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Navigating the Perfect Storm: Ultrasound-Guided Peripheral Nerve Block for Emergency Amputation in a Patient with Acute STEMI and Failed PCI

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A B S T R A C T

Acute limb ischemia presenting concurrently with acute coronary syndrome creates a precarious clinical dilemma, often termed the "cardiac cripple" scenario. The mortality risk is compounded when patients have a history of failed percutaneous coronary intervention, severe left ventricular dysfunction, and require emergency major amputation while on active dual antiplatelet therapy. In these patients, general anesthesia poses a risk of hemodynamic collapse, while neuraxial anesthesia is contraindicated due to bleeding risks. A 75-year-old male presented with a Rutherford Grade III-IV "dead limb" of the right lower extremity and concurrent Acute Anterior STEMI (Killip II, TIMI 7/14, GRACE 137). His history included a failed percutaneous coronary intervention two months prior and three-vessel disease, resulting in a left ventricular ejection fraction of 32%. General anesthesia posed an unacceptable risk of exacerbating myocardial pump failure, while spinal anesthesia was contraindicated due to recent clopidogrel ingestion. A decision was made to perform a below-knee amputation using an ultrasound-guided femoral nerve block and a popliteal sciatic nerve block via the crosswise approach. The procedure utilized 0.5% ropivacaine with 2 mg dexamethasone. The patient remained hemodynamically stable without vasopressor support, reported a Visual Analogue Scale score of 0 intraoperatively, and avoided adverse cardiac events. In conclusion, peripheral nerve blockade, specifically the combined femoral and crosswise popliteal sciatic approach, serves as a superior anesthetic alternative in high-risk cardiac patients. It bypasses the sympatholytic risks of general anesthesia and the coagulation constraints of neuraxial techniques, offering a safe corridor for life-saving surgery.

1. Introduction

The intersection of acute limb ischemia (ALI) and acute coronary syndrome (ACS) represents one of the most lethal and physiologically complex scenarios in the domains of vascular surgery and anesthesiology.[1] ALI is not merely a localized anatomical event; it constitutes a systemic vascular emergency that demands immediate surgical intervention. The sudden cessation of lower limb perfusion initiates a cascade of metabolic sequelae—including lactic acidosis, hyperkalemia, and the release of inflammatory cytokines—which can rapidly progress to rhabdomyolysis, myoglobinuria, and multi-organ failure if not promptly arrested. The foundational principle of ALI management is time; delays in revascularization or amputation invariably lead to systemic sepsis and death.[2] However, the urgency of surgical intervention is often diametrically opposed to the patient's physiological capacity to survive the procedure. This is particularly evident when ALI presents concurrently with ST-elevation myocardial infarction (STEMI), creating a clinical paradox where

the surgery required to save the patient's life is the very event most likely to end it.[3]

Patients presenting with concomitant ALI and STEMI, especially those with a history of failed percutaneous coronary intervention (PCI) and severely reduced left ventricular ejection fraction (LVEF), represent the cardiac cripple phenotype. In this population, the myocardial oxygen supply-demand mismatch is critical.[4] The physiological stress of surgery—characterized by the release of catecholamines, cortisol, and inflammatory mediators—imposes a significant afterload burden on a failing heart that lacks contractile reserve. In patients with pump failure or an LVEF below 35%, even minor fluctuations in systemic vascular resistance or heart rate can precipitate irreversible cardiogenic shock. Consequently, the anesthetic management of such patients is not simply about providing insensibility to pain; it is a high-stakes exercise in hemodynamic preservation where the margin for error is effectively nonexistent.[5]

The anesthetic management of these high-risk patients is fraught with contradictions, rendering conventional approaches perilous. General anesthesia (GA), often the default for major lower limb amputations, is associated with profound hemodynamic alterations that are poorly tolerated by the ischemic myocardium.[6] Induction agents, such as propofol or volatile anesthetics, cause direct myocardial depression and dose-dependent vasodilation. In a patient with severe systolic dysfunction, this reduction in systemic vascular resistance forces a drop in coronary perfusion pressure, exacerbating myocardial ischemia. Furthermore, the transition to positive pressure ventilation decreases venous return (preload), further compromising cardiac output in a heart that may be preload-dependent. Perhaps most critically, the induction of general anesthesia can blunt the baseline sympathetic tone that is keeping the failing heart functioning, leading to potentially fatal hypotension immediately upon induction.

