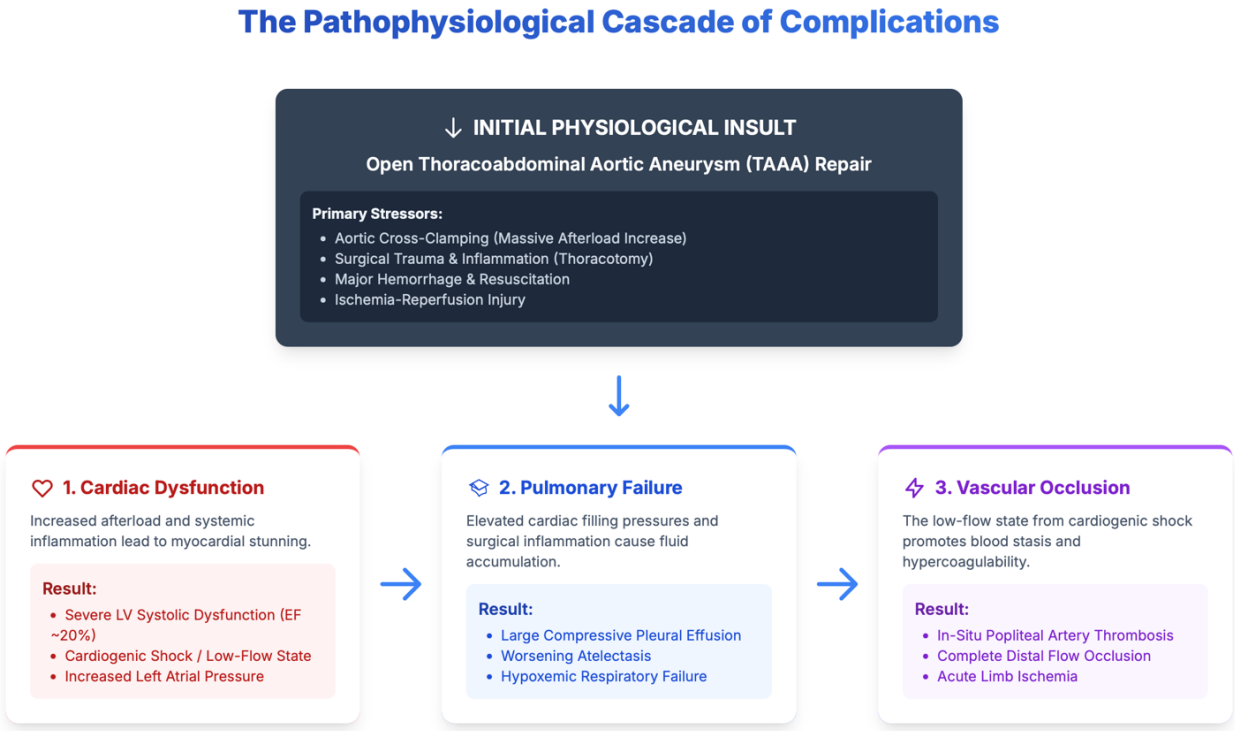

Figure 2. The pathophysiological cascade of complications.

