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Laporan Kasus

BERADA DI UJUNG TANDUK: MANAJEMEN ANESTESI PADA LANSIA DENGAN PERITONITIS YANG MENGALAMI GAGAL JANTUNG STADIUM AKHIR

BALANCING ON THE BRINK: ANESTHETIC MANAGEMENT IN GERIATRIC PERITONITIS WITH END-STAGE HEART FAILURE

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A B S T R A K

Manajemen anestesi pada pasien geriatri dengan peritonitis emergensi dan gagal jantung stadium lanjut merupakan tantangan perioperatif yang kompleks karena respons inflamasi intraabdomen, kebutuhan perfusi jaringan, dan keterbatasan cadangan miokard harus dikendalikan secara bersamaan. Artikel ini melaporkan seorang laki-laki 72 tahun dengan nyeri abdomen difus, demam, sesak saat aktivitas, frailty sedang, kapasitas fungsional sangat rendah, hiponatremia, koagulopati, efusi pleura bilateral, LVEF 22%, regurgitasi mitral iskemik berat, serta disfungsi ventrikel kanan. Setelah evaluasi multidisiplin, pasien diklasifikasikan ASA IV dan menjalani anestesi umum teritrasi untuk pembedahan abdomen emergensi. Strategi perioperatif difokuskan pada stabilitas hemodinamik melalui induksi bertahap dengan midazolam, fentanyl, dan rokuronium, pemeliharaan sevofluran dosis rendah, ventilasi tekanan positif yang tidak berlebihan, serta restriksi cairan berbasis respons perfusi. Sekitar 1000 mL kristaloid diberikan dengan kehilangan darah minimal, diuresis adekuat, dan tanpa kebutuhan vasopresor atau inotropik dosis tinggi. Pasien berhasil diekstubasi, dipantau ketat pascaoperasi, mendapat analgesia multimodal, dan tidak mengalami edema paru, aritmia maligna, hipoksemia berulang, atau eksaserbasi gagal jantung akut. Pemulihan stabil hingga pulang hari keempat menegaskan pentingnya anestesi presisi, manajemen cairan konservatif, kesiapan vasoaktif, ventilasi protektif, dan kolaborasi multidisiplin pada pasien berisiko ekstrem.

A B S T R A C T

Anesthetic management for emergency peritonitis in geriatric patients with end-stage heart failure represents a critical perioperative challenge, where intra-abdominal inflammation, tissue perfusion, and severely restricted myocardial reserve must be balanced within a narrow therapeutic margin. This article reports a 72-year-old man with diffuse abdominal pain, fever, exertional dyspnea, moderate frailty, markedly limited functional capacity, hyponatremia, coagulopathy, bilateral pleural effusion, LVEF 22%, severe ischemic mitral regurgitation, and right ventricular dysfunction. After multidisciplinary assessment, the patient was classified as ASA IV and underwent titrated general anesthesia for emergency abdominal surgery. Management prioritized hemodynamic stability through gradual induction with midazolam, fentanyl, and rocuronium, low-dose sevoflurane maintenance, avoidance of excessive positive-pressure ventilation, and restrictive fluid therapy guided by perfusion response. Approximately 1000 mL crystalloid was administered with minimal blood loss, preserved urine output, and no high-dose vasopressor or inotropic support. The patient was safely extubated, monitored postoperatively, treated with multimodal analgesia, and developed no pulmonary edema, malignant arrhythmia, recurrent hypoxemia, or acute heart failure exacerbation. Recovery until discharge on postoperative day four highlights precision anesthesia, conservative fluid management, vasoactive readiness, protective ventilation, and multidisciplinary coordination.

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INTRODUCTION

The perioperative management of geriatric patients undergoing emergency abdominal surgery represents one of the most demanding scenarios in contemporary anesthetic practice, particularly when complicated by advanced heart failure. The coexistence of peritonitis-driven systemic inflammation and severely impaired cardiac reserve creates a fragile physiological equilibrium characterized by limited compensatory capacity and heightened susceptibility to hemodynamic instability.[1] In such settings, anesthesia extends beyond its conventional supportive role and becomes a decisive factor influencing perioperative survival. The anesthesiologist must navigate a narrow therapeutic window, maintaining adequate tissue perfusion while simultaneously avoiding myocardial depression and volume overload. This delicate balance requires a highly individualized, physiology-guided anesthetic approach grounded in a comprehensive understanding of cardiovascular pathophysiology and systemic inflammatory responses.[2]

The global burden of intra-abdominal sepsis and heart failure with reduced ejection fraction (HFrEF) continues to rise in parallel with population aging, contributing significantly to perioperative morbidity and mortality. Emergency surgical intervention for peritonitis is associated with substantial mortality, with rates ranging from 10% to 30%, and even higher in elderly patients with multiple comorbidities.[3] Concurrently, heart failure affects more than 64 million individuals worldwide, with HFrEF representing a major subtype associated with high rates of hospitalization and reduced survival. Patients with advanced cardiac dysfunction undergoing non-cardiac surgery face disproportionately elevated perioperative risk, with reported mortality exceeding 20–40%, particularly in the presence of systemic infection, frailty, or multiorgan dysfunction.[4,5] These epidemiological trends underscore the convergence of surgical urgency and cardiovascular vulnerability as a critical determinant of adverse outcomes, especially in geriatric populations where physiological reserves are inherently diminished.