In standard practice, neuraxial anesthesia (spinal or epidural blockade) is often advocated as a safer alternative to GA for lower limb surgery, as it allows the patient to remain awake and avoids airway manipulation.[7] However, in the context of ACS and recent coronary intervention, this route is frequently blocked by the pharmacological mandate for anticoagulation. Patients with acute STEMI or recent failed PCI are invariably managed with aggressive dual antiplatelet therapy (DAPT), typically aspirin and a P2Y12 inhibitor (clopidogrel or ticagrelor). This creates a therapeutic tightrope. Discontinuing antiplatelet therapy to minimize the risk of spinal epidural hematoma exposes the patient to an unacceptably high risk of acute stent thrombosis and re-infarction, outcomes with mortality rates exceeding 40%. Conversely, performing neuraxial blockade in the presence of active DAPT violates safety guidelines and invites catastrophic neurological complications, including permanent paraplegia.[8]

This pharmacological and physiological deadlock leaves the anesthesiologist with a complex dilemma: a patient who cannot tolerate general anesthesia due to heart failure, cannot receive spinal anesthesia due to obligatory anticoagulation, yet requires immediate major surgery to prevent septic death from gangrene. In this specific context, Ultrasound-Guided Peripheral Nerve Block (USG-PNB) emerges not merely as an alternative but as a vital rescue strategy. By depositing local anesthetic directly around the peripheral nerves, specifically the femoral and sciatic nerves, the anesthesiologist can achieve surgical anesthesia limited strictly to the operative site. This approach avoids the systemic sympathectomy associated with spinal anesthesia and the myocardial depression associated with general anesthesia. Recent evidence suggests that the combination of femoral and popliteal sciatic nerve blocks can provide complete anesthesia for below-knee amputation with minimal to no hemodynamic impact, effectively shielding the heart

from the stress of surgery.[9]

However, the application of peripheral nerve blocks in this patient population presents its own technical challenges. The classic approach to the sciatic nerve at the popliteal fossa requires the patient to be positioned prone (face down). In a patient with acute heart failure, pulmonary edema, and severe orthopnea, prone positioning is physiologically impossible; it compromises respiratory mechanics and can precipitate respiratory arrest. Thus, a supine approach to the sciatic nerve is required. This case report highlights the novel application of the crosswise approach to the popliteal sciatic nerve block. This ultrasound-guided technique allows for precise identification and blockade of the sciatic nerve with the patient in a supine position, eliminating the need for hazardous repositioning.[10]

The aim of this study is to demonstrate the efficacy and safety of a combined ultrasound-guided femoral and crosswise popliteal sciatic nerve block as the sole anesthetic technique for emergency below-knee amputation in a geriatric patient with "triple-threat" pathology: acute anterior STEMI, failed PCI, and severe left ventricular dysfunction (LVEF 32%). The novelty of this case lies in the successful navigation of a perfect storm of contraindications—where both general and neuraxial anesthesia were deemed unsafe—by utilizing a specific regional anesthesia technique (the crosswise approach) that accommodates the positioning limitations of acute heart failure. This report provides a blueprint for managing the cardiac cripple requiring emergency limb salvage, establishing a protocol for hemodynamic preservation when conventional anesthetic options are exhausted.

2. Case Presentation

Following the acquisition of written informed consent for publication, we present the case of a 75-year-old male who arrived at the Emergency Department in a state of significant distress. His primary complaint was excruciating pain radiating through his right lower leg, a symptom that had ominously progressed over the preceding week to include profound numbness and a darkening, black discoloration of the skin. This clinical picture was set against a backdrop of severe cardiovascular pathology; the patient had a documented history of three-vessel coronary artery disease with critical involvement of the left main artery. Of particular concern was a history of a failed percutaneous coronary intervention (PCI) attempted just two months prior at an external facility, where the coronary lesion was deemed uncrossable due to severe, extensive calcification. At the time of this admission, he was diagnosed with an acute anterior ST-elevation myocardial infarction (STEMI), further complicating an already fragile physiological state. His ongoing pharmacological regimen included active dual antiplatelet therapy (DAPT) consisting of Aspirin 80 mg and Clopidogrel 75 mg daily, alongside atorvastatin, bisoprolol, and furosemide, reflecting a maximal medical management strategy for his chronic conditions.