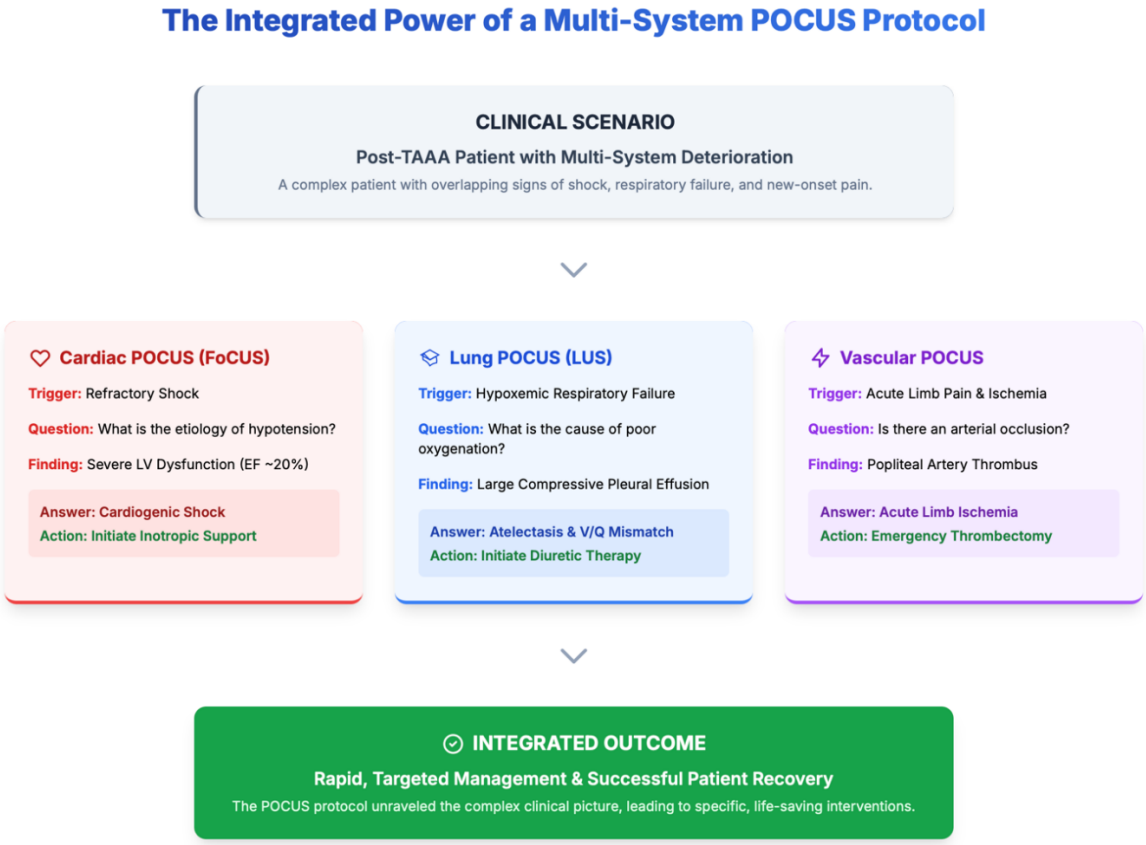

Figure 3. The integrated power of a multi-system POCUS protocol.

The role of lung ultrasound was pivotal in this phase. A standard portable chest X-ray in a supine ICU patient is notoriously insensitive and non-specific, often showing just a diffuse "white-out" of the hemithorax.[18] It cannot reliably distinguish between effusion, consolidation, or atelectasis. In contrast, POCUS provided a wealth of diagnostic information. It not only confirmed the presence of a large fluid collection but also characterized it as a simple, anechoic effusion (suggesting a transudative component) and allowed for a semi-quantitative estimation of its volume (over 1000 mL based on a depth of 5.2 cm). This finding immediately explained the patient's worsening hypoxemia: the large effusion was causing compressive atelectasis of the left lower lobe, creating a massive intrapulmonary shunt and V/Q mismatch.

This definitive diagnosis guided a critical management decision. The team was faced with two options: invasive therapeutic thoracentesis or medical management with diuretics. In a hemodynamically fragile patient with likely procedural coagulopathy, an invasive procedure carries a significant risk of pneumothorax or hemothorax. The ultrasound finding of a simple, anechoic effusion, strongly suggesting a significant cardiogenic/hydrostatic component, provided the confidence to pursue a less invasive strategy first. Aggressive diuretic therapy with a continuous furosemide infusion was a logical choice to offload the hydrostatic pressure. This ultrasound-informed decision mitigated risk while still directly addressing the underlying pathophysiology.[18]