The anesthetic management of patients with advanced heart failure in the setting of peritonitis is further complicated by competing and often opposing physiological demands. Sepsis-induced vasodilation, capillary leak, and relative hypovolemia necessitate cautious fluid resuscitation to sustain organ perfusion, yet excessive fluid administration may precipitate pulmonary congestion and exacerbate ventricular dysfunction.[6,7] In addition, commonly used anesthetic agents induce varying degrees of myocardial depression and systemic vasodilation, potentially leading to abrupt reductions in cardiac output and perfusion pressure. This challenge is magnified in the presence of severe left ventricular systolic dysfunction, valvular abnormalities such as mitral regurgitation, and concomitant right ventricular impairment, where the margin for hemodynamic compensation is exceedingly narrow.[8,9] The absence of universally standardized perioperative strategies for such complex clinical scenarios highlights a critical gap in current practice and reinforces the need for individualized, high-precision anesthetic management.[5]

Addressing these challenges necessitates a paradigm shift toward precision hemodynamic management, integrating goal-directed therapy with tailored anesthetic selection and vigilant perioperative monitoring. Contemporary approaches emphasize the careful titration of anesthetic agents to minimize myocardial depression, alongside restrictive yet targeted fluid administration guided by dynamic parameters of fluid responsiveness. Early and judicious use of vasoactive agents may be required to maintain adequate perfusion pressure without exacerbating cardiac workload.[10] Furthermore, multidisciplinary collaboration involving anesthesiologists, cardiologists, and intensivists is essential to optimize perioperative planning and postoperative care. This report aims to present the anesthetic management of a geriatric patient with peritonitis and advanced heart failure, highlighting critical considerations in maintaining hemodynamic stability under extreme physiological constraints and contributing to the evolving framework of perioperative care in high-risk surgical populations

CASE ILLUSTRATION

A 72-year-old male presented with a two-week history of progressively worsening diffuse abdominal pain accompanied by intermittent fever over the preceding month. Two days prior to admission, he developed exertional dyspnea and left-sided chest discomfort. His medical history was notable for longstanding chronic heart failure of ischemic origin, diagnosed approximately 10 years earlier, with poor adherence to therapy in the last six months. His medications included furosemide, spironolactone, bisoprolol, and ramipril. Functional capacity was severely limited (MET =1), and frailty assessment (Clinical Frailty Scale 5) indicated moderate frailty, reflecting markedly reduced physiological reserve.On examination, the patient appeared moderately ill but fully conscious (Glasgow Coma Scale 15). Vital signs revealed blood pressure of 112/72 mmHg, heart rate of 62 beats per minute, respiratory rate of 18 breaths per minute, and oxygen saturation of 96% on 3 L/min oxygen via nasal cannula. Cardiovascular examination demonstrated a displaced apical impulse and a systolic murmur at the apex, consistent with mitral regurgitation. Pulmonary findings included decreased breath sounds at both lung bases, suggesting bilateral pleural effusion. Abdominal examination revealed diffuse mild tenderness with decreased bowel sounds. Bilateral pitting edema indicated systemic congestion. Despite stable vital signs, the overall clinical profile suggested a compensated yet fragile hemodynamic state at high risk of decompensation.

Laboratory evaluation demonstrated hyponatremia (Na^+ 125 mmol/L), prolonged coagulation (PT 19.2 seconds, INR 1.43), mild hypoalbuminemia (3.4 g/dL), and borderline renal impairment (urea 49.2 mg/dL, creatinine 1.12 mg/dL, eGFR 69.8 mL/min/1.73 m²). Arterial blood gas analysis revealed mild metabolic alkalosis (pH 7.48, HCO_3^- 28 mmol/L) with adequate oxygenation. These abnormalities reflected multisystem involvement in the setting of chronic cardiac dysfunction and systemic inflammatory stress. Chest radiography revealed cardiomegaly, characterized by an enlarged cardiac silhouette, consistent with chronic cardiac remodeling due to advanced heart failure. Additionally, there was evidence of bilateral pleural effusion, as indicated by blunting of both costophrenic angles and basal opacities. These findings are indicative of chronic volume overload and systemic venous congestion, supporting the diagnosis of decompensated heart failure physiology. The presence of pleural effusion further implies reduced pulmonary compliance and increased risk of perioperative hypoxia, particularly during positive pressure ventilation. Abdominal radiography (supine view) demonstrated a non-specific bowel gas pattern, with mild gaseous distension of intestinal loops without clear evidence of mechanical obstruction. There was no obvious free intraperitoneal air visualized; however, the sensitivity of supine radiographs for detecting pneumoperitoneum is limited. In the clinical context of persistent abdominal pain and systemic inflammatory features, these findings are suggestive of paralytic ileus or inflammatory intra-abdominal pathology, supporting the working diagnosis of peritonitis.

