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Ultrasound-Guided Combined Femoral and Popliteal-Sciatic Nerve Block for Foot Debridement in a Patient with Peripheral Arterial Disease, Heart Failure with Reduced Ejection Fraction, and Pulmonary Oedema: A High-Risk Case Report

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A B S T R A C T

Peripheral arterial disease (PAD) in its advanced stage leads to critical limb ischaemia requiring operative debridement, and coexisting heart failure with reduced ejection fraction (HFrEF) and pulmonary oedema make general and neuraxial anaesthesia hazardous. We describe a 59-year-old man with three-vessel coronary artery disease previously treated by multiple percutaneous coronary interventions, HFrEF (ejection fraction 36%), hypertension, diabetes mellitus and bilateral PAD who presented with acute decompensated heart failure, pulmonary oedema and bilateral pleural effusion together with an extensive left foot ulcer requiring urgent debridement and necrotomy. An ultrasound-guided combined femoral and popliteal-sciatic nerve block was performed using 20 mL of 1.5% lidocaine and 20 mL of 0.25% bupivacaine. The patient remained conscious and haemodynamically stable with systolic blood pressure 90–110 mmHg, heart rate ~85 beats/min and SpO_2 100%. The 60-minute procedure was uneventful with no local anaesthetic toxicity, new neurological deficit or respiratory compromise, and the patient was transferred to the high-care unit. Ultrasound-guided peripheral nerve blockade represents a cardiopulmonary-sparing anaesthetic strategy that can deliver adequate operating conditions for lower-extremity surgery in patients with PAD and severe cardiac and respiratory comorbidity. Keywords: Peripheral nerve block, Peripheral arterial disease, Heart failure with reduced ejection fraction, regional anesthesia, ultrasound guidance.

1. Introduction

Peripheral arterial disease (PAD) is a chronic atherosclerotic disorder that reduces tissue perfusion in the extremities and, in its advanced stages, produces rest pain, non-healing ulcers and tissue necrosis, collectively described as chronic limb-threatening ischaemia (CLTI).[1,2] Global epidemiological analyses estimate that PAD affects more than 230 million adults worldwide and that its prevalence is growing more rapidly in low- and middle-income countries than in high-income settings, reflecting both the aging of populations and the increasing burden of diabetes mellitus, hypertension and smoking.[2,3] The Asian region in particular, has seen a steep increase in PAD-related hospitalisations over the past two decades.[3] Unlike the often silent nature of early PAD, CLTI mandates urgent surgical attention: debridement, necrotomy and, in selected cases, revascularisation or amputation are required to arrest the progression of infection, preserve limb function and improve survival.[1,4,5] A substantial proportion of CLTI patients present with diffuse multi-

vessel atherosclerosis and concomitant coronary and cerebrovascular disease, contributing to a 30-day mortality after lower-extremity amputation that has been reported between 5% and 15% in contemporary registries.[4,6]

The anaesthetic management of patients presenting for lower-extremity debridement in the setting of CLTI is complicated by the dense burden of cardiovascular comorbidity typically accompanying the disease. Heart failure with reduced ejection fraction (HFrEF), multivessel coronary artery disease with previous myocardial infarction or percutaneous coronary intervention, hypertension, diabetes mellitus, chronic kidney disease, and pulmonary disorders such as pneumonia, pleural effusion and pulmonary oedema are each common and interactively hazardous.[4,6-9] General anaesthesia in this context requires airway instrumentation, positive-pressure ventilation and administration of intravenous induction agents and volatile anaesthetics that can produce myocardial depression, reductions in systemic vascular resistance, and impairment of ventilation–perfusion matching.[6,9,10] Neuraxial anaesthesia, while avoiding airway manipulation, produces sympathetic blockade of the T10–L2 segments and can precipitate severe hypotension when ejection fraction is already compromised, particularly in the presence of fixed cardiac output lesions or chronic diuretic therapy.[11-13] In patients with multivessel CAD, sudden hypotension can precipitate myocardial ischaemia, while sudden increases in sympathetic tone during intubation can raise myocardial oxygen demand.[6,9,12]

Peripheral nerve blockade (PNB) has emerged as a cardiopulmonary-sparing alternative for lower-extremity surgery in this high-risk population.[11,13-17] By targeting specific nerves—most commonly the femoral nerve in combination with the sciatic nerve at the popliteal level for foot and ankle surgery—PNB provides selective sensory and motor blockade of the operative field without extensive sympathectomy.[11,18,19] Modern practice relies on ultrasound guidance, which significantly improves block success, shortens onset time, reduces local anaesthetic dose and decreases the risk of systemic toxicity.[14,18,20,21] Patient-centred advantages include the preservation of spontaneous ventilation and airway reflexes, a reduced requirement for systemic opioids, improved postoperative analgesia and lower rates of postoperative nausea and vomiting.[11,13,22] For cardiac patients specifically, contemporary consensus statements emphasise that PNB can avoid many of the adverse cardiovascular consequences of general and neuraxial techniques.[9,11,13]

Despite these theoretical and clinical advantages, there is a paucity of detailed case-based literature describing the practical execution and perioperative course of ultrasound-guided femoral plus popliteal-sciatic block in patients whose comorbid profile includes concurrent decompensated HFrEF, pulmonary oedema, bilateral pleural effusions and recent coronary revascularisation. Most published literature either addresses PNB for elective orthopedic procedures in relatively healthy patients or focuses on amputation as opposed to debridement and limb-salvage surgery.[11,16,17] Case reports specifically describing CLTI-related foot debridement under combined femoral–popliteal block in patients with ejection fractions below 40% are relatively uncommon, particularly in South-East Asian populations, and consolidated descriptions of hemodynamic and respiratory trajectories during such procedures remain limited.[15,16,19]