The final and most limb-threatening complication was the acute popliteal artery occlusion on day four. In the post-TAAA repair setting, the differential for such an event is primarily between two mechanisms: atheroembolism and in-situ thrombosis. Atheroembolism involves the dislodgement of friable thrombus or atherosclerotic plaque from the native aorta during surgical clamping or manipulation, which then travels downstream to occlude a distal vessel.[19] While this is always a possibility, a second mechanism, in-situ thrombosis, is strongly suggested by the clinical sequence in this case. The patient had entered a profound low-flow state secondary to his severe cardiogenic shock. This period of circulatory stasis, combined with the systemic hypercoagulable state that follows major surgery, perfectly fulfills the components of Virchow's triad: 1) Endothelial Injury: The aorta and distal vessels were subjected to surgical manipulation, clamping, and the systemic inflammatory response, all of which activate the vascular endothelium, making it prothrombotic; 2) Stasis of Blood Flow: The severe cardiogenic shock, with an EF of ~20%, would have dramatically reduced blood flow velocity in the distal arterial tree, allowing clotting factors to accumulate and promoting thrombus formation; and 3) Hypercoagulability: Major surgery is a potent trigger for a systemic hypercoagulable state, driven by the release of tissue factor and inflammatory cytokines, leading to an increase in circulating procoagulant factors.

It is highly plausible that this "perfect storm" of endothelial injury, stasis, and hypercoagulability created the ideal conditions for a thrombus to form de novo within the popliteal artery. The preceding cardiac failure was not merely an incidental finding; it was the direct cause of the low-flow state that precipitated the thrombosis. The diagnostic contribution of POCUS at this juncture cannot be overstated.[20] The patient was too unstable and encumbered for safe and timely transport to a CT angiography suite. Bedside vascular ultrasound provided a definitive diagnosis within minutes. It not only identified the intraluminal, hypoechoic thrombus but also, crucially, confirmed its functional significance by demonstrating the complete absence of color and pulsed-wave Doppler flow. This rapid, unequivocal diagnosis eliminated any ambiguity, allowing for immediate surgical consultation and mobilization for an emergency thrombectomy, which was undoubtedly the key intervention in salvaging the limb.

This case powerfully advocates for moving beyond single-system POCUS examinations towards an integrated, protocol-based approach in complex critical care patients (Figure 3). The intensivist was

able to seamlessly transition from a cardiac assessment (FoCUS) to a pulmonary assessment (LUS) and finally to a vascular assessment, with each examination building upon the findings of the last. This systematic approach, often referred to as a "head-to-toe" ultrasound survey, transforms POCUS from a tool that simply answers isolated questions into a powerful narrative device that allows the clinician to understand the underlying pathophysiological story of their patient's decline.

The identification of cardiogenic shock provided the crucial context for all subsequent events. Understanding the presence of severe LV dysfunction immediately raised the suspicion that the developing respiratory failure had a cardiogenic component, which the lung ultrasound confirmed by identifying a large effusion. The knowledge of a persistent low-flow state (from the cardiac dysfunction) made in-situ thrombosis a leading hypothesis for the acute limb pain, a suspicion that vascular POCUS rapidly confirmed. This structured approach facilitates rapid, rational, and targeted therapy, as demonstrated at each stage of this patient's care, and represents a paradigm shift in the management of the undifferentiated, critically ill patient.[20] It embodies the principle of bringing the diagnostic modality to the patient, rather than the patient to the modality, a cornerstone of modern critical care medicine.

4. Conclusion

This case report highlights the complex and often interconnected nature of complications following open thoracoabdominal aortic aneurysm repair. It underscores the indispensable role of a serial, multi-system point-of-care ultrasound protocol as a primary diagnostic tool for the modern anesthesiologist-intensivist. By facilitating the rapid, sequential diagnosis of severe cardiac dysfunction, a compressive pleural effusion, and an occlusive arterial thrombus at the bedside, POCUS enabled a cascade of timely and targeted interventions that were critical to achieving a successful outcome. This case serves as an important reminder of the diagnostic power of this versatile technology in navigating the multifaceted and life-threatening challenges of critical care medicine.

5. References

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