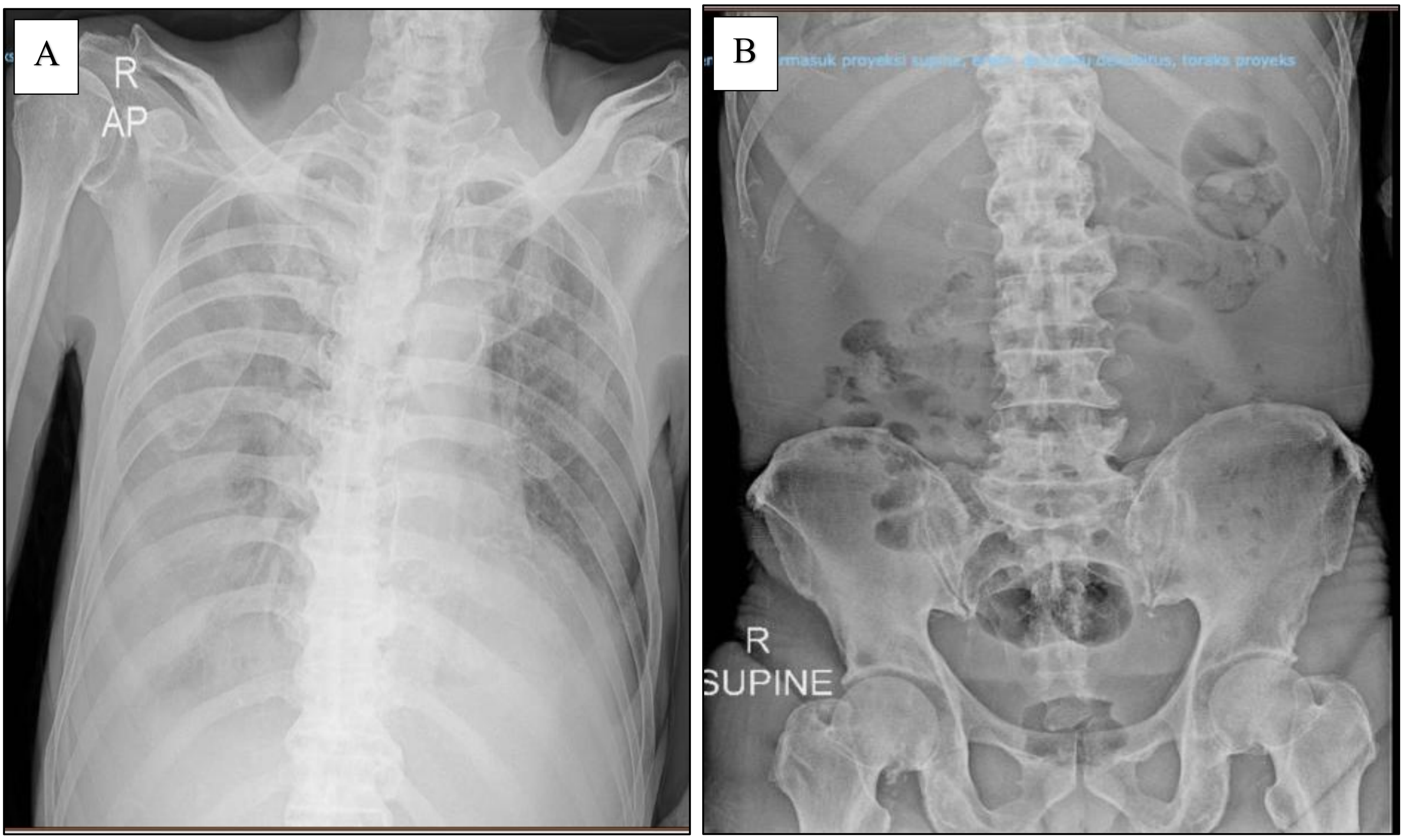

Figure 1. Radiological Findings; (A) Chest radiograph demonstrating cardiomegaly with bilateral pleural effusion, consistent with chronic congestive heart failure and reduced pulmonary reserve. (B) Abdominal radiograph (supine view) showing a non-specific bowel gas pattern, suggestive of inflammatory intra-abdominal pathology without clear evidence of mechanical obstruction.

Electrocardiographic evaluation demonstrated sinus rhythm with a regular ventricular response, accompanied by features consistent with left atrial enlargement (LAE) and left ventricular hypertrophy (LVH). The presence of increased voltage in precordial leads (V5–V6) and associated repolarization abnormalities suggested chronic pressure and volume overload of the left ventricle. These findings are in line with the patient’s history of longstanding hypertensive heart disease and ischemic cardiomyopathy. Importantly, no acute ischemic changes or malignant arrhythmias were identified. However, the presence of structural electrical remodeling reflects chronic myocardial stress and reduced cardiac reserve, which significantly increases the risk of perioperative hemodynamic instability and arrhythmic events under anesthetic stress.Transthoracic echocardiography revealed severely reduced left ventricular systolic function (LVEF 22%), with global hypokinesia and ventricular dilation. Severe ischemic mitral regurgitation was present, along with mild aortic and tricuspid regurgitation. Right ventricular function was impaired (TAPSE 11 mm), indicating biventricular dysfunction.