The present report aims to address that gap. We describe in detail the clinical presentation, perioperative assessment, intraoperative anaesthetic technique, hemodynamic and respiratory monitoring and early postoperative outcome of a 59-year-old man with bilateral PAD, HFrEF (EF 36%), three-vessel coronary artery disease, pulmonary oedema and bilateral pleural effusion who underwent debridement and necrotomy of the left foot under an ultrasound-guided combined femoral and popliteal-sciatic nerve block. The novelty of the report lies in (i) integrating in a single practical narrative the multimodal imaging, laboratory, ultrasound and intraoperative data that

together define a contemporary cardiopulmonary-sparing anaesthetic approach; (ii) providing a replicable, step-by-step description of block technique and local anaesthetic dosing suitable for adoption in resource-limited operating rooms in Indonesia and similar settings; and (iii) analysing the intraoperative hemodynamic profile against the backdrop of current heart-failure and perioperative guidelines. The study aim is therefore to demonstrate the feasibility, safety and hemodynamic sparing effect of a carefully executed ultrasound-guided femoral plus popliteal-sciatic block in a highly comorbid patient and to situate the observations within the evolving literature on regional anaesthesia in the cardiopulmonary-compromised patient.

2. Case Presentation

Written informed consent for the described anaesthetic technique, surgical procedure and anonymised publication of clinical details and images was obtained from the patient. Institutional ethics review was waived in accordance with local policy for single-case reports. Consent for publication was obtained from the patient.

Table 1 presents the demographic, clinical and comorbidity profile of the patient at the time of emergency presentation. A 59-year-old man of Javanese descent, a retired civil servant, BMI 27.8 kg/m², was transferred from a peripheral clinic to the emergency department of Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, with a chief complaint of progressive dyspnoea that had begun approximately twenty-four hours before arrival and had worsened through the early morning hours. He described the dyspnoea as persistent at rest, aggravated by lying supine, and partially relieved by sitting upright. Several episodes of paroxysmal nocturnal dyspnoea during the preceding week were also reported. Associated symptoms included an occasional non-productive cough, generalised weakness and bilateral lower-extremity swelling. In addition to these cardiorespiratory complaints, the patient reported painful, progressive blackening of the first and second digits of the left foot over the preceding ten days, with superimposed malodorous discharge from an enlarging ulcer on the plantar surface of the same foot. He denied fever, chest pain, palpitations, haemoptysis, orthopnoea unrelated to dyspnoea, or intermittent claudication pain of the contralateral limb.

The patient's past medical history was substantial. He had a thirty-year history of essential hypertension treated with a combination of amlodipine and ramipril, a fifteen-year history of type 2 diabetes mellitus managed with metformin and subcutaneous basal insulin, and extensive coronary atherosclerosis documented six years earlier as three-vessel disease, for which he had undergone multiple percutaneous coronary interventions with drug-eluting stent placement. Subsequent echocardiography had demonstrated a reduced left ventricular ejection fraction of 36%, meeting contemporary criteria for HFrEF; guideline-directed medical therapy included bisoprolol, ramipril, spironolactone and dapagliflozin, with adherence described as intermittent. Twelve months before the current admission, he had been diagnosed with bilateral PAD of the cruris after the development of ischaemic changes in the right foot, which had ultimately required amputation of the first and second digits of the right foot; an amputation of the second digit of the left foot had also been performed at that time. He was a former smoker (40 pack-years, discontinued eight years prior to admission) and denied significant alcohol intake.

On presentation, the patient was alert and oriented but mildly distressed by dyspnoea. Vital signs revealed a blood pressure of 138/77 mmHg, a heart rate of 96 beats/min, a respiratory rate of 20 breaths/min, tympanic temperature of 36.7°C and oxygen saturation of 98% on room air in the sitting position. Auscultation of the lungs demonstrated coarse crackles throughout both lower zones and fine crackles extending into the mid zones, consistent with pulmonary oedema and possible pneumonia.

Table 1. Demographic, clinical, and comorbidity characteristics of the patient.

Characteristic	Value/Description
Age	59 years
Gender	Male
Body mass index	27.8 kg/m² (grade I obesity)
Ethnicity/occupation	Javanese; retired civil servant
Chief complaint	Progressive dyspnoea (<24 h) worsening on the morning of admission
Associated symptoms	Orthopnoea, paroxysmal nocturnal dyspnoea, occasional cough, generalised weakness, bilateral lower-extremity oedema, painful blackening of the first and second digits of the left foot*
Hypertension	Treated with amlodipine + ramipril (30-year history)
Diabetes mellitus type 2	15-year history; metformin + basal insulin; HbA1c 7.4%
Coronary artery disease	Three-vessel; multiple PCIs with drug-eluting stents
Heart failure with reduced ejection fraction (HFrEF)	EF 36% on prior echocardiography; GDMT: bisoprolol, ramipril, spironolactone, dapagliflozin
Peripheral arterial disease	Bilateral; prior amputation right 1st/2nd digits and left 2nd digit (1 year ago)
Smoking	Former (40 pack-years, quit 8 years ago)
Vital signs on admission	BP 138/77 mmHg; HR 96/min; RR 20/min; T 36.7°C; SpO_2 98% RA (sitting)
Pulmonary examination	Bilateral coarse and fine crackles (lower→mid zones)
Cardiac examination	Regular rhythm; S3 gallop; grade 3/6 pansystolic murmur at LLSB†
Peripheral pulses (left / right)	Femoral diminished bilaterally; popliteal diminished bilaterally; dorsalis pedis: weak / non-palpable; posterior tibial: weak / non-palpable
Left foot toe SpO_2 (I–V)	96 / — / 98 / 99 / 97 (%)
Revised cardiac risk index	3 points (high-risk)
ASA physical status	III