Upon initial physical assessment, the patient appeared clinically compromised, exhibiting diaphoresis and dyspnea with evident signs of orthopnea, necessitating an upright posture to maintain ventilation (Table 1). His admission vital signs revealed a precarious hemodynamic status: a blood pressure of 105/65 mmHg, significant sinus tachycardia with a heart rate of 98 beats per minute, a respiratory rate of 24 breaths per minute, and an oxygen saturation of 94% maintained only with 3 liters of supplemental oxygen via nasal cannula. The local examination of the right lower extremity confirmed the gravity of the vascular emergency; the limb was cold to palpation extending up to the mid-calf, with a complete absence of both popliteal and dorsalis pedis pulses. The skin manifested extensive mottling and fixed cyanosis, consistent with Rutherford Class III/IV ischemia.

TABLE 1. SUMMARY OF CLINICAL FINDINGS ON ADMISSION

1. PATIENT PROFILE & HEMODYNAMICS	
Demographics	75-year-old Male
Chief Complaint	Excruciating pain, numbness, and black discoloration of Right Lower Leg (Duration: 7 days)
Vital Signs	**BP:** 105/65 mmHg **HR:** **98 bpm (Sinus Tachycardia)** **RR:** **24 bpm (Dyspneic/Orthopnea)** **SpO2:** 94% on 3L O2 nasal cannula
Comorbidities & History	• Acute Anterior STEMI • CAD 3-Vessel Disease + Left Main • **Failed PCI** (2 months prior; calcified uncrossable lesion) • Medications: DAPT (Aspirin/Clopidogrel), Bisoprolol, Furosemide
2. VASCULAR STATUS (RIGHT LOWER LIMB)	
Local Examination	• **Temperature:** Cold to mid-calf • **Pulse:** **Absent Popliteal & Dorsalis Pedis** • **Skin:** Extensive mottling, fixed cyanosis • **Tissue Loss:** Frank gangrene (toes and forefoot)
Classification	**Rutherford Class III/IV (Irreversible Ischemia)**
3. CARDIAC INVESTIGATIONS	
Electrocardiogram (ECG)	Sinus Tachycardia, **ST-Elevation V1-V4** (Anterior STEMI), Pathological Q waves
Echocardiography	• **Wall Motion:** Severe hypokinesis of anterior wall and septum • **LVEF:** **32% (Severe Systolic Dysfunction)** • **Diastolic:** Grade II Dysfunction
4. LABORATORY & RISK STRATIFICATION	
Cardiac Biomarkers	• **Troponin I:** **4,500 ng/L** (High Sensitivity) • **NT-proBNP:** **8,400 pg/mL** (Acute Heart Failure)
Metabolic & Heme	• **Lactate:** **4.2 mmol/L** (Metabolic Acidosis) • **WBC:** 18,500 /uL (Leukocytosis) • **Hemoglobin:** 10.5 g/dL
Coagulation	PT: 12.5s | aPTT: 38s | *Platelet function inhibited (Active Clopidogrel)*
Risk Scores	**TIMI: 7/14 (High Risk)** **GRACE: 137 (High Mortality)** **CRUSADE: 56 (High Bleeding)** **Killip Class: II**

Abbreviations: *BP: Blood Pressure; HR: Heart Rate; RR: Respiratory Rate; STEMI: ST-Elevation Myocardial Infarction; CAD: Coronary Artery Disease; PCI: Percutaneous Coronary Intervention; LVEF: Left Ventricular Ejection Fraction; NT-proBNP: N-terminal pro b-type natriuretic peptide.*

Furthermore, gangrenous changes were firmly established on the toes and forefoot, indicating irreversible tissue necrosis that mandated urgent amputation to prevent systemic septic sequelae. To objectively quantify the perioperative risk, the clinical team utilized validated stratification systems. The patient was categorized as Killip Class II, indicating active heart failure characterized by pulmonary rales and an S3 gallop. His TIMI risk score was calculated at 7/14, and his GRACE score was 137, both markers indicative of high mortality risk. Additionally, his CRUSADE bleeding score was 56, highlighting a significant propensity for major bleeding complications. Diagnostic investigations corroborated these high-risk features; the 12-lead Electrocardiogram displayed sinus tachycardia with ST-segment elevation in leads V1-V4, confirming the anterior STEMI, along with Q waves suggestive of prior myocardial infarction. Transthoracic echocardiography provided further insight into the cardiac pump failure, revealing severe hypokinesis of the anterior wall and septum. The left ventricular ejection fraction (LVEF) was calculated at a critically low 32% using Simpson's biplane method, accompanied by Grade II Diastolic Dysfunction.