Multidisciplinary evaluation involving internal medicine, cardiology, and pulmonology demonstrated that advanced cardiac dysfunction was the central driver of systemic instability. Cardiologist assessment revealed severely reduced left ventricular systolic function (LVEF 22%), severe mitral regurgitation, right ventricular dysfunction, and a mild pericardial effusion, indicating markedly limited cardiac reserve and high susceptibility to perioperative hemodynamic collapse.

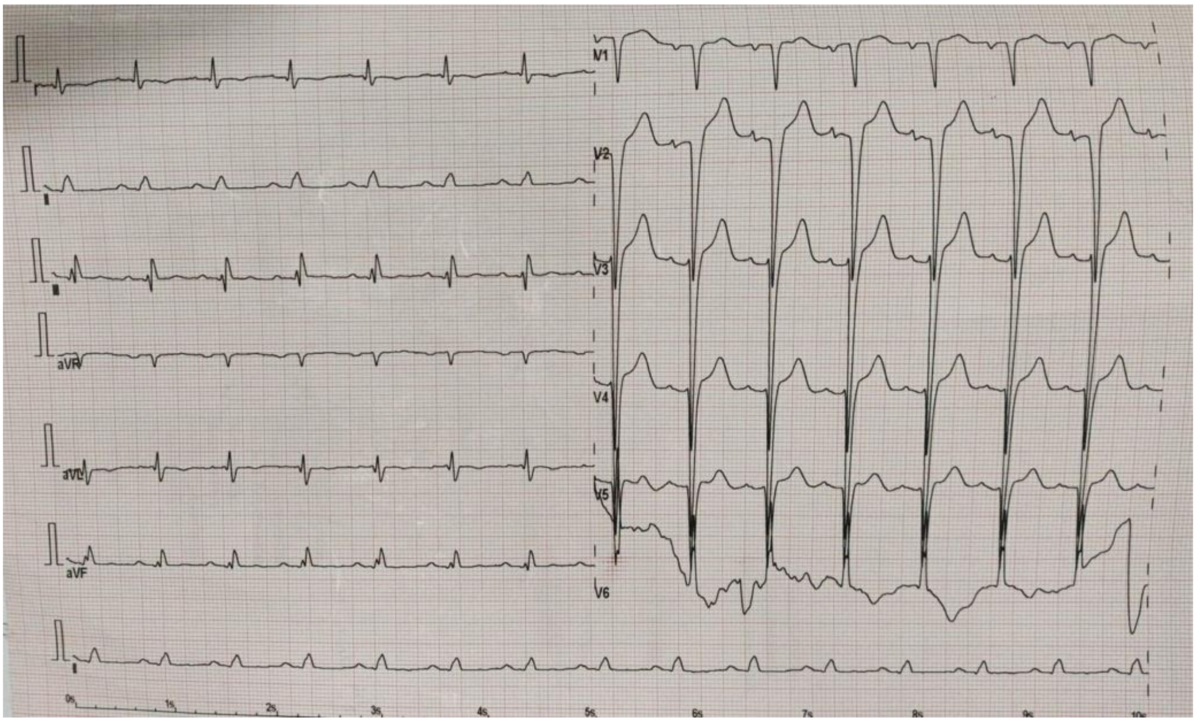

Figure 2. Electrocardiographic Findings. Electrocardiogram demonstrating sinus rhythm with features of left atrial enlargement and left ventricular hypertrophy, accompanied by repolarization abnormalities in the precordial leads (V1–V6), consistent with chronic structural cardiac remodeling .

From the pulmonologist perspective, the presence of bilateral transudative pleural effusion reflected cardiogenic pulmonary involvement with reduced respiratory reserve, while internal medicine evaluation highlighted metabolic vulnerability, reinforcing a cardio–pulmonary–metabolic continuum rather than isolated organ dysfunction. The coexistence of suspected peritonitis requiring urgent surgical intervention created a critical clinical dilemma, where delaying surgery risked progression to sepsis, whereas proceeding posed a substantial risk of cardiovascular decompensation. Preoperative optimization was therefore directed toward stabilizing modifiable factors without disrupting this fragile equilibrium, including cautious correction of hyponatremia using 3% NaCl (target Na^+ ≥130 mmol/L), correction of coagulopathy with 700 mL fresh frozen plasma, and preparation of one unit of packed red blood cells. The patient was classified as ASA IV, reflecting a severe systemic disease with constant threat to life, and overall, these interconnected findings highlight a synergistic risk profile in which cardiac dysfunction amplifies pulmonary and metabolic derangements, necessitating a precision-based perioperative strategy to maintain a narrow hemodynamic balance.

In the operating room, standard monitoring (electrocardiography, non-invasive blood pressure, and pulse oximetry) was applied, with readiness for escalation to invasive monitoring if instability occurred. General anesthesia was induced using a hemodynamically titrated approach: Midazolam 1 mg IV for premedication, followed by 5 mg IV during induction, Fentanyl 125 mcg IV (2.4 mcg/kg) to attenuate sympathetic response, Rocuronium 40 mg IV (0.77 mg/kg) to facilitate intubation. Preoxygenation was performed using 100% oxygen at 6 L/min for 3–5 minutes. Endotracheal intubation was successfully achieved using a 7.0 mm tube. Mechanical ventilation was initiated with volume-controlled settings (tidal volume 400 mL, respiratory rate 10–12 breaths per minute), avoiding excessive positive pressure to preserve venous return and right ventricular preload.

