e-ISSN: 2807-6257

Open Access Indonesian Journal of Medical Reviews

[OAIJMR]

https://hmpublisher.com/index.php/oaijmr

Malignant Pericardial Effusion in Lung Adenocarcinoma: When to Escalate from Pericardiocentesis to Open Pericardiostomy

Ibnu Kharisman[1*], Prima Kharisma Hayuningrat[2]

[1]Department of Surgery, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

[2]Department of Cardiac, Thoracic, and Vascular Surgery, Dr. Moewardi Regional General Hospital, Surakarta, Indonesia

ARTICLE INFO

Keywords:
Cardiac tamponade
Lung adenocarcinoma
Malignant pericardial effusion
Palliative surgery
Pericardiocentesis

***Corresponding author:**
Ibnu Kharisman

E-mail address:
ibnukharisman@gmail.com

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

https://doi.org/10.37275/oaijmr.v5i6.810

A B S T R A C T

Malignant pericardial effusion (MPE) is a grave, life-limiting complication of advanced cancer, where lung adenocarcinoma is a leading cause. Its management is a cornerstone of palliative cardio-oncology, focused on alleviating debilitating dyspnea and enhancing the quality of remaining life. The optimal therapeutic pathway, especially following the failure of initial interventions, remains a critical challenge, demanding a careful balance between efficacy and treatment burden. A 58-year-old female with stage IV lung adenocarcinoma and a good baseline performance status (ECOG 1) presented with progressive, life-limiting dyspnea (NYHA Class IV). A massive pericardial effusion was diagnosed, and an initial pericardiocentesis provided only transient relief, with severe symptoms recurring within 48 hours. Following a multidisciplinary discussion centered on the patient's goals of care, the decision was made to escalate to a definitive surgical procedure. A subxiphoid open pericardiostomy was performed, yielding hemorrhagic fluid and pericardial tissue that confirmed metastatic adenocarcinoma. The procedure resulted in complete, durable resolution of her symptoms. In conclusion, open pericardiostomy provides durable relief from the life-limiting dyspnea of MPE, a goal often unachievable with pericardiocentesis alone. For appropriately selected patients with advanced cancer, escalating to a definitive surgical procedure is not merely a treatment for effusion but a crucial intervention to restore function and dignity. This case underscores that for patients with recurrent MPE and adequate performance status, timely surgical intervention is a vital component of effective palliative care, maximizing quality of life.

1. Introduction

The pericardium is a fibroserous sac that envelops the heart, providing mechanical protection, preventing acute chamber over-distension, and anchoring it within the mediastinum.[1] The potential space between its visceral and parietal layers normally contains a physiologic quantity of lubricating fluid, typically 15 to 50 mL. An abnormal accumulation of fluid within this space, termed pericardial effusion, disrupts this delicate equilibrium. The clinical sequelae of an effusion are dictated less by its absolute volume and more by the rapidity of its accumulation.[2] Slow, chronic accumulation allows the pericardium to stretch and accommodate large volumes, whereas rapid accumulation can precipitate a steep rise in intrapericardial pressure, culminating in cardiac tamponade—a state of life-threatening circulatory collapse. Among the diverse etiologies of pericardial effusion, malignancy represents one of the most common and prognostically significant causes in modern clinical practice. Malignant pericardial effusion (MPE) is a pre-terminal event, a stark indicator of advanced, metastatic disease that signals a median survival measured in months, typically ranging from three to six.[3] This grim reality must serve as the lens through which all clinical decisions are

viewed. MPE complicates the course of 5-15% of all cancer patients, with lung cancer being the most frequent culprit, responsible for up to 50% of all cases.[4] The pathophysiology is driven by direct tumor invasion, lymphatic obstruction, or the seeding of malignant cells onto the pericardial surface.[5] These tumor deposits actively secrete vasculogenic and pro-inflammatory cytokines, increasing capillary permeability and promoting a protein-rich exudate, which is often hemorrhagic due to tumor neovascularity.

The clinical presentation of MPE is frequently insidious, with symptoms like dyspnea, cough, and chest pain that can be mistakenly attributed to the underlying cancer or its treatments.[6] The primary goal of intervention is palliative: to relieve the debilitating symptoms, prevent impending cardiac tamponade, and, most importantly, improve the patient's quality of remaining life.[7] The management of MPE thus resides at the complex intersection of cardiology, oncology, and palliative care. Every intervention carries a "treatment burden"—the physical and psychological cost of a procedure, hospitalization, and recovery—which must be carefully weighed against the potential benefit in a patient with a finite life expectancy. This creates a central clinical challenge: how to select an intervention that maximizes symptom relief while minimizing treatment burden in the final chapter of a patient's life. The standard initial intervention for a hemodynamically significant effusion is echo-guided pericardiocentesis.[8] This procedure offers immediate symptomatic relief and provides a fluid sample for definitive cytological diagnosis. However, its efficacy as a standalone treatment is severely hampered by high recurrence rates, ranging from 40% to 70%, as it fails to address the underlying fluid-producing pathology.[9] This predictable failure necessitates a clear, well-reasoned strategy for escalation to a more durable solution. Options include intrapericardial sclerotherapy, the placement of an indwelling pericardial catheter, or the surgical creation of a pericardial window (pericardiostomy).[10]