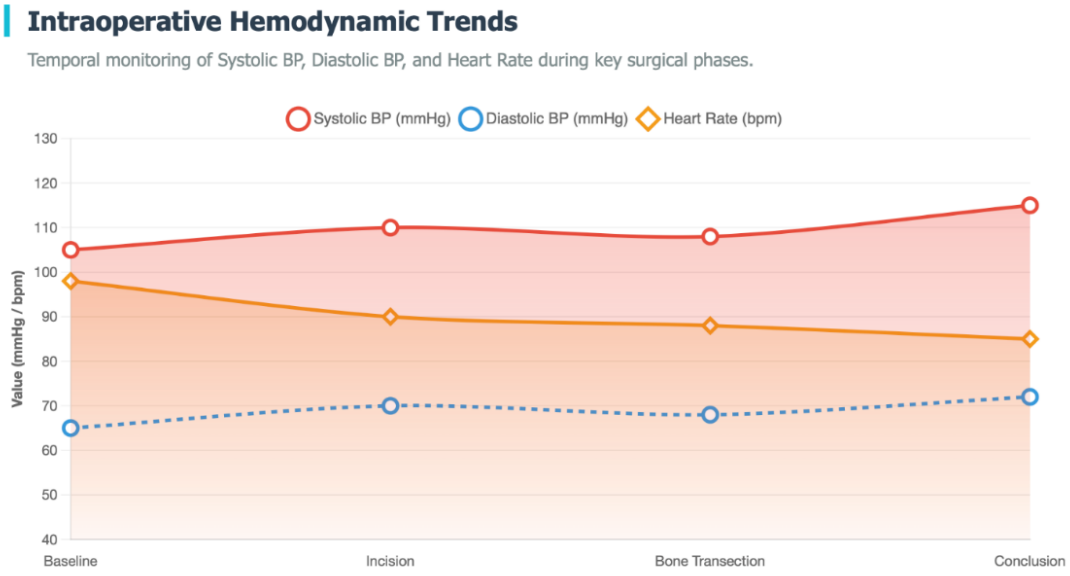
Biochemical analysis further illuminated the severity of the systemic insult. High-sensitivity Troponin I levels were markedly elevated at 4,500 ng/L, and NT-proBNP levels reached 8,400 pg/mL, signaling an acute exacerbation of heart failure and significant ongoing myocardial injury. Metabolic derangement was evident with elevated lactate levels of 4.2 mmol/L, consistent with metabolic acidosis secondary to the ischemic burden of the gangrenous limb. While coagulation profiles showed a prothrombin time of 12.5 seconds and an aPTT of 38 seconds, platelet function was clinically presumed to be profoundly inhibited due to the recent and active ingestion of clopidogrel.

Confronted with this complex clinical dyad of acute limb ischemia and acute cardiac failure, a multidisciplinary meeting involving anesthesiology, cardiology, and vascular surgery was convened to formulate an optimal surgical approach. The consensus was that general anesthesia posed an unacceptably high risk; the combination of an LVEF of 32%, stage C heart failure, and acute anterior STEMI meant that the administration of induction agents or volatile anesthetics could precipitate vasodilation and myocardial depression, leading to fatal cardiogenic shock. Conversely, neuraxial anesthesia was deemed absolutely contraindicated. The patient's ingestion of Clopidogrel and Aspirin that very morning violated safety guidelines, which typically mandate a 5 to 7-day washout period for Clopidogrel to avoid the catastrophic complication of a spinal epidural hematoma. Consequently, the team selected ultrasound-guided peripheral nerve block as the primary and sole anesthetic technique. Specifically, a combined femoral and popliteal sciatic nerve block was chosen to provide complete anesthesia for the below-knee amputation, effectively "threading the needle" between the hemodynamic risks of general anesthesia and the bleeding risks of neuraxial blockade.

The procedural execution was conducted with attention to the patient's limited physiological reserve. Standard ASA monitoring was applied, and invasive arterial pressure monitoring was established via the left radial artery to ensure beat-to-beat hemodynamic tracking. To minimize the risk of respiratory depression in this orthopneic patient, sedation was strictly limited to a minimal dose of Midazolam 0.5 mg intravenously. The Femoral Nerve Block was performed first via an inguinal approach with the patient supine. Using a high-frequency linear probe, the femoral nerve was identified lateral to the femoral artery, and 20 mL of 0.5% Ropivacaine combined with 2 mg Dexamethasone was injected under real-time ultrasound guidance, ensuring circumferential hydrodissection. Subsequently, the popliteal sciatic nerve block was administered using

the crosswise approach. This technique was specifically selected because the patient's severe orthopnea and acute heart failure precluded the prone positioning required for the classic posterior approach. With the leg slightly elevated on a pillow, the probe was placed transversely at the popliteal fossa, and the nerve bifurcation was identified. A volume of 25 mL of 0.5% Ropivacaine combined with 2 mg Dexamethasone was deposited within the paraneural sheath.