Anesthesia was maintained using sevoflurane 1.5–1.8 vol% in an oxygen-enriched mixture. Additional intraoperative medications included ondansetron 8 mg IV, tranexamic acid 1 g IV, and paracetamol 1 g IV for multimodal support. A central challenge during intraoperative management was maintaining a balance between adequate perfusion and avoidance of fluid overload. Fluid therapy was administered conservatively using Ringer's lactate, guided by calculated requirements and clinical monitoring. Total intraoperative fluid administration was approximately 1000 mL, resulting in a net positive balance of +650 mL after accounting for urine output (300 mL) and minimal blood loss (50 mL).Hemodynamic parameters remained relatively stable throughout the procedure without the need for high-dose vasopressor or inotropic support, reflecting effective maintenance of the delicate equilibrium between preload, afterload, and myocardial contractility. The duration of anesthesia was approximately two hours. At the end of the procedure, the patient demonstrated adequate spontaneous respiration and protective airway reflexes. Extubation was performed cautiously following suctioning and oxygenation optimization.

Following the conclusion of the surgical procedure, the patient's transition from anaesthesia occurred in a gradual and controlled manner. The patient demonstrated the restoration of adequate spontaneous ventilation, protective airway reflexes, and satisfactory oxygenation prior to extubation. Extubation was performed without incident following airway suctioning and optimisation of oxygen delivery. Post-extubation complications were not observed in the immediate period following the procedure. These complications include laryngospasm, aspiration, recurrent hypoxemia, and respiratory fatigue. Following a comprehensive evaluation, the patient was subsequently transferred to a monitored postoperative care setting, owing to his high-risk cardiovascular profile. On arrival, the patient was conscious, cooperative, and hemodynamically stable, with an Aldrete score of 10, indicating adequate recovery of consciousness, respiration, circulation, oxygen saturation, and motor activity. Postoperative oxygen therapy was continued using a nasal cannula at a rate of 3 L/min. The patient's oxygen saturation levels remained consistent, obviating the necessity for non-invasive ventilation or reintubation. The provision of analgesia was achieved through the implementation of a multimodal strategy, encompassing tramadol infusion and non-opioid analgesic support. This approach resulted in adequate pain control and the absence of any evidence of opioid-related respiratory depression. Subsequent serial postoperative assessments did not reveal any signs of acute pulmonary oedema, worsening pleural effusion, clinically significant myocardial ischaemia, or exacerbation of acute heart failure. Peripheral perfusion remained adequate, urine output was maintained, and there was no requirement for aggressive fluid resuscitation, high-dose vasoactive therapy, or mechanical ventilatory support.

In the 24-hour period following the procedure, the patient exhibited clinical stability, as evidenced by continuous cardiopulmonary monitoring. The patient's blood pressure was successfully maintained without any occurrence of persistent hypotension. Furthermore, the patient's heart rate remained within the acceptable range, and the oxygenation levels were adequately supported by low-flow oxygen supplementation. The absence of episodes of malignant arrhythmia, altered consciousness, progressive dyspnea, or signs of systemic hypoperfusion was observed. The surgical recovery course was not complicated by immediate adverse events related to anaesthesia. In view of the patient's pre-existing severe cardiac dysfunction, particular attention was directed towards avoiding fluid overload, monitoring respiratory workload, and identifying early signs of decompensated heart failure.

On the second postoperative day, the patient exhibited sustained clinical recovery, remaining fully conscious, breathing comfortably, and maintaining stable hemodynamic parameters without any requirement for escalation of cardiopulmonary support. The absence of clinical evidence indicates that recurrent hypoxemia, pulmonary congestion, worsening peripheral oedema, low-output state, and organ hypoperfusion are not present. The commencement of oral intake and gradual mobilisation was initiated in accordance with surgical tolerance, while analgesic requirements underwent a progressive decline and pain remained adequately controlled according to routine Visual Analog Scale assessment. This favourable trajectory persisted throughout the third postoperative day, with a gradual reduction in oxygen requirement and no need for non-invasive ventilation, reintubation, high-dose vasoactive therapy, or intensive cardiopulmonary intervention. A series of clinical evaluations were conducted, revealing no exacerbation of pleural effusion, pulmonary oedema, progressive congestion, malignant arrhythmia, respiratory compromise, or immediate anaesthesia-related complications. On the fourth postoperative day, the patient exhibited no acute heart failure exacerbation or recurrent cardiopulmonary deterioration, and remained clinically improved and hemodynamically stable. Following a comprehensive multidisciplinary reassessment by the surgical and perioperative care teams, the patient was deemed suitable for discharge. The patient was discharged on the fourth postoperative day, receiving detailed instructions regarding wound care, the continuation and optimisation of chronic heart failure therapy, outpatient cardiology follow-up, and return precautions for dyspnoea, chest pain, fever, recurrent abdominal pain, reduced urine output, or worsening peripheral oedema.