* Clinical findings visible in Figure 1. † LLSB = left lower sternal border. BP = blood pressure; HR = heart rate; RR = respiratory rate; GDMT = guideline-directed medical therapy; PCI = percutaneous coronary intervention.

Cardiac examination revealed a regular rhythm, an audible third heart sound (S3 gallop), and a grade 3/6 pansystolic murmur at the left lower sternal border, suggestive of secondary tricuspid or functional mitral regurgitation. There was bilateral pitting oedema of the lower extremities. Inspection of the left foot disclosed an extensive ulcer extending from the plantar surface toward the medial aspect of the foot, with irregular

margins, and a wound base consisting of black necrotic tissue, yellow slough and partial granulation; clear demarcation of distal necrosis was present at the first digit. Peripheral arterial pulses were markedly diminished bilaterally at the femoral and popliteal sites. The dorsalis pedis pulse was non-palpable on the right and weak on the left, while the posterior tibial pulse was non-palpable on the right and weak on the left. Toe oxygen saturation measurements on the left foot were recorded as 96/–/98/99/97 for the first through fifth digits, respectively. The neurological examination of the lower extremities showed diminished light touch and vibration sensation bilaterally. As shown in Table 1, his American Society of Anesthesiologists (ASA) physical status was classified as III, with an estimated Revised Cardiac Risk Index of 3 points. Figure 1 illustrates the clinical appearance of the left foot on admission, including the extensive plantar ulcer and the necrosis of the first digit.

Laboratory findings, which are summarised in Table 2, revealed a haemoglobin concentration of 11 g/dL, a leukocyte count of 8.4 × 10^3/μL with a normal differential, and a platelet count of 218 × 10^3/μL. Mild renal impairment was present with serum urea of 91 mg/dL and creatinine of 1.4 mg/dL, corresponding to an estimated glomerular filtration rate of approximately 52 mL/min/1.73 m^2, consistent with chronic kidney disease stage 3a. Fasting blood glucose was 112 mg/dL and glycated haemoglobin was 7.4%, indicating reasonable recent glycaemic control. Serum electrolytes and liver enzymes were within reference ranges. C-reactive protein was mildly elevated at 18 mg/L, suggestive of low-grade systemic inflammation related to the necrotic foot rather than overt sepsis. Serum lactate was 1.3 mmol/L. As demonstrated in Table 2, the composite laboratory pattern was interpreted as mild anaemia of chronic disease, moderate renal impairment, stable glycaemic control and low-level inflammation.

Twelve-lead electrocardiography demonstrated sinus rhythm at a rate of 100 beats/min, left axis deviation, and left ventricular hypertrophy fulfilling the Peguero–Lo Presti voltage criteria; there were no acute ischaemic repolarisation changes. Chest radiography revealed bilateral perihilar alveolar opacities consistent with pulmonary oedema and bilateral small-to-moderate pleural effusions. Thoracic ultrasonography confirmed bilateral B-line pattern and confluent pleural effusions measuring approximately 3 cm on the right and 4 cm on the left at the posterior axillary line. Bedside transthoracic echocardiography performed by the anaesthesia team documented left ventricular ejection fraction of 34% (Simpson biplane), mild left atrial enlargement, global hypokinesis with relative preservation of basal segments, grade II diastolic dysfunction, moderate tricuspid regurgitation with estimated systolic pulmonary artery pressure of 48 mmHg, and no pericardial effusion. Imaging findings are also summarised in Table 2.

A multidisciplinary discussion involving the anaesthesia, cardiology and vascular surgery teams concluded that urgent surgical debridement and necrotomy of the left foot were indicated to control the necrotising soft-tissue process and to prevent the progression of sepsis, which would otherwise likely precipitate further decompensation. Given the concurrent acute decompensated heart failure, pulmonary oedema, bilateral pleural effusion, reduced left ventricular ejection fraction, three-vessel CAD, and baseline mild renal dysfunction, the patient was considered to be at particularly high risk for perioperative cardiovascular and respiratory complications under general anaesthesia. A plan was therefore made to proceed with ultrasound-guided combined femoral and popliteal-sciatic nerve block as the primary anaesthetic technique, supplemented by low-dose intravenous analgesia as needed, with general anaesthesia reserved as a rescue in case of block failure or hemodynamic instability. Following informed consent, the patient received intravenous furosemide 40 mg prior to transfer to the operating theatre for further decongestion and was positioned with the trunk elevated to 30°.

Table 2. Laboratory and imaging investigations with interpretation.