The intraoperative course validated the efficacy of this strategy (Figure 1). Complete sensory and motor blockade was achieved within 15 minutes, allowing the surgical team to proceed with a standard Below-Knee Amputation with a tourniquet time of 45 minutes. The patient's hemodynamics remained remarkably stable throughout the surgical stress: baseline blood pressure of 105/65 mmHg and heart rate of 98 bpm shifted minimally to 110/70 mmHg and 90 bpm at incision, and 108/68 mmHg and 88 bpm during bone transection, concluding at 115/72 mmHg and 85 bpm. Notably, no vasopressors were required at any stage. The patient remained awake, conversant, and comfortable, reporting a visual analogue scale (VAS) score of 0 out of 10 even during the critical phase of bone sawing. Following the procedure, which resulted in less than 200 mL of blood loss, the patient was transferred to the Intensive Care Unit. The block provided effective analgesia for 18 hours, managed subsequently with multimodal analgesia sparing opioids for the first 24 hours. Serial ECGs showed no extension of infarction, and the patient was discharged from the ICU on day 3, surviving to hospital discharge on day 10, demonstrating the life-saving utility of this tailored regional anesthetic approach.



Surgical Phase	Systolic BP (mmHg)	Diastolic BP (mmHg)	Heart Rate (bpm)
Baseline (T0)	105	65	98
Incision (T15)	110	70	90
Bone Transection (T45)	108	68	88
Conclusion (T90)	115	72	85

Figure 1. Intraoperative hemodynamic trends.

3. Discussion

This case report serves as a paradigmatic example of the high-stakes environment inherent in modern geriatric anesthesia, a field increasingly defined by the collision of advanced, multi-system pathology and acute surgical urgency. The patient described—a septuagenarian with a triple threat of acute anterior ST-elevation myocardial infarction (STEMI), severe left ventricular systolic dysfunction, and critical limb ischemia—represents a phenotype often described in the literature as the cardiac cripple. In such patients, the physiological margin for error is effectively non-existent. The successful management of this case via a combined ultrasound-guided femoral and crosswise popliteal sciatic nerve block does more than demonstrate a successful technique; it elucidates three critical physiological and methodological concepts that should guide the perioperative care of the high-risk vascular patient.[11]

The primary and most immediate challenge in this case was the patient's severely compromised cardiac reserve, quantified by a left ventricular ejection fraction (LVEF) of 32% and a history of failed coronary intervention. To understand the rationale for avoiding general anesthesia (GA), one must appreciate the precarious physiology of the failing ventricle. In a patient with severe systolic dysfunction and acute ischemia, the heart operates on the flat, depressed portion of the Frank-Starling curve. Cardiac output is fixed and critically dependent on the maintenance of both preload and afterload. Induction agents routinely used in general anesthesia, such as propofol or thiopental, along with volatile maintenance anesthetics like sevoflurane, are profound vasodilators.[12] They act to reduce systemic vascular resistance (SVR) and venous tone. In a healthy heart, a drop in SVR is compensated by a reflex increase in heart rate and contractility.[13] However, in this patient with a "fixed-output" state and active ischemia, such compensatory mechanisms are blunted or deleterious. A sudden reduction in SVR leads to a precipitous drop in diastolic blood pressure. Since the left ventricle is perfused primarily during diastole, this hypotension collapses the coronary perfusion pressure (CPP), creating a vicious cycle: hypotension leads to ischemia, ischemia worsens pump failure, and pump failure worsens hypotension, rapidly culminating in cardiovascular collapse or cardiac arrest.

Furthermore, the induction of general anesthesia invariably blunts the endogenous sympathetic tone (Figure 2). This patient's hemodynamic stability was likely maintained by high levels of circulating catecholamines, a compensatory stress response driving his residual cardiac output. Ablating this drive with anesthesia induction poses the risk of immediate hemodynamic decompensation. Neuraxial anesthesia, specifically spinal anesthesia, was also physiologically hazardous. The mechanism of spinal anesthesia involves a pharmacological sympathectomy from the level of the blockade (often T10 or higher for leg surgery) down to the sacral roots. This blockade results in profound venodilation and pooling of blood in the splanchnic and lower limb vascular beds, significantly reducing venous return (preload) to the heart. In a patient with ischemic cardiomyopathy and diastolic dysfunction, the left ventricle is stiff and volume-dependent; a sudden reduction in preload can result in a catastrophic drop in stroke volume that is unresponsive to inotropes.[14]

Our intraoperative data, showing the maintenance of mean arterial pressure within 10% of baseline without the use of vasopressors, confirms the physiological superiority of the peripheral nerve block (PNB) in this context. Unlike neuraxial techniques, PNB causes no central neuraxial blockade and no systemic sympathectomy. By blocking only the specific afferent somatic pain pathways of the lower extremity, the procedure effectively de-afferents the surgical site.