DISCUSSION

The central pathophysiological problem in this case was the collision between two opposing hemodynamic requirements. Peritonitis and intra-abdominal inflammatory stress may promote vasodilation, capillary leakage, relative intravascular depletion, and impaired tissue perfusion .[11] In contrast, advanced heart failure markedly limits the capacity to tolerate volume expansion because additional preload may increase ventricular filling pressures, worsen functional mitral regurgitation, intensify pulmonary venous congestion, and precipitate respiratory compromise. [12,13] This creates a clinically important fluid–cardiac paradox; inadequate resuscitation may aggravate hypoperfusion, whereas excessive fluid administration may worsen pulmonary edema and biventricular failure.[14,15] In the present case, this paradox was evident even before surgery, as the patient already demonstrated bilateral pleural effusion, peripheral edema, severe left ventricular dysfunction, and impaired right ventricular function. Therefore, liberal fluid resuscitation would have carried a substantial risk of cardiopulmonary decompensation.[16,17] Consequently, the anesthesia management prioritized dynamic hemodynamic monitoring over static pressure measurements to guide fluid administration, as pulse pressure and stroke volume variations are more reliable indicators of preload responsiveness in ventilated patients. [18] For this reason, the intraoperative fluid strategy was deliberately conservative and individualized. Approximately 1000 mL of crystalloid was administered during the procedure, with minimal blood loss, preserved urine output, and a modestly positive fluid balance. This strategy was not intended to normalize every hemodynamic variable through volume loading, but to maintain adequate organ perfusion while avoiding further congestion. Contemporary perioperative and critical care concepts increasingly support goal-directed rather than fixed-volume resuscitation in high-risk surgical patients, particularly when ventricular compliance and cardiac reserve are limited. [19–21] In this patient, the absence of refractory hypotension, systemic hypoperfusion, pulmonary edema, or acute heart failure exacerbation suggests that conservative volume administration was physiologically appropriate.[14]

The anaesthetic technique was also determined by the patient's cardiac vulnerability. Induction of general anaesthesia has been demonstrated to reduce systemic vascular resistance, venous return and myocardial contractility, effects that may be poorly tolerated in patients with severe systolic dysfunction. [22,23] Propofol-based induction, although widely utilised, has been shown to produce clinically significant vasodilation and myocardial depression in patients with limited cardiac reserve. In this case, the titrated use of midazolam, fentanyl and rocuronium was chosen to reduce abrupt sympathetic and hemodynamic perturbations during induction and tracheal intubation.[24] Opioid-based attenuation of the laryngoscopic response was a particularly salient consideration, given that uncontrolled sympathetic stimulation has been demonstrated to increase myocardial oxygen demand, exacerbate ischemic burden, and destabilise mitral regurgitation physiology. [25] The maintenance of low-dose sevoflurane further exemplifies a strategy of anaesthetic precision rather than deep volatile suppression. Volatile anaesthetic agents have been demonstrated to exert a dose-dependent effect on myocardial contractility and systemic vascular resistant. Consequently, it is conceivable that excessive anaesthetic depth could have precipitated hypotension in this patient. [26,27] Conversely, inadequate anaesthetic depth could have resulted in sympathetic activation, tachycardia, increased afterload, and worsening regurgitant flow. The intraoperative course was found to be clinically stable, with no occurrence of malignant arrhythmias and a lack of requirement for high-dose vasoactive or inotropic support. These findings suggest that the depth of anaesthesia, vascular tone, and myocardial workload were successfully maintained within acceptable physiological parameters.[28,29]

The postoperative course is a crucial component of this case, as it demonstrates that perioperative stability was sustained beyond the operating room. In the 24-hour period following the procedure, the patient exhibited clinical stability while undergoing continuous cardiopulmonary monitoring. The patient's blood pressure was successfully maintained without any occurrence of persistent hypotension. Furthermore, the patient's heart rate remained within an acceptable range, and the patient received adequate oxygenation support through low-flow oxygen supplementation. No malignant arrhythmias, altered consciousness, progressive dyspnoea, recurrent hypoxemia, or signs of systemic hypoperfusion were observed. This early postoperative profile is of clinical significance because patients with severe heart failure are at continued risk of delayed decompensation after anaesthesia due to residual myocardial depression, fluid shifts, inflammatory stress, pain-related sympathetic activation, and respiratory workload. [14,30,31] Postoperative monitoring in such cases is therefore directed towards the early detection of three major threats: fluid overload, respiratory deterioration, and decompensated heart failure. The patient did not develop pulmonary congestion, worsening peripheral oedema, an acute low-output state, or a requirement for escalation to ventilatory or vasoactive support. The management of pain was also a significant consideration. Inadequate analgesia has been demonstrated to intensify sympathetic tone and increase myocardial oxygen consumption.[32,33] Conversely, excessive opioid administration has been shown to impair ventilation, a phenomenon that is especially prevalent in elderly patients with limited pulmonary reserve. The utilisation of multimodal postoperative analgesia has been shown to facilitate pain control without clinically evident opioid-related respiratory depression, thereby supporting a stable transition from anesthetized physiology to spontaneous postoperative recovery.[34,35]