The aim of this case report is to meticulously document and analyze the clinical course of a patient with recurrent MPE secondary to metastatic lung adenocarcinoma, thereby illustrating the critical, nuanced decision-making process that necessitates an escalation of care. We present a real-world scenario where initial management with pericardiocentesis proved insufficient, compelling a transition to open pericardiostomy for definitive palliation. The novelty of this study lies in its detailed, narrative exploration of the therapeutic pivot point, framed within a patient-centered, palliative care context. By providing a comprehensive analysis of the indications, outcomes, and rationale—including performance status and goals of care—this report seeks to reinforce the clinical framework for managing this specific, high-risk patient population. We aim to highlight the paramount importance of early recognition of pericardiocentesis failure and the timely, judicious application of surgical intervention to prevent catastrophic hemodynamic consequences and maximize the quality of remaining life.

2. Case Presentation

The patient was a 58-year-old female with a recently diagnosed (two months prior) stage IV lung adenocarcinoma. She had a good baseline functional capacity with an Eastern Cooperative Oncology Group (ECOG) performance status of 1, indicating she was fully ambulatory and capable of light work. Figure 1 is structured to guide the clinician's understanding, moving from foundational demographics to the nuanced complexities of the patient's oncological and symptomatic state. Each panel is designed not merely as a repository of data, but as a building block in the construction of a patient-centered treatment strategy. The profile begins with Patient Demographics, establishing the fundamental identity of the individual at the heart of this case: a 58-year-old female. This initial data point immediately grounds the case in a relatable human context, moving beyond abstract pathology. The subsequent panel, Oncological History, presents the core diagnosis with stark clarity: Stage IV

Lung Adenocarcinoma. This is the central, immutable fact that governs the patient's prognosis and dictates the palliative nature of all subsequent interventions. The explicit mention of "Stage IV" is a critical anchor, immediately signaling advanced, incurable disease. The detailed listing of metastatic sites—spanning the mediastinal and supraclavicular lymph nodes as well as the thoracic and lumbar vertebrae—paints a vivid picture of a significant and widespread disease burden. This information is not just anatomical; it is profoundly prognostic, underscoring the systemic nature of the malignancy and the unlikelihood of long-term survival. Transitioning from the diagnosis to the patient's functional reality, the Performance Status panel is perhaps the most crucial element for surgical and interventional decision-making. The specification of an Eastern Cooperative Oncology Group (ECOG) score of 1 is a potent piece of data. It conveys that, despite her advanced cancer, the patient was fully ambulatory and capable of light activities prior to the onset of her acute symptoms. This detail is paramount because it serves as a primary justification for considering a more invasive, definitive procedure. A good performance status suggests that the patient possesses the physiological reserve to withstand the "treatment burden" of surgery and to benefit meaningfully from the resulting improvement in quality of life. The figure 1cthen thoughtfully integrates the Palliative Context & Goals. This section elevates the profile from a simple medical summary to a true patient-centered document. By explicitly stating that the "Goals of Care" were to "Maximize comfort, maintain functional independence, and enhance quality of life," the figure provides an ethical and humanistic framework for all clinical actions. It confirms that a shared decision-making process has occurred and that the patient's wishes are the guiding principle. This alignment of therapeutic intent with patient goals is the cornerstone of modern palliative medicine and is essential for justifying any intervention, particularly one as significant as a surgical pericardiostomy. Finally, the Presenting Complaints panel provides a compelling narrative of the patient's acute clinical decline. The list of symptoms—worsening dyspnea (quantified as NYHA Class IV), significant orthopnea, persistent cough, and right-sided pleuritic chest pain—articulates the severe symptomatic burden that precipitated her hospital admission. The quantification of her dyspnea as NYHA Class IV is a powerful descriptor, communicating a state of profound physical limitation where even the slightest exertion is intolerable. These symptoms represent the tangible, human cost of the underlying pathology and serve as the explicit target for the palliative intervention. This figure 1 is a masterful synthesis of objective medical data and patient-centered palliative principles. It tells a complete story, providing the essential information needed to understand not only what was wrong with the patient, but also who the patient was and why a specific, aggressive palliative strategy was both medically appropriate and ethically sound.

The patient presented to the emergency department with a one-week history of rapidly progressing dyspnea. Her symptoms had advanced to the point where she was unable to perform activities of daily living, such as bathing, and could not speak in complete sentences without pausing for breath, consistent with New York Heart Association (NYHA) Class IV symptoms. Figure 2 presents a concise yet comprehensive clinical snapshot of the patient at the moment of presentation, meticulously documenting the objective physiological data and the profound subjective symptom burden that necessitated urgent medical intervention. The top row of the figure is dedicated to the patient's vital signs, which serve as the foundational parameters of hemodynamic stability. The recorded Blood Pressure of 116/70 mmHg and Heart Rate of 95 bpm are critically important findings. They indicate that, despite the immense internal pressure being exerted by the massive pericardial effusion, the patient was maintaining a state of compensated circulation.