Investigation	Patient value	Reference range	Interpretation
Haemoglobin	11.0 g/dL	13.5–17.5 g/dL	Mild anaemia of chronic disease
Leukocyte count	8.4 × 10^3/μL	4.0–10.0 × 10^3/μL	Normal
Platelet count	218 × 10^3/μL	150–400 × 10^3/μL	Normal
Urea	91 mg/dL	7–20 mg/dL	Elevated; suggests impaired clearance
Creatinine/eGFR	1.4 mg/dL / 52 mL/min/1.73 m^2	0.7–1.3 / ≥ 60	CKD stage 3a
Fasting blood glucose	112 mg/dL	70–99 mg/dL	Mildly elevated (on therapy)
HbA1c	7.4%	< 7.0%	Marginal glycaemic control
C-reactive protein	18 mg/L	< 5 mg/L	Low-grade inflammation
Serum lactate	1.3 mmol/L	0.5–2.2 mmol/L	Normal
Electrolytes (Na/K)	138 / 4.2 mmol/L	135–145 / 3.5–5.0	Normal
Electrocardiography	Sinus tachycardia 100/min, LAD, LVH (Peguero–Lo Presti)	—	Chronic structural heart disease
Chest radiography	Bilateral perihilar alveolar opacities + bilateral pleural effusions	—	Acute pulmonary oedema
Thoracic ultrasound	Diffuse B-lines; pleural effusions 3 cm R, 4 cm L	—	Confirms congestion
Bedside echocardiography	LVEF 34% (Simpson biplane); grade II diastolic dysfunction; moderate TR; SPAP 48 mmHg	LVEF ≥ 52%	HFrEF with pulmonary hypertension

Abnormal values are shown in bold red. CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; LAD = left axis deviation; LVH = left ventricular hypertrophy; LVEF = left ventricular ejection fraction; SPAP = systolic pulmonary artery pressure; TR = tricuspid regurgitation.

Table 3 outlines the step-by-step anaesthetic management, including equipment, drug doses, monitoring and intraoperative response. Standard ASA monitors were applied on arrival in the operating theatre, including continuous five-lead electrocardiography with ST-segment analysis, pulse

oximetry, end-tidal CO_2 via nasal cannula, non-invasive blood pressure measurement every three minutes, and urinary output monitoring via a Foley catheter. Supplemental oxygen was delivered by nasal cannula at 3 L/min to target an oxygen saturation above 96%. Aseptic skin preparation with 2% chlorhexidine in 70% alcohol and sterile draping were performed. A high-frequency linear probe (6–13 MHz) covered by a sterile sleeve was used for both blocks, and a 22-gauge, 50-mm short-bevel insulated nerve block needle was selected. The local anaesthetic mixture consisted of 20 mL of 1.5% lidocaine and 20 mL of 0.25% bupivacaine, drawn up in separate syringes, with total dosing carefully kept within the recommended safe thresholds for body weight.

The femoral nerve block was performed first. With the patient supine and the left lower limb slightly externally rotated, the probe was placed in a transverse orientation over the inguinal crease to identify the femoral artery, the femoral vein medially, and the femoral nerve as a hyperechoic, flattened triangular structure lateral to the artery and superficial to the iliopsoas muscle. The needle was advanced in-plane from lateral to medial at an angle of approximately 45°, traversing the fascia iliaca. After negative aspiration for blood, 10 mL of 1.5% lidocaine and 10 mL of 0.25% bupivacaine were injected incrementally in 5-mL aliquots, confirming circumferential spread around the nerve without intraneural injection. The patient was then repositioned to the lateral decubitus position with the operated limb uppermost, and the popliteal-sciatic block was performed. The probe was placed in the popliteal fossa with transverse orientation to identify the popliteal artery and its accompanying popliteal vein, and the tibial and common peroneal branches of the sciatic nerve were traced proximally until they converged into a common trunk. At this level, the needle was inserted in-plane from lateral to medial, and after negative aspiration, the remaining 10 mL of lidocaine and 10 mL of bupivacaine were injected incrementally, producing perineural spread in a donut-shaped pattern around the sciatic trunk. Sonographic images confirmed circumferential perineural local anaesthetic spread around both the femoral nerve in the inguinal region and the common sciatic trunk in the popliteal fossa.

Sensory and motor block testing at 15 minutes after injection confirmed a dense sensory block in the distributions of the saphenous nerve (medial leg and foot), the posterior tibial nerve (plantar and heel) and the superficial peroneal nerve (dorsum of foot), as well as motor weakness of knee extension (femoral component) and plantarflexion/dorsiflexion (sciatic component), giving a modified Bromage score of 3. Surgical incision was made thereafter (Figure 1). Intraoperatively, systolic blood pressure was maintained between 90 and 110 mmHg and diastolic between 30 and 60 mmHg; heart rate remained at approximately 85 beats/min in regular rhythm, and oxygen saturation stayed at 100% on supplemental oxygen. No new ST-segment changes were observed. The intraoperative hemodynamic parameters—summarised in Table 3—remained within the preoperative baseline range throughout the procedure. The patient remained awake and cooperative throughout the procedure, conversing comfortably with the anaesthesia team. Adjuvant intraoperative medications included ondansetron 4 mg intravenously and paracetamol 1 g intravenously. No intravenous vasopressors, sedatives or opioid boluses were required. Surgical debridement proceeded uneventfully with resection of the necrotic first toe, removal of slough and devitalised subcutaneous tissue from the plantar ulcer, and thorough wound irrigation, and the operation was completed in approximately 60 minutes. No signs of local anaesthetic systemic toxicity, intraoperative arrhythmia, hemodynamic instability, respiratory compromise or neurological deficit related to the nerve block were observed.