The sustained improvement from the second to the fourth postoperative day strengthened the favorable perioperative outcome in this high-risk patient. He remained conscious, breathed comfortably, and maintained stable hemodynamic parameters without escalation to non-invasive ventilation, reintubation, high-dose vasoactive therapy, or intensive cardiopulmonary support. The absence of pulmonary edema, worsening peripheral edema, low-output state, malignant arrhythmia, respiratory compromise, or acute heart failure exacerbation suggests that conservative fluid management and close cardiopulmonary surveillance were effective in preventing delayed decompensation, which is a major concern in patients with severe ventricular dysfunction and limited preload tolerance. [14,36] Gradual oral intake and mobilization according to surgical tolerance further indicated functional recovery rather than transient physiologic stability. Postoperative analgesia was also clinically relevant, as inadequate pain control may increase sympathetic activation, myocardial oxygen demand, and respiratory workload, whereas excessive opioid use may impair ventilation in elderly patients with limited pulmonary reserve.[37] Routine Visual Analog Scale assessment allowed analgesic titration, and the progressive reduction in analgesic requirement without respiratory depression supported the adequacy of the multimodal analgesic strategy. Discharge on the fourth postoperative day was considered appropriate after the patient passed the highest-risk early perioperative window without cardiopulmonary deterioration.[38] In advanced cardiac dysfunction, discharge readiness should be based not only on surgical recovery, but also on sustained hemodynamic stability, preserved oxygenation, absence of progressive congestion, adequate pain control, oral intake tolerance, and no need for organ-support escalation.[30] Multidisciplinary reassessment and clear discharge instructions regarding wound care, heart failure therapy optimization, cardiology follow-up, and warning symptoms provided an essential safety framework for post-hospital recovery.

The conceptual framework presented in **Figure 3** further illustrates the physiological rationale underlying the anesthetic strategy used in this case. The figure summarizes three interconnected domains that shaped perioperative decision-making: hemodynamic vulnerability with the fluid–cardiac paradox, pharmacologic hemodynamic optimization with anesthetic precision, and integrated cardiopulmonary management. In this patient, peritonitis-related inflammatory stress could promote vasodilation, capillary leakage, and relative intravascular depletion, whereas advanced cardiac failure limited tolerance to fluid loading. This explains why a restrictive, goal-directed fluid strategy was preferred over liberal resuscitation.