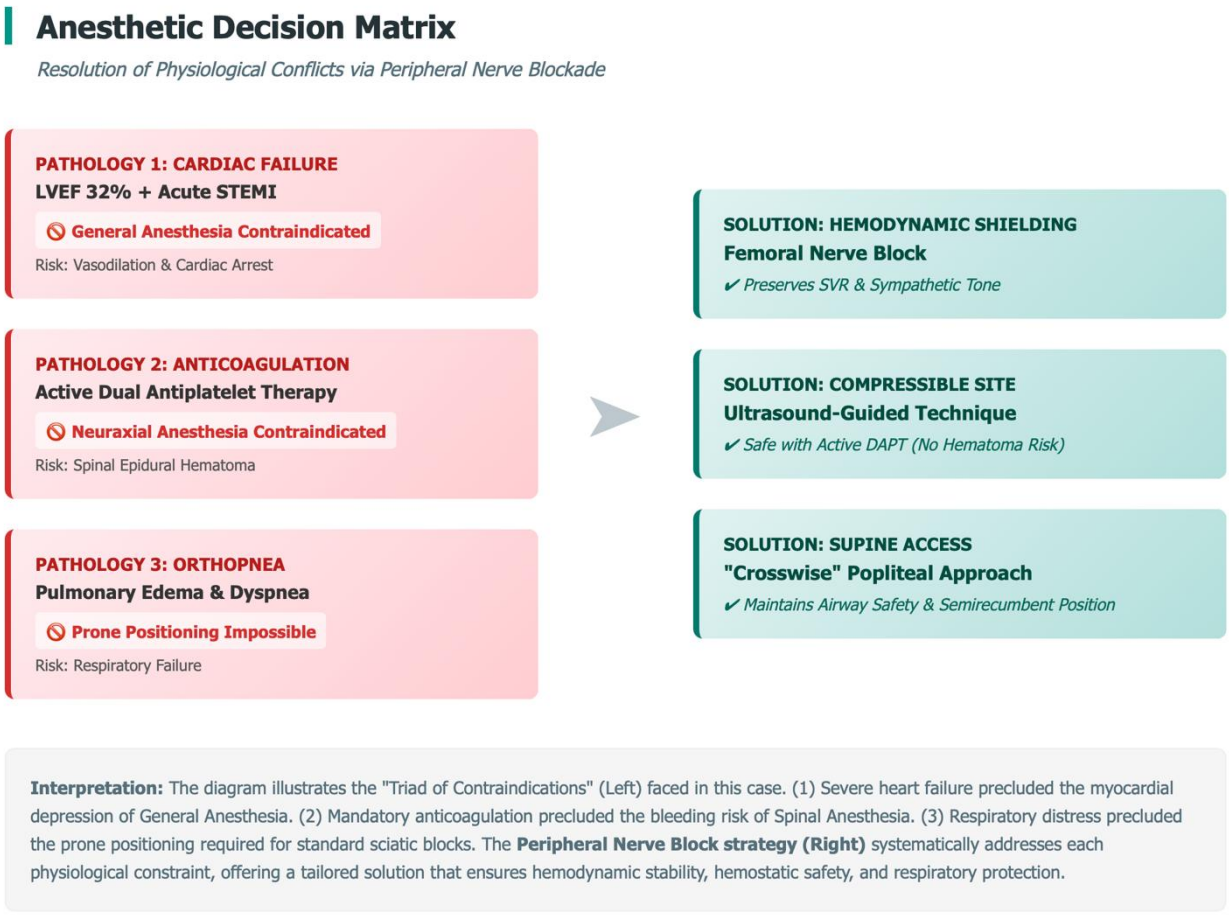


Figure 2. Anesthetic decision matrix.