Postoperatively, the patient was transferred to the high-care unit for intensive haemodynamic monitoring and continued decongestive therapy. The postoperative course was uncomplicated; no further deterioration of cardiac or respiratory function occurred, and progressive resolution of pulmonary

oedema was observed over the following 48 hours with continued diuresis and optimisation of guideline-directed medical therapy. On the second postoperative day, repeat chest radiography demonstrated reduced pulmonary congestion and a persistent but diminished bilateral pleural effusion. Wound healing progressed with daily dressing changes and early vascular-surgery review for future revascularisation planning. The patient was discharged on day seven in clinically improved condition, with further scheduled outpatient review by the anaesthesia, cardiology, and vascular surgery services.

Table 3. Perioperative anaesthetic protocol and intraoperative response.

Phase	Intervention/drug	Dose/setting	Response
Pre-operative	Intravenous furosemide for decongestion; trunk elevation 30°	Furosemide 40 mg IV	Reduction of pulmonary congestion
Monitoring	5-lead ECG with ST analysis, SpO_2, nasal-cannula end-tidal CO_2, NIBP q3min, urinary catheter	Continuous	Baseline values recorded
Supplemental oxygen	Nasal cannula	3 L/min	SpO_2 100%
Femoral nerve block	Ultrasound-guided, in-plane, fascia-iliaca approach	10 mL lidocaine 1.5% + 10 mL bupivacaine 0.25%	Circumferential perineural spread; dense sensory/motor block at 15 min
Sciatic (popliteal) block	Ultrasound-guided, in-plane, lateral decubitus	10 mL lidocaine 1.5% + 10 mL bupivacaine 0.25%	Donut-shaped perineural spread; complete foot anaesthesia
Total local anaesthetic dose	Lidocaine 300 mg + bupivacaine 50 mg	Within safe thresholds for 70 kg	No LAST symptoms
Adjuvant intra-operative drugs	Ondansetron + paracetamol	4 mg + 1 g IV	Nil vomiting; adequate analgesia
Intra-operative SBP / DBP	Non-invasive cuff	90–110 / 30–60 mmHg	Stable
Intra-operative HR	5-lead ECG	~ 85 beats/min, regular	Stable, no ischaemic ST changes
Intra-operative SpO_2	Pulse oximetry	100% on supplemental O_2	Stable; spontaneous breathing preserved
Surgery	Debridement and necrotomy of the left foot	Duration ~ 60 min	Resection of necrotic 1st toe; cleaning of plantar ulcer; no major bleeding
Post-operative disposition	High-care unit admission for continued decongestion and monitoring	—	Uneventful; diuresis continued

DBP = diastolic blood pressure; HR = heart rate; LAST = local anaesthetic systemic toxicity; NIBP = non-invasive blood pressure; SBP = systolic blood pressure.