Figure 1. Summary of patient demographics, oncological history, and palliative context.

This absence of overt hypotension is a key feature that distinguishes a chronic, slowly accumulating effusion from an acute tamponade, where circulatory collapse is the defining characteristic. The normal temperature and respiratory rate further contextualize her stability. However, Figure 2 pivots dramatically with the central, highlighted panel: Symptom Burden. The quantification of her dyspnea as New York Heart Association (NYHA) Class IV is the most powerful piece of data presented. This is not a subtle finding; it is a declaration of severe, life-limiting functional impairment. NYHA Class IV denotes a state where symptoms are present even at rest, and any physical activity is impossible without extreme discomfort. This single metric transforms the clinical picture from one of stable vital signs to one of profound patient suffering, providing the undeniable impetus for intervention. The final two panels detail the Cardiovascular and Respiratory Examinations, offering the physical clues that corroborate the underlying pathology. The finding of "Distant heart sounds" is a classic, albeit non-specific, sign of a large pericardial effusion, caused by the insulating effect of the fluid between the heart and the stethoscope. Equally significant are the negative findings: the absence of jugular venous distention and pulsus paradoxus. This reinforces the initial assessment that the patient was not in a state of classic cardiac

tamponade, but rather suffering from the compressive effects of the effusion. The respiratory examination, noting "Diminished breath sounds" on the right side, points to the significant mass effect of the fluid-filled pericardial sac and the primary lung tumor, which were compressing the lung parenchyma and preventing adequate aeration. Figure 2 provides a powerful visual summary of the patient's clinical status. It juxtaposes the reassuring stability of the patient's vital signs against the alarming severity of her functional collapse, as defined by her NYHA Class IV status. It provides a clear, evidence-based rationale for the urgency of the situation, demonstrating that while the patient was not in immediate danger of hemodynamic collapse, she was in a state of extreme physical distress that demanded immediate and effective palliative intervention.

Figure 2. Initial clinical findings and symptom burden.

A comprehensive diagnostic workup was initiated to identify the cause of her severe symptoms. The definitive diagnosis was established by a transthoracic echocardiogram (TTE), which revealed a massive, circumferential effusion causing a "swinging heart" motion and demonstrating early diastolic collapse of the right ventricle—a specific indicator of significant hemodynamic compromise. Figure 3 provides a synthesized, multimodal overview of the diagnostic pathway undertaken to confirm the presence and

significance of the patient's pericardial effusion. This figure representation masterfully integrates findings from three distinct but complementary modalities—echocardiography, electrocardiography, and radiography—to construct a comprehensive and irrefutable diagnostic conclusion. At the heart of the assessment, and appropriately given prominence in the figure, is the Transthoracic Echocardiogram (TTE). This modality serves as the cornerstone for the diagnosis and hemodynamic evaluation of pericardial effusions. The sonographic findings were unequivocal. The identification of a "Massive, Circumferential Effusion" with an estimated volume exceeding one liter immediately quantified the scale of the pathology. This was further characterized by the classic "'Swinging Heart' Motion," a pathognomonic sign where the heart, untethered by the vast amount of surrounding fluid, oscillates freely within the pericardial sac. Most critically, the echocardiogram revealed "Early Diastolic Collapse of the Right Ventricle." This is not merely an anatomical observation but a profound physiological one; it is a direct visualization of the point at which intrapericardial pressure surpasses the filling pressure of the right-sided heart chambers, representing a critical step along the continuum towards overt cardiac tamponade and providing a definitive indication for urgent drainage. Complementing the anatomical and hemodynamic data from the echocardiogram are the electrical findings from the Electrocardiogram (ECG). The figure highlights two classic electrical correlates of a large pericardial effusion. "Low-Voltage QRS Complexes" result from the insulating effect of the pericardial fluid, which dampens the amplitude of the heart's electrical signals as they are transmitted to the surface electrodes. "Electrical Alternans," a beat-to-beat variation in the QRS complex amplitude, is a direct electrical manifestation of the swinging heart motion observed on the echocardiogram. As the heart swings forwards and backwards within the fluid, its electrical axis relative to the ECG leads changes, producing the characteristic alternating pattern. Finally, the Chest Radiograph (X-Ray) provides essential contextual evidence. The finding of "Marked Cardiomegaly" with a globular, "water bottle" shaped cardiac silhouette is the classic radiographic sign of a large, chronic pericardial effusion. Crucially, the radiograph also offered a glimpse into the underlying etiology, concurrently revealing the primary right upper lobe mass and bony metastatic lesions, strongly suggesting that the effusion was malignant in nature. Figure 3 expertly illustrates the concept of diagnostic synergy. While the echocardiogram provided the definitive diagnosis, the ECG and chest radiograph offered powerful corroborating evidence, creating a cohesive and compelling clinical picture that not only confirmed the presence of a massive, hemodynamically significant pericardial effusion but also strongly pointed towards its malignant origin, thereby setting the stage for the subsequent therapeutic interventions.