This prevents the nociceptive input from reaching the central nervous system, thereby blocking the surgical stress response and the associated surge in cortisol and catecholamines, without inducing the dangerous vasodilation associated with spinal or general anesthesia. The heart is effectively shielded from the stress of surgery while maintaining its baseline hemodynamic equilibrium.[15]

The second critical challenge was the management of the patient's anticoagulation status, creating a conflict between the risk of surgical bleeding and the risk of thrombotic cardiovascular events. The patient was on active dual antiplatelet therapy (DAPT), consisting of aspirin and clopidogrel, a mandatory regimen following his diagnosis of acute coronary syndrome (ACS) and recent catheterization attempts. This presented a rigid contraindication to neuraxial anesthesia. Guidelines from the American Society of Regional Anesthesia and Pain Medicine (ASRA) are unequivocal: clopidogrel must be discontinued for a minimum of five to seven days prior to the performance of a spinal or epidural block.[16] This washout period allows for the restoration of adequate platelet function to prevent the formation of a spinal epidural hematoma (SEH). While rare, an SEH is a catastrophic complication; bleeding within the non-expandable spinal canal compresses the spinal cord, potentially leading to permanent paraplegia. However, the option of discontinuing DAPT to facilitate spinal anesthesia was clinically untenable. In a patient with an active

STEMI and complex coronary anatomy (failed PCI), the coronary vasculature is in a pro-thrombotic state. The premature cessation of antiplatelet therapy in this setting skyrockets the risk of acute stent thrombosis or recurrent myocardial infarction. The mortality associated with such thrombotic events far outweighs the benefits of neuraxial anesthesia.

The ultrasound-guided peripheral nerve block emerged as the only strategy capable of resolving this clinical deadlock. The safety of PNB in anticoagulated patients rests on the anatomical principle of compressibility. The femoral nerve is located in the inguinal region, and the sciatic nerve in the popliteal fossa. Unlike the neuraxial space, which is encased in bone and inaccessible to direct pressure, the femoral and popliteal regions are superficial and contain soft tissue.[17] In the unlikely event of accidental vascular puncture during the block, the anesthesiologist can apply direct manual pressure to the site to achieve hemostasis. Furthermore, the use of real-time ultrasound guidance significantly mitigates the risk of vascular injury by allowing for the direct visualization of the needle, the nerve, and the adjacent vessels. This anatomical advantage allowed us to maintain the patient on his life-saving antiplatelet regimen throughout the perioperative period, effectively decoupling the risk of neurological injury from the necessity of anticoagulation.

While the decision to use regional anesthesia was driven by physiology and pharmacology, the specific selection of the crosswise approach to the popliteal sciatic nerve block was driven by the imperatives of respiratory mechanics and airway safety. The classic approach to blocking the sciatic nerve in the popliteal fossa requires the patient to be positioned prone (face down). In a healthy patient, this is routine. However, in a geriatric patient with Stage C Heart Failure, acute pulmonary edema, and severe orthopnea, prone positioning is physiologically dangerous. Placing such a patient prone increases intrathoracic pressure, reduces chest wall compliance, and causes cephalad displacement of the diaphragm due to abdominal compression. This splinting of the diaphragm exacerbates ventilation-perfusion mismatch and can precipitate acute hypoxic respiratory failure. Furthermore, managing an airway emergency or performing cardiopulmonary resuscitation (CPR) is nearly impossible in the prone position.[18]

The crosswise approach, as utilized in this case, represents a critical technical refinement. It allows the anesthesiologist to access the sciatic nerve with the patient in the supine position, with the operative leg merely elevated and supported. This position preserves the patient's respiratory mechanics, allowing them to remain semi-recumbent to manage their orthopnea. It permits continuous, unobstructed monitoring of the airway, chest excursion, and 12-lead ECG, and ensures that the patient is in the optimal position for rapid airway intervention or resuscitation should a cardiac event occur. The success of this block relies on a thorough understanding of the cross-sectional anatomy of the popliteal fossa, where the sciatic nerve can be visualized via a transverse ultrasound view proximal to its bifurcation, ensuring a complete block of the distal lower extremity. This technical modification was not merely a matter of convenience; it was a safety maneuver that prevented the respiratory decompensation that prone positioning would have almost certainly triggered.[19]

The final pillar of our successful management strategy was the deliberate selection of the pharmacological agents: 0.5% Ropivacaine combined with Dexamethasone. The choice of Ropivacaine over the more traditionally used Bupivacaine was dictated by the patient's cardiac fragility.[20] Local anesthetics work by blocking sodium channels, but they can also block cardiac sodium channels if systemic absorption occurs, leading to re-entrant arrhythmias and cardiovascular collapse. Bupivacaine is notorious for its high affinity for cardiac sodium channels and its slow dissociation rate, making bupivacaine-induced cardiotoxicity difficult to resuscitate. Ropivacaine, as a

pure S-enantiomer, possesses lower lipid solubility and a significantly faster dissociation rate from cardiac sodium channels. In a patient with pre-existing conduction abnormalities (Q waves, STEMI) and active ischemia, the margin for cardiotoxicity is reduced. Ropivacaine offers a wider therapeutic safety index, ensuring that even in the event of minor systemic absorption, the risk of inducing a fatal arrhythmia is minimized.