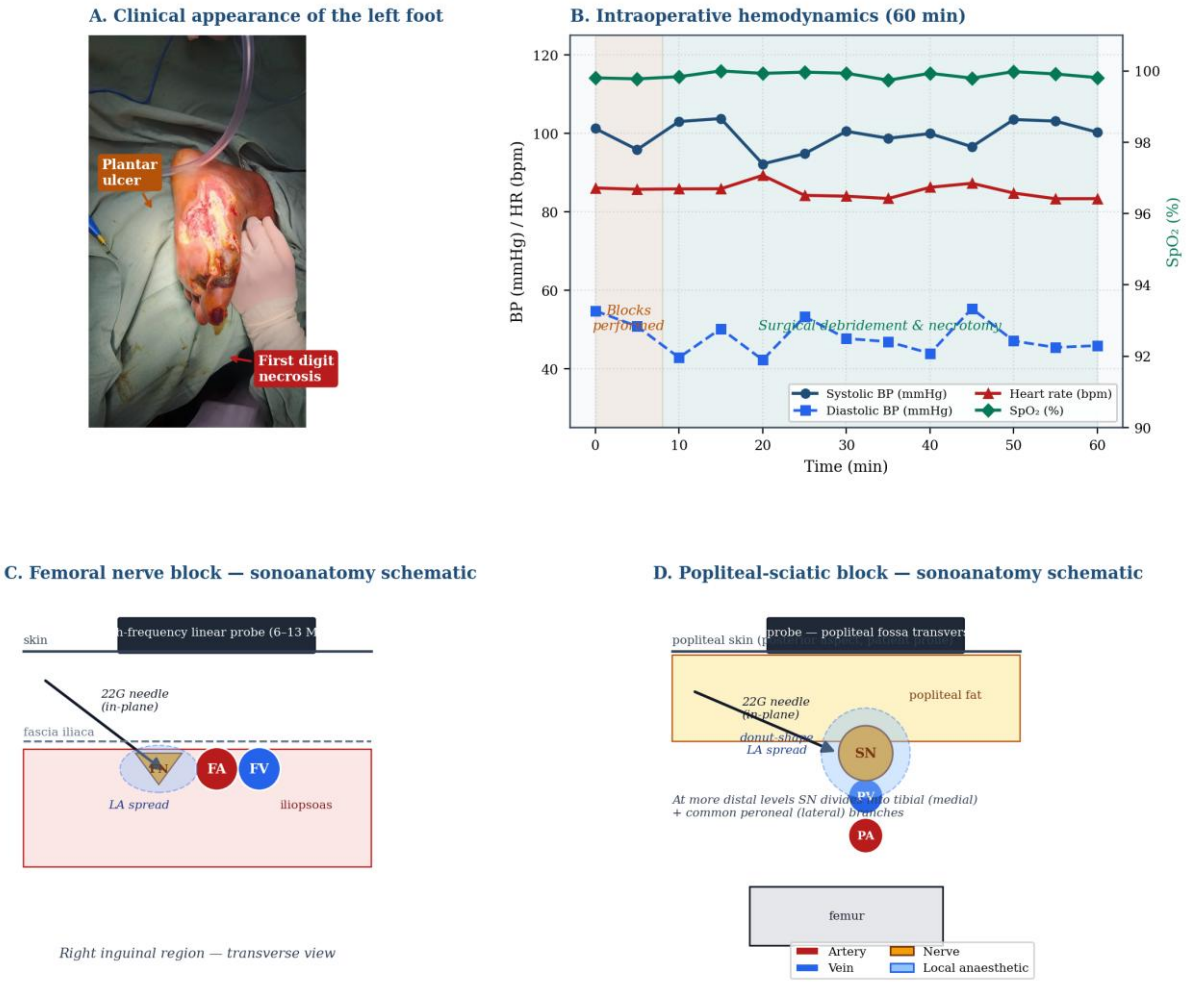

Figure 1. Integrated perioperative summary of the case. (A) Clinical appearance of the left foot on admission showing the plantar ulcer with a black necrotic base and the demarcated distal necrosis of the first digit. (B) Intraoperative hemodynamic and respiratory trend across the 60-minute procedure (minute-by-minute systolic and diastolic blood pressure, heart rate, and peripheral oxygen saturation) demonstrating stability without the need for vasoactive support. (C) Sonoanatomy schematic of the femoral nerve block — transverse view at the inguinal region, with the femoral nerve (FN) located lateral to the femoral artery (FA) and superficial to the iliopsoas, and the 22-gauge needle advanced in-plane from lateral to medial to achieve circumferential perineural spread. (D) Sonoanatomy schematic of the popliteal-sciatic block — transverse view at the popliteal fossa with the sciatic nerve (SN) superficial and lateral to the popliteal artery (PA) and vein (PV), and the needle advanced in-plane to produce a donut-shape local anaesthetic spread around the common sciatic trunk.

3. Discussion

The present case highlights the decisive role of anaesthetic strategy in determining perioperative outcome in patients whose cardiopulmonary reserve is severely limited by chronic multisystem disease. Our patient's clinical composite—three-vessel CAD with previous PCI, HFrEF with an ejection fraction of 34–36%, acute decompensated heart failure with pulmonary oedema and bilateral pleural effusions, chronic kidney disease stage 3a, diabetes mellitus, and longstanding bilateral PAD requiring foot debridement—represents one of the most challenging scenarios confronted in contemporary anaesthetic practice.[4,6-9] In such patients, the hemodynamic

stability provided by a carefully executed regional anaesthetic may be pivotal in avoiding the cascade of perioperative myocardial injury, pulmonary decompensation and renal dysfunction that otherwise follows general anaesthesia or neuraxial blockade.[11,13-16]

Peripheral arterial disease is a systemic vascular disease whose local consequence—CLTI—often brings patients into contact with the operating theatre for debridement or amputation. Epidemiologic data indicate that the global prevalence of PAD in adults exceeds 10% after the age of 65 years, and that more than 80% of patients with PAD have concurrent coronary or cerebrovascular disease.[2,3] In Asia, recent modeling studies estimate that the absolute number of individuals affected by PAD has doubled in the past two decades, driven by aging populations and a steep rise in diabetes mellitus.[3] The 30-day mortality after major lower-extremity amputation in PAD patients has remained disappointingly high in contemporary registries, and cardiovascular events are the dominant cause.[4-6] These observations underscore the urgency of designing anaesthetic strategies that protect the heart and lungs during inevitable surgical interventions. For the present patient, the decision to avoid general anaesthesia was anchored in three distinct pathophysiologic concerns: myocardial depression, hemodynamic fluctuations associated with sympathetic swings, and ventilatory impairment, each of which could have precipitated decompensation in his fragile cardiopulmonary state.

From a physiologic standpoint, general anaesthesia induces several interacting disturbances in patients with severely reduced left ventricular systolic function. Intravenous induction agents such as propofol reduce systemic vascular resistance by both direct vasodilation and attenuation of sympathetic tone, with the corresponding effect on cardiac output depending on the balance between reduced afterload and reduced preload.[6,9] Etomidate, though relatively hemodynamically neutral, is associated with adrenal suppression and with limited experience in HFrEF-rich populations. Volatile anaesthetics additionally impair myocardial contractility in a dose-dependent fashion and can exacerbate diastolic dysfunction.[9,10] In patients with severe pulmonary oedema and pleural effusion, the conversion from spontaneous ventilation to positive-pressure ventilation increases intrathoracic pressure, reduces venous return to the heart, and alters ventilation–perfusion relationships in alveoli already partially collapsed by effusion and interstitial congestion.[10] Furthermore, airway instrumentation during induction triggers sympathetic surges that raise myocardial oxygen demand precisely when coronary perfusion pressure is vulnerable to induction hypotension, a combination well recognised in patients with multivessel CAD as a precipitant of perioperative myocardial injury.[6,9]

Neuraxial techniques, while often considered to reduce cardiac morbidity in certain surgical contexts, are not always ideal in patients with HFrEF and acute decompensation. Spinal anaesthesia induces a dense sympathectomy that, in the compromised ventricle, can abruptly reduce preload and afterload and precipitate severe hypotension with poor tolerance of fluid resuscitation in the face of pulmonary oedema.[11-13] Epidural techniques allow more titratable onset but still entail bilateral sympathetic blockade and can be technically challenging in the presence of diuretic-induced dehydration and altered coagulation profile from concurrent antiplatelet therapy in CAD patients.[11,13] In our patient, chronic dual antiplatelet therapy for previous stent placement was maintained, which—while not an absolute contraindication—further discouraged neuraxial intervention.