The patient's management followed a two-stage, escalation-of-care protocol, a clinical pathway detailed with procedural specifics and outcomes in Figure 4. Figure 4 provides a powerful and scientifically precise visual exposition of the therapeutic journey undertaken in this case, illustrating a critical decision pathway in the palliative management of malignant pericardial effusion. The figure is meticulously structured as a two-stage timeline, moving from a temporizing initial intervention to a definitive surgical solution. This visual representation serves not merely as a summary of events but as a scholarly argument, articulating the rationale, procedural details, and divergent outcomes that define the modern, evidence-based approach to this complex clinical challenge. The first panel, "Stage 1: Initial Intervention," is dedicated to the Pericardiocentesis. This section details the standard-of-care, first-line response to a hemodynamically significant effusion. The procedural details are concisely presented, noting the echo-guided, subxiphoid approach performed on Day 1 of admission, which yielded 800 mL of hemorrhagic fluid—a finding highly suggestive of malignancy. The rationale is clearly stated: this intervention was pursued for immediate hemodynamic stabilization and

to acquire a fluid sample for essential diagnostic analysis. However, the critical takeaway is encapsulated in the "Outcome" section, which unequivocally labels the procedure a "Procedural Failure." The figure highlights the core limitation of this modality, emphasizing that while it provided transient symptomatic relief, the patient's severe dyspnea recurred in less than 48 hours. This rapid reaccumulation is not presented as a complication but as the predictable natural history of the underlying pathology, thereby underscoring the insufficiency of pericardiocentesis as a standalone therapy. A prominent arrow guides the viewer to the second panel, "Stage 2: Definitive Palliation," signifying a necessary and deliberate Escalation of Care. This section focuses on the Open Pericardiostomy, the definitive surgical intervention. The procedural details are again outlined—a surgical subxiphoid approach performed on Day 3, draining an additional 700 mL of fluid. The rationale for this more invasive step is directly linked to the failure of the initial procedure, framing it as the logical next step to achieve long-term control. The outcome here is starkly contrasted with the first stage, labeled a "Palliative Success." The figure emphasizes that this intervention achieved a "complete and durable resolution of symptoms," a statement that speaks directly to the primary goal of palliative care. By restoring the patient's quality of life and enabling the resumption of systemic chemotherapy, the surgical window is positioned not just as a successful procedure, but as a critical enabler of the patient's broader oncological and personal goals.