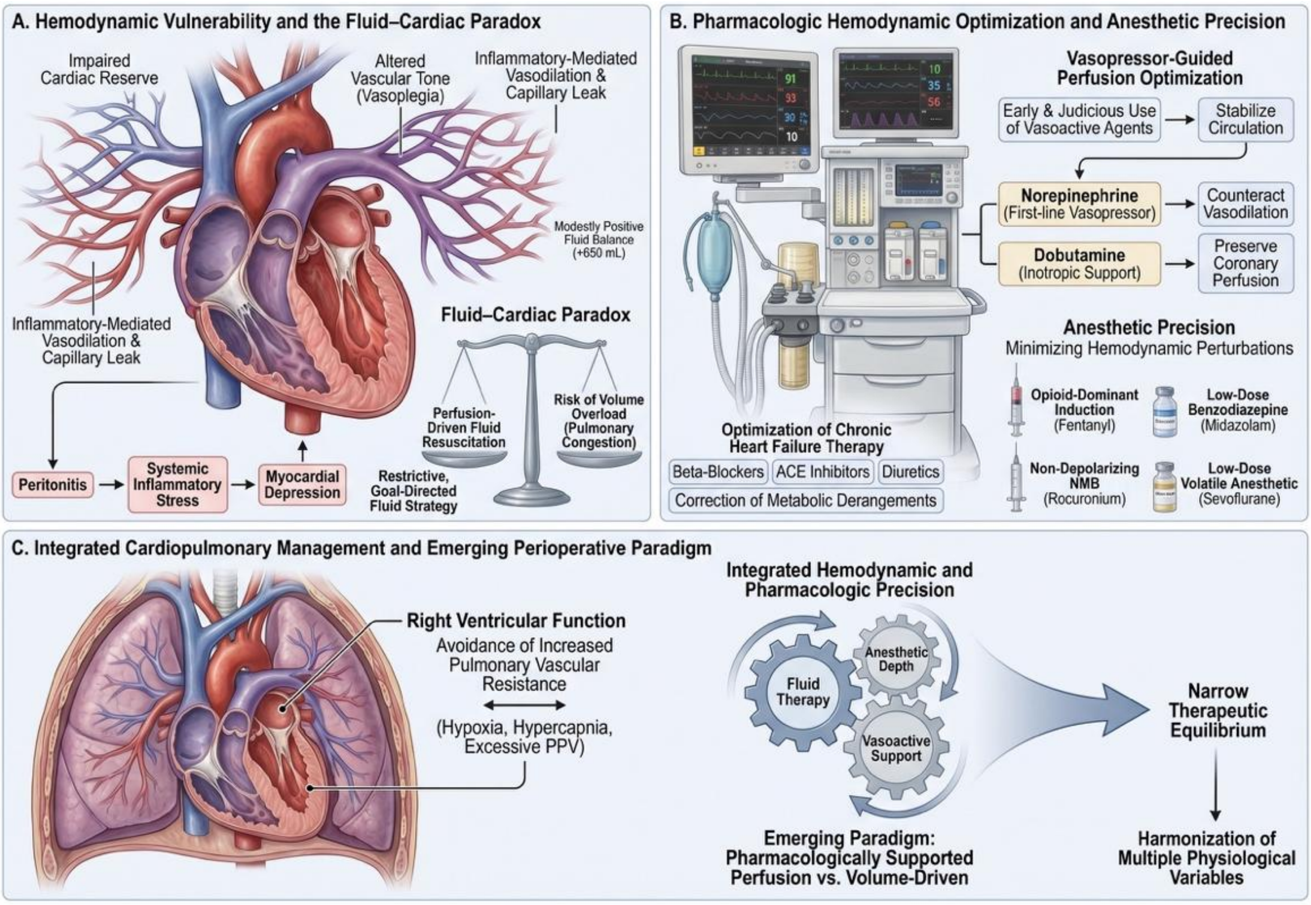

Figure 3. Integrated Hemodynamic and Pharmacologic Framework. (A) Hemodynamic vulnerability and fluid–cardiac paradox, illustrating the imbalance between sepsis-induced vasodilation, reduced cardiac reserve, and the risk of fluid overload. (B) Pharmacologic hemodynamic optimization, highlighting vasopressor-guided perfusion (norepinephrine, dobutamine) and anesthetic precision to minimize hemodynamic instability. (C) Integrated cardiopulmonary management, emphasizing right ventricular–pulmonary interaction and the concept of maintaining a narrow therapeutic equilibrium through coordinated fluid, ventilatory, and pharmacologic strategies.

The second component of the figure emphasizes the role of pharmacologic support and careful anesthetic titration. Although high-dose vasoactive therapy was not required in this case, preparedness for early vasopressor or inotropic intervention was clinically important to maintain perfusion without excessive fluid administration. The third component highlights the interaction between right ventricular function, pulmonary vascular resistance, and ventilatory management. This was particularly relevant because the patient had right ventricular dysfunction and bilateral pleural effusion, making avoidance of hypoxia, hypercapnia, and excessive positive-pressure ventilation essential. Overall, the framework supports the central principle of this case: perioperative stability in geriatric peritonitis with advanced cardiac failure depends on harmonizing fluid therapy, anesthetic depth, vasoactive readiness, and ventilatory strategy within a narrow therapeutic equilibrium.

This case supports a more specific recommendation that geriatric patients with peritonitis and advanced cardiac failure should be managed through an individualized, physiology-guided perioperative strategy rather than a routine anesthetic approach. In comparable high-risk patients, preoperative assessment should involve early multidisciplinary collaboration among anesthesiologists, surgeons, cardiologists, and intensive care teams to identify modifiable risks such as electrolyte imbalance, coagulation abnormality, pulmonary congestion, impaired ventricular function, and limited functional reserve. Intraoperatively, fluid administration should be restrictive, titrated, and guided by perfusion response rather than liberal volume replacement, particularly in patients with severely reduced ejection fraction, significant mitral regurgitation, right ventricular dysfunction, pleural effusion, or peripheral congestion. Anesthetic drugs should be administered gradually to minimize myocardial depression, abrupt vasodilation, tachycardia, hypotension, and excessive sympathetic stimulation during airway manipulation. Readiness for early vasoactive or inotropic support is essential when hypotension or hypoperfusion occurs, as perfusion in such patients should not depend solely on fluid loading. Ventilatory management should also be individualized to maintain oxygenation while avoiding excessive positive-pressure ventilation that may impair venous return and right ventricular function. Postoperatively, these patients require structured cardiopulmonary monitoring to detect delayed heart failure exacerbation, recurrent hypoxemia, pulmonary congestion, malignant arrhythmia, low-output state, reduced urine output, or worsening edema. Discharge should be considered only after sustained hemodynamic stability, preserved oxygenation, controlled pain, tolerance of oral intake, absence of progressive congestion, and no requirement for escalation of cardiopulmonary support, as demonstrated by the patient’s stable recovery until discharge on the fourth postoperative day.

CONCLUSION

The anesthetic management of geriatric patients with peritonitis complicated by advanced cardiac failure represents a critical convergence of surgical urgency and profound cardiovascular vulnerability, where perioperative outcomes are strongly influenced by the precision of anesthetic strategy rather than pathology alone. The coexistence of sepsis-induced hemodynamic instability and severely reduced myocardial reserve creates an extremely narrow therapeutic window, in which both inadequate resuscitation and excessive intervention may precipitate rapid clinical deterioration. Consequently, a physiology-guided, individualized anesthetic approach is essential to maintain adequate tissue perfusion while minimizing myocardial depression and fluid overload. This study underscores the importance of dynamic hemodynamic monitoring, judicious fluid administration, and the tailored use of vasoactive and anesthetic agents, supported by multidisciplinary coordination to optimize intraoperative stability and reduce postoperative complications. Ultimately, anesthetic management in this high-risk population should be regarded as a decisive determinant of survival, highlighting the need for continued refinement of goal-directed strategies and advanced monitoring to improve outcomes in this increasingly vulnerable geriatric cohort.

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