Ultrasound-guided femoral plus popliteal-sciatic block occupies a distinct position among anaesthetic options for this clinical picture. By selectively targeting the peripheral nerves, PNB avoids extensive sympathetic blockade and therefore exerts minimal effect on systemic vascular resistance, allowing preservation of baseline blood pressure and cardiac output.[11,13,14] The femoral nerve, arising from the posterior divisions of L2–L4, supplies motor innervation to the anterior compartment of the thigh and sensory innervation to the anteromedial thigh,

medial lower leg, and medial foot via the saphenous nerve.[18,23] The sciatic nerve, originating from L4–S3, divides at the popliteal fossa into the tibial and common peroneal branches, which together supply motor and sensory innervation to the posterior compartment of the leg, the foot, and the ankle.[14,18] Blockade at the femoral and popliteal levels thus provides anaesthesia to virtually the entire distal lower limb and is eminently suited for foot debridement.[11,17,19] Ultrasound guidance has become the standard of care for these blocks because it enables direct visualisation of the needle, the nerves and the surrounding vasculature, leading to superior block success, faster onset, lower local anaesthetic dose, and reduced risk of neurovascular injury and systemic toxicity.[14,18,20,21]

The intraoperative hemodynamic and respiratory profile observed in our patient aligns with the expected physiologic signature of a cardiopulmonary-sparing anaesthetic technique. Systolic blood pressure remained in the 90–110 mmHg range throughout the procedure, heart rate stayed close to 85 beats/min in sinus rhythm, and oxygen saturation remained at 100% with supplemental oxygen at 3 L/min via nasal cannula. These values contrast with the substantial fluctuations observed in comparable patients receiving general anaesthesia for comparable procedures.[6,10] In one recent Danish nationwide cohort, general anaesthesia for peripheral vascular surgery in patients with significant cardiac comorbidity was associated with worse short-term outcomes compared with locoregional techniques after risk adjustment, a finding that supports the current clinical decision.[6] Jung and colleagues similarly reported successful use of ultrasound-guided peripheral nerve block for lower-limb surgery in an HFrEF patient, with intraoperative blood pressures and cardiac output remaining stable and without the need for inotropic support, mirroring our experience.[15] Shamim and colleagues described a series of high-risk patients undergoing above- and below-knee amputations under ultrasound-guided PNB with favourable intraoperative and postoperative outcomes.[16] Bisono and colleagues documented the use of combined popliteal and femoral blocks in a patient with congestive heart failure stage III undergoing foot amputation, reporting stable intraoperative parameters and smooth postoperative recovery.[19] As demonstrated in Table 4, our case adds to this accumulating evidence base by providing a detailed description of a patient with HFrEF, three-vessel CAD, pulmonary oedema and bilateral pleural effusion undergoing foot debridement—a combination that is not fully captured by the previously reported cases.

A second dimension of the discussion relates to local anaesthetic pharmacology and safety. We used a dual-agent mixture of 20 mL of 1.5% lidocaine (300 mg total) and 20 mL of 0.25% bupivacaine (50 mg total), distributed evenly between the two blocks. This approach combines the relatively rapid onset of lidocaine with the prolonged duration of bupivacaine, producing surgical anaesthesia for the 60-minute procedure and carrying over into the early postoperative period for analgesia.[14,18,22,23] Total doses were well within the recommended maximum safe doses based on the patient's weight (approximately 70 kg), and incremental injection with ultrasound visualisation virtually eliminated the risk of intravascular administration.[18,20,21,24] Nevertheless, local anaesthetic systemic toxicity (LAST) remains a potentially catastrophic complication, and adherence to the ASRA practice advisory—including availability of lipid emulsion, continuous ECG monitoring, and careful assessment of early neurological symptoms such as metallic taste or tinnitus—was strictly observed.[24] No signs of LAST were detected at any time. The risk of nerve injury after ultrasound-guided PNB is small but not zero; in contemporary databases, the rate of transient neurological symptoms has been reported at approximately 0.18%, and permanent injury at less than 0.04%.[21,25] In our patient, no postoperative neurological deficit was observed beyond the expected duration of block resolution.