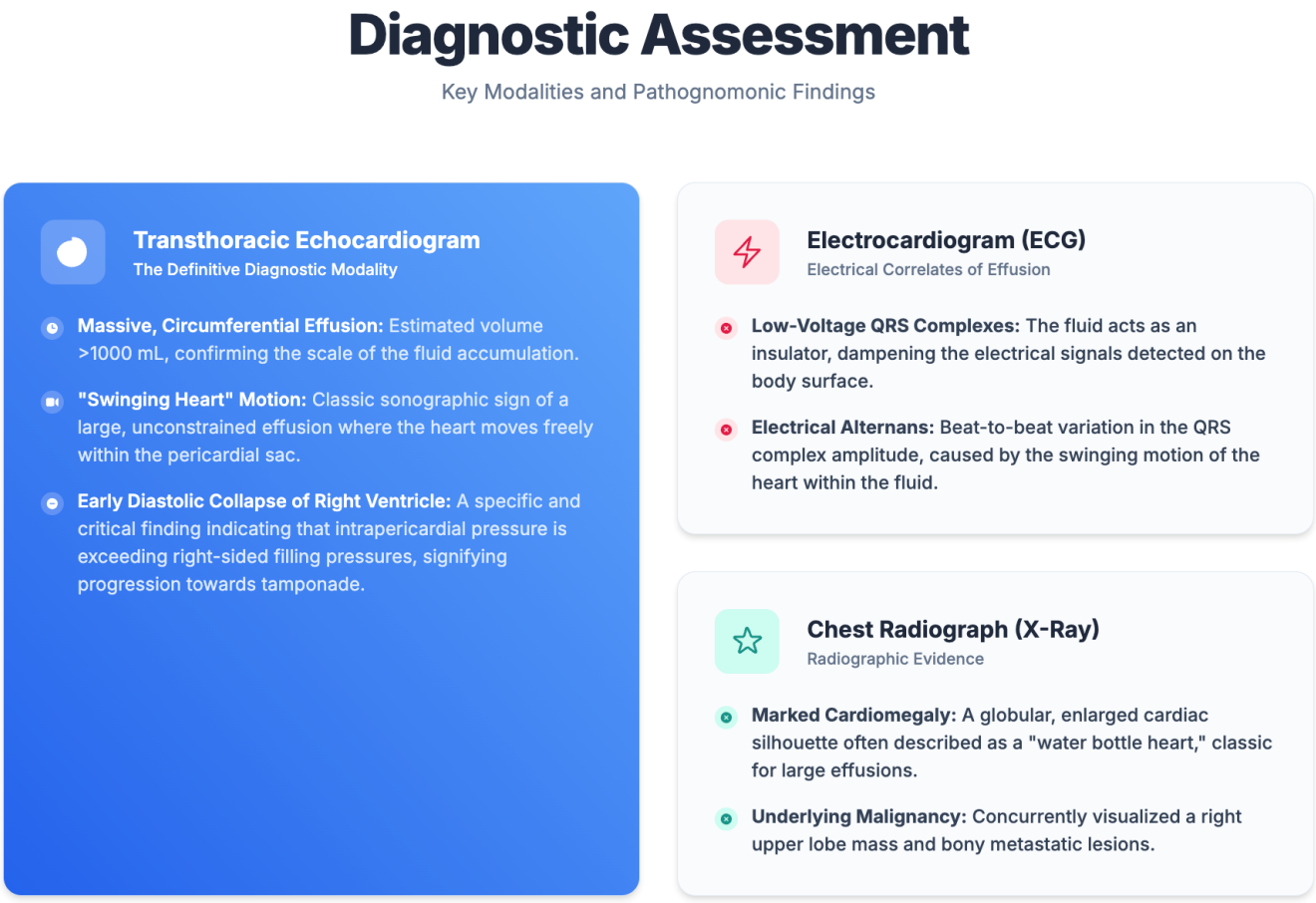

Figure 3. Summary of diagnostic assessments.

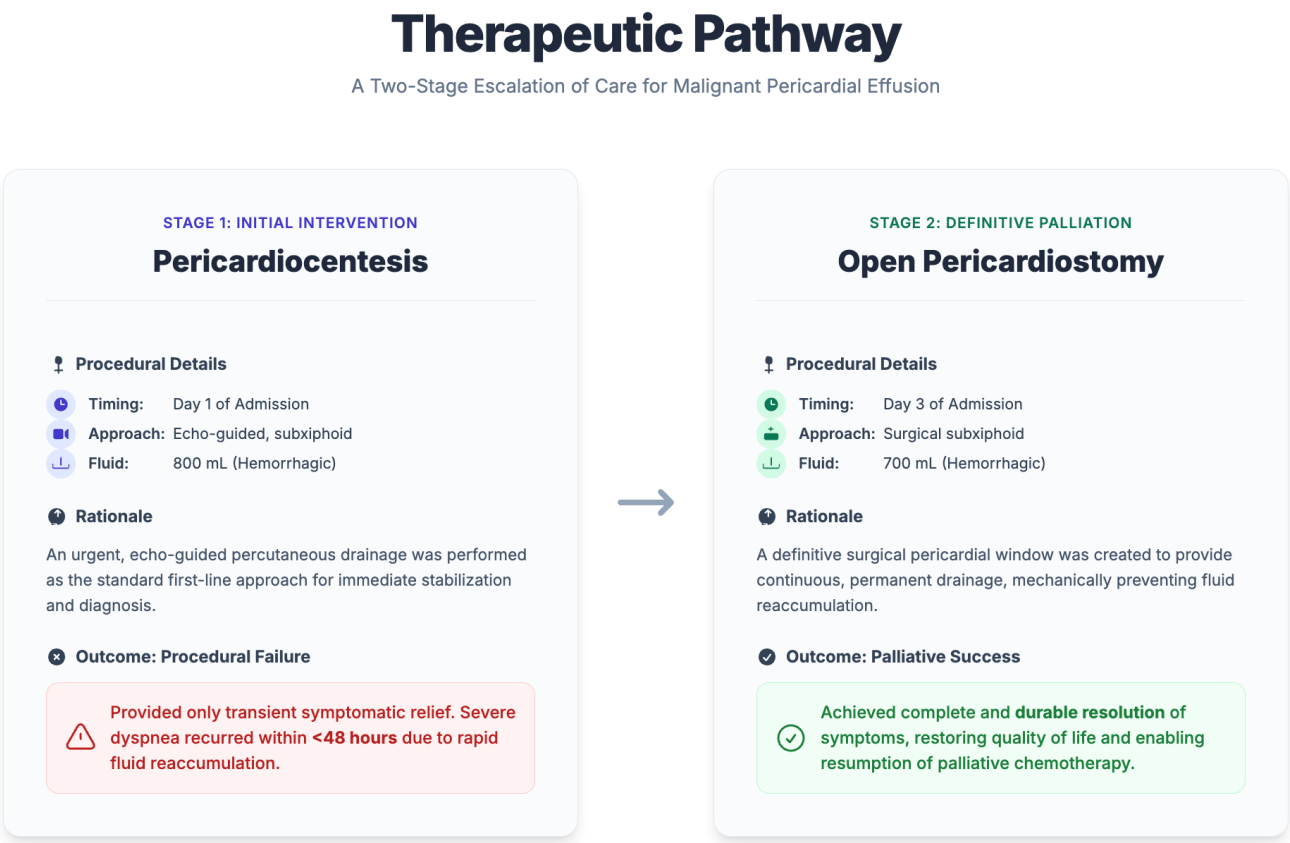

Figure 4. Therapeutic interventions and procedural details.