The addition of Dexamethasone as a perineural adjuvant served a dual purpose. First, it significantly prolongs the duration of the sensory block, extending analgesia well into the postoperative period—in this case, up to 18 hours. Mechanistically, perineural corticosteroids are thought to inhibit the transmission of nociceptive C-fibers and reduce local release of inflammatory mediators. Second, this prolonged regional analgesia provided a crucial opioid-sparing effect. Postoperative opioids, such as morphine or fentanyl, are potent respiratory depressants. In a patient with heart failure, opioid-induced hypoventilation leads to hypercapnia and hypoxia. Hypoxia causes pulmonary vasoconstriction, which increases right ventricular afterload and can precipitate right heart failure. By eliminating the need for opioids in the first 24 hours, the peripheral nerve block prevented this cascade, facilitating a smooth recovery and protecting the right ventricle from afterload stress.[17,18]

While this case report illustrates a promising paradigm for managing the cardiac cripple, it is subject to the inherent limitations of single-patient anecdotal evidence. The successful outcome observed—specifically the preservation of hemodynamic stability and the avoidance of airway manipulation—cannot be statistically generalized to the broader population of geriatric vascular patients without further validation. Additionally, the utility of the crosswise popliteal approach is intrinsically operator-dependent; it requires a high degree of proficiency in ultrasound-guided regional anesthesia to ensure precise nerve localization in the supine position without compromising adjacent vascular structures. Consequently, the reproducibility of these results may vary across institutions based on the availability of advanced regional anesthesia expertise and ultrasound technology.

Future research efforts must pivot from isolated case reports to robust, multi-center prospective cohort studies or randomized controlled trials comparing peripheral nerve blockade against general and neuraxial anesthesia in patients with severe left ventricular dysfunction (LVEF < 35%). Specifically, future investigations should focus on quantifying the incidence of perioperative major adverse cardiac events (MACE) and 30-day mortality rates to definitively establish the superiority of regional techniques in this high-risk demographic. Furthermore, longitudinal studies are warranted to evaluate whether the profound opioid-sparing benefits of this approach translate into a reduced incidence of chronic phantom limb pain and improved long-term functional rehabilitation. Only through such rigorous inquiry can this technique be elevated from a novel rescue strategy to a standardized clinical guideline for the high-risk vascular patient.[19,20]

4. Conclusion

The case presented herein underscores a pivotal evolution in the management of the high-risk surgical patient. We navigated a complex clinical scenario characterized by acute anterior STEMI, failed coronary intervention, severe left ventricular dysfunction, and critical limb ischemia—a combination that rendered standard anesthetic modalities potentially lethal. The management of emergency amputation in such patients requires an anesthetic strategy that prioritizes hemodynamic preservation above all other considerations. This case demonstrates that the ultrasound-guided femoral and crosswise popliteal sciatic nerve block provides a safe, effective, and physiologically superior alternative to general or

neuraxial anesthesia.

By utilizing this regional technique, we achieved three distinct clinical victories. First, we avoided the myocardial depression and vasodilatory collapse associated with general anesthesia, maintaining the patient's fragile perfusion pressure. Second, we successfully navigated the constraints of dual antiplatelet therapy, utilizing a compressible site for anesthesia to bypass the risks of spinal hematoma and stent thrombosis. Third, by employing the crosswise approach, we accommodated the patient's respiratory pathology, avoiding the hazards of prone positioning. This approach effectively converts a high-mortality systemic crisis into a managed, focal regional procedure. It respects the limited physiological reserve of the cardiac cripple and aligns with the principles of enhanced recovery by minimizing opioid consumption and systemic stress. As the population of geriatric patients with complex cardiovascular comorbidities requiring vascular intervention continues to grow, we advocate for the adoption of combined peripheral nerve blockade as the standard of care for lower limb amputation in this high-risk cohort. Future clinical practice guidelines should reflect this shift, positioning peripheral nerve blockade not merely as an alternative, but as the primary anesthetic modality for the critical cardiac patient facing emergency limb salvage.

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