The third dimension worth exploring is the postoperative clinical trajectory, particularly the role of PNB in mitigating pain, reducing opioid

requirements, and facilitating continued decongestive management. Postoperative pain in CLTI-related foot debridement can be substantial, and opioid-sparing strategies are especially valuable in patients with HFrEF because opioids reduce sympathetic tone and depress respiration, risks of particular concern in the setting of decompensated heart failure.[11,13,22] Longer-acting bupivacaine provides residual analgesia in the early postoperative period and facilitates a smoother transition to multimodal analgesia consisting of scheduled paracetamol and low-dose weak opioids as needed. In our patient, pain scores on postoperative days 1–2 remained low (2–3/10 on the numerical rating scale), allowing aggressive diuresis and mobilisation without hemodynamic destabilisation. Unlike general anaesthesia, PNB does not impair the patient's ability to cooperate with chest physiotherapy and incentive spirometry, both of which are important components of postoperative pulmonary recovery in patients with pulmonary oedema and pleural effusion.[10,13]

Several caveats must also be recognised. First, successful PNB in the cardiopulmonary-compromised patient depends heavily on operator expertise. Ultrasound-guided regional anaesthesia has a learning curve, and the quality of image acquisition, needle tracking, and local anaesthetic deposition are non-trivial determinants of block success.[14,18] Institutional credentialing and structured training programmes (including hands-on workshops and supervised clinical rotations) are therefore essential complements to the clinical decision to use regional anaesthesia in such patients. Second, not every patient is a candidate for PNB; important relative contraindications include patient refusal or inability to cooperate, significant pre-existing neuropathy in the nerve distribution, severe coagulopathy, and local infection at the site of injection. In our patient, none of these contraindications were present. Third, a fallback plan for conversion to general anaesthesia must always be available; our theatre was prepared with difficult-airway equipment, rapid-induction agents, and a low-dose vasoactive infusion ready for immediate administration if required.[23-25]

From a health-systems perspective, the adoption of PNB for CLTI-related foot debridement in resource-limited settings offers potential advantages that extend beyond the individual patient. The technique reduces perioperative complications that might otherwise require intensive-care admission, shortens postoperative recovery, and can be performed without reliance on advanced anaesthetic machines and volatile agents, all of which are economically important in low- and middle-income countries.[11,26] Capacity-building initiatives to expand ultrasound availability and training in regional anaesthesia are therefore of strategic value for vascular and trauma care, particularly in rural and peripheral hospitals where general anaesthesia infrastructure may be limited.

Finally, the limitations of the present report should be acknowledged. As a single-case report, generalisability is inherently constrained; the outcome observed reflects a specific confluence of patient factors, institutional capability and team expertise, and should not be extrapolated uncritically. Follow-up data are reported for the index hospitalisation only; longer-term outcomes—including wound healing, limb salvage rate, subsequent revascularisation or amputation, and cardiovascular events—will require ongoing surveillance. Formal health-economic analysis and comparison with a counterfactual anaesthetic strategy were not available. Future prospective studies with standardised anaesthetic protocols, larger samples, and objective hemodynamic and respiratory endpoints are needed to consolidate the role of ultrasound-guided PNB in PAD patients with severe cardiopulmonary comorbidity. Despite these limitations, the present report contributes to the growing evidence that regional anaesthesia—specifically ultrasound-guided femoral plus popliteal-sciatic block—can serve as an effective, safe, and cardiopulmonary-sparing anaesthetic option for high-risk patients presenting for lower-extremity surgical debridement.

Table 4. Comparison with similar published cases of ultrasound-guided peripheral nerve block in cardiopulmonary-compromised patients.

Study	Year	Patient profile	Block technique	Hemodynamic outcome
Jung et al.*	2021	HFrEF (EF 35%), scheduled lower-limb surgery	US-guided femoral + sciatic block	Stable BP/HR; no vasopressor required
Shamim et al.	2018	High-risk patients undergoing above-/below-knee amputation	US-guided femoral + sciatic block	Uneventful hemodynamics; improved analgesia
Bisono et al.	2023	Congestive heart failure stage III, pedis amputation	US-guided popliteal + femoral block	Stable intraoperative course; no LAST
Körner et al.†	2024	Danish nationwide cohort of PAS with cardiac comorbidity	Regional vs. general anaesthesia	Regional associated with better 30-day outcomes
Present case ‡	2026	59-y male, three-vessel CAD, HFrEF 34–36%, pulmonary oedema, bilateral pleural effusion, bilateral PAD	US-guided femoral + popliteal-sciatic block with 20 mL 1.5% lidocaine + 20 mL 0.25% bupivacaine	SBP 90–110, HR ~85, SpO_2 100%; no vasopressor/opioid use

* Case report; † nationwide cohort analysis; ‡ the present case. CAD = coronary artery disease; PAD = peripheral arterial disease; PAS = peripheral arterial surgery.

4. Conclusion

Ultrasound-guided combined femoral and popliteal-sciatic nerve block provided effective surgical anaesthesia, stable hemodynamics, and preserved spontaneous ventilation in a 59-year-old man with bilateral peripheral arterial disease, HFrEF (EF 34–36%), three-vessel coronary artery disease, acute decompensated heart failure with pulmonary oedema and bilateral pleural effusion who underwent urgent debridement and necrotomy of the left foot. The technique avoided the myocardial depression and ventilatory compromise associated with general anaesthesia, as well as the sympathectomy and hypotension associated with neuraxial approaches, and permitted continuous patient cooperation throughout a 60-minute procedure without the need for vasopressors, systemic opioids or sedatives. The present case reinforces the emerging body of literature indicating that ultrasound-guided peripheral nerve blockade is a safe and feasible cardiopulmonary-sparing anaesthetic strategy for lower-extremity surgery in patients with severe combined cardiovascular and respiratory comorbidity. Broader adoption should be supported by formal training programmes, institutional credentialing and continued outcome research in multicentre settings.

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