The postoperative course was uncomplicated, leading to a highly successful palliative outcome. The patient experienced a complete and durable resolution of her dyspnea and orthopnea, which was the primary goal of the intervention. The diagnostic analyses from the procedures confirmed the etiology of the effusion, and as detailed in Figure 5, the successful management of her effusion had a direct, positive impact on her broader oncological care and quality of life. She was discharged five days post-surgery with an improved performance status (ECOG 1) and was able to resume systemic palliative chemotherapy. At her two-month follow-up, she remained free of effusion-related symptoms, and a repeat echocardiogram showed only a trivial, hemodynamically insignificant residual effusion.

3. Discussion

This case report provides a detailed account of the successful management of a recurrent, massive malignant pericardial effusion in a patient with advanced lung adenocarcinoma. It highlights a critical inflection point in clinical decision-making: the imperative to escalate from a temporizing percutaneous procedure to a definitive surgical intervention for effective palliation. The patient's journey—from acute respiratory distress, through the ephemeral relief of pericardiocentesis, to the durable symptomatic control achieved with open pericardiostomy—offers a compelling clinical narrative. This narrative underscores several foundational principles in the modern management of MPE, rooted deeply in the underlying pathophysiology of the disease and the mechanistic rationale of the interventions employed.[11]

Figure 5. Follow-up and outcomes.

Figure 6 provides a comprehensive and scientifically grounded visualization of the complex pathophysiological cascade that culminates in malignant pericardial effusion (MPE), as exemplified by the case under discussion. This schematic is not merely illustrative; it is a conceptual model that deconstructs the multifaceted biological processes, translating them into a clear, four-stage narrative.[12] The central, anatomically inspired diagram of the heart serves as the focal point, around which the key pathogenic events are annotated, providing a clear visual anchor for understanding the disease process. The figure systematically elucidates how the insidious spread of malignancy transitions into a life-threatening state of hemodynamic compromise, offering a powerful educational tool for clinicians, trainees, and researchers. The cascade begins with the foundational event detailed in Annotation 1: Malignant Seeding. This initial step represents the arrival and colonization of the pericardial surfaces by metastatic adenocarcinoma cells. As depicted by the discrete malignant cell icons on the visceral pericardium, this process is the sine qua non of MPE. In the context of lung adenocarcinoma, these cells typically reach the pericardium through one of three primary routes: direct invasion from adjacent mediastinal tumors or lymph nodes, hematogenous dissemination via the coronary microcirculation, or retrograde lymphatic spread.[13] Once these cells adhere to the serosal lining, they proliferate to form micrometastases. These are not passive, inert deposits; they are biologically active colonies that commandeer the local microenvironment, setting the stage for the subsequent pathological events. This seeding fundamentally transforms the pericardium from a passive, protective sac into an active, tumor-involved organ. Following successful colonization, the process advances to the dual mechanisms that drive fluid accumulation, detailed in Annotation 2: Fluid Overproduction and Annotation 3: Impaired Drainage. These two processes occur in concert, creating a vicious cycle. As noted in Annotation 2, the malignant cells are potent factories for pro-inflammatory and vasculogenic cytokines, most notably vascular endothelial growth factor (VEGF). The secretion of VEGF into the pericardial space induces a state of

pathological hyperpermeability in the capillaries of the visceral and parietal pericardium. This leads to the constant, uncontrolled exudation of a protein-rich, plasma-like fluid into the pericardial cavity. The hemorrhagic nature of the effusion, a key finding in the clinical case, is also explained by this mechanism, as the tumor-induced neovasculature is often friable and prone to rupture. Simultaneously, as highlighted in Annotation 3, the growing tumor nodules and the associated inflammatory response create a physical obstruction of the delicate lymphatic channels responsible for draining the physiologic pericardial fluid. The schematic visually represents this with a blocked lymphatic channel, signifying a critical failure in the fluid resorption pathway. This dual assault—pathological overproduction combined with mechanical under-resorption—is the core engine of MPE, leading to a rapid and relentless increase in the volume of the effusion.[14] The inexorable accumulation of fluid leads directly to the final and most clinically significant stage of the cascade, detailed in Annotation 4: Cardiac Compression. The pericardial space, while capable of chronic expansion, is ultimately a fixed-volume compartment. As the effusion volume increases, the intrapericardial pressure rises, eventually equalling and then exceeding the filling pressures of the cardiac chambers. The schematic powerfully illustrates this concept, with the expansive red "Pericardial Effusion" layer visibly compressing the underlying "Myocardium" and "Chambers." The right ventricle, being a lower-pressure chamber, is the first to be affected. Its thin wall is unable to withstand the external pressure, leading to the pathognomonic sign of early diastolic collapse. This collapse impedes the normal filling of the right ventricle during diastole, which in turn reduces the preload delivered to the left ventricle. The ultimate hemodynamic consequence is a progressive reduction in stroke volume and cardiac output, leading to profound dyspnea and, if left unchecked, the circulatory collapse of cardiac tamponade.[15] Figure 6 synthesizes a complex sequence of molecular, cellular, and physiological events into a coherent and accessible visual narrative. It masterfully connects the microscopic event of malignant cell seeding to the macroscopic, life-threatening reality of cardiac compression. It underscores that MPE is not merely a collection of fluid but the end result of a dynamic and aggressive oncological process.

The pathophysiology of malignant pericardial effusion is a dynamic and aggressive process, a far cry from the passive fluid shifts seen in transudative effusions.[16] It is a direct consequence of the tumor's biological activity within the pericardial space. In lung adenocarcinoma, malignant cells gain access to the pericardium through several routes: contiguous spread from adjacent mediastinal lymph nodes, hematogenous seeding via the coronary circulation, or retrograde lymphatic invasion. Once these cells establish a foothold on the serosal surfaces, they proliferate into micrometastases that fundamentally disrupt the homeostatic balance of pericardial fluid. These tumor deposits are not inert; they are metabolically active, secreting a cocktail of potent signaling molecules. Key among these is vascular endothelial growth factor (VEGF), a powerful mediator of angiogenesis and vascular permeability. The overexpression of VEGF by adenocarcinoma cells leads to the formation of leaky, immature capillaries on the pericardial surface.[17] This neovascular network, coupled with the increased permeability of existing vessels, results in the constant exudation of a protein-rich, plasma-like fluid into the pericardial sac. Furthermore, the tumor cells and the host's inflammatory response release a cascade of cytokines, such as interleukins, which further amplify the inflammatory milieu and contribute to fluid production. Concurrently, the physical presence of tumor nodules, along with associated fibrinous material and cellular debris, systematically clogs the delicate lymphatic stomata responsible for draining fluid from the pericardial space. This combination of pathologically increased fluid production and mechanically impaired drainage creates a vicious, self-perpetuating cycle, leading to the relentless accumulation of effusion. The characteristic

hemorrhagic appearance of the fluid, as was observed in our patient, is a direct result of the fragile and poorly formed nature of the tumor-induced blood vessels, which are prone to rupture and spontaneous bleeding. This not only adds to the fluid volume but also intensifies the inflammatory response, further perpetuating the cycle of effusion.

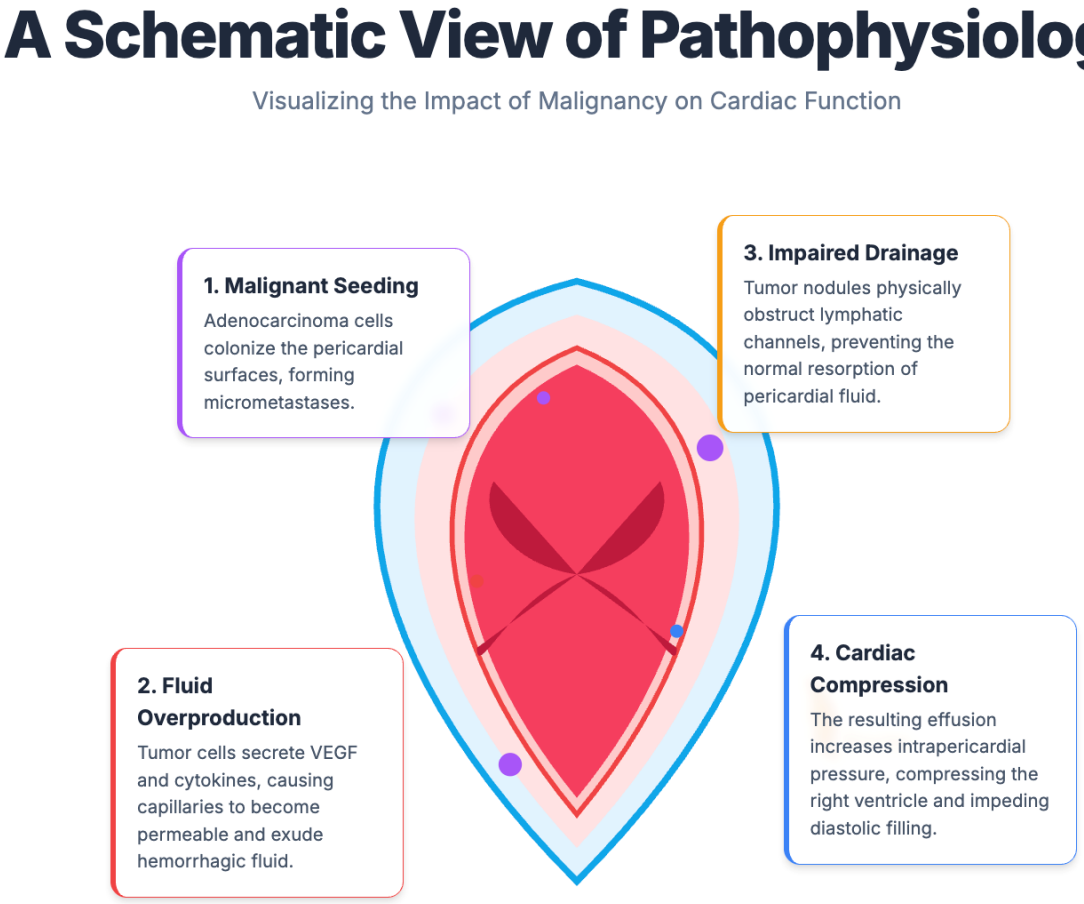

Figure 6. A schematic view of pathophysiology.

The clinical presentation of our patient, with a massive effusion but without the classic signs of acute cardiac tamponade, is a critically important physiological lesson.[18] The pericardium's response to accumulating fluid is governed by the principles of viscoelasticity and stress relaxation. Its fibrous, collagen-rich structure gives it a finite compliance, described by a steep pressure-volume curve. In an acute setting, such as trauma, the rapid influx of even a small amount of fluid (150-200 mL) can quickly exceed the pericardium's elastic limit, causing a precipitous rise in intrapericardial pressure that leads to tamponade. However, when the fluid accumulates insidiously over weeks or months, as is typical in MPE, the pericardium has time to adapt. Through a process of biological remodeling and mechanical creep, the collagen fibers gradually stretch and realign, allowing the sac to dilate significantly. This chronic adaptation shifts the pressure-volume curve to the right, enabling the pericardium to accommodate vast volumes of fluid—often more than a liter—before the intrapericardial pressure reaches the critical threshold that would compromise cardiac filling. This explains why our patient presented with compressive, rather

than constrictive, symptoms. Her severe dyspnea was not primarily caused by the hemodynamic failure of tamponade, but by the immense mass effect of the fluid-filled sac compressing adjacent structures, particularly the lungs, which reduced her vital capacity and led to a sensation of breathlessness. This distinction is paramount, as it clarifies that the trigger for intervention in such chronic, massive effusions is the profound symptomatic burden and the imminent risk of decompensation, not necessarily the presence of Beck's triad or pulsus paradoxus.[19]

The initial management with echo-guided pericardiocentesis was, without question, the correct first step. It is the cornerstone of initial MPE management, providing rapid, minimally invasive relief while simultaneously yielding a large fluid volume for definitive cytological diagnosis. It is both a therapeutic and diagnostic triumph in the acute setting. However, its fundamental, inherent flaw is that it is a purely ablative therapy. It removes the consequence of the disease (the fluid) but leaves the cause (the fluid-producing tumor) entirely untouched. With the malignant machinery on the pericardial surface still fully operational, fluid reaccumulation is not a risk but a near certainty. The recurrence of severe, life-limiting symptoms in our patient within 48 hours is a stark and classic demonstration of this principle. This rapid failure is not a complication of the procedure but an expected outcome based on the underlying pathophysiology. It is the definitive clinical signal that a temporizing measure has proven insufficient and that a durable, long-term palliative solution is required. Upon the failure of pericardiocentesis, the clinical team is faced with a crucial decision, and a careful consideration of the available therapeutic options is mandatory. Repeated pericardiocentesis is a strategy of diminishing returns, subjecting a palliative patient to recurrent procedures, hospitalizations, and the associated discomfort and risk, making it an untenable long-term plan.[20] Intrapericardial sclerotherapy, which involves instilling a chemical irritant like talc, bleomycin, or doxycycline to provoke an inflammatory response that fuses the visceral and parietal pericardial layers, is another option. However, its efficacy is variable, with success rates often lower than surgical methods. Furthermore, it can induce significant side effects, including severe chest pain, fever, and systemic inflammation, and its effectiveness is often compromised in the presence of hemorrhagic fluid or loculations, which can prevent uniform distribution of the sclerosing agent. A more modern and less invasive alternative is the placement of a long-term, tunneled indwelling pericardial catheter. This approach offers the significant advantage of outpatient management, allowing the patient or their family to drain the fluid intermittently at home, thereby reducing the burden of rehospitalization. However, it carries its own set of risks, including a persistent risk of infection, the potential for catheter tract metastasis, and the possibility of fluid loculation over time, which can render drainage ineffective.

This leads to the consideration of surgical pericardiostomy, or a pericardial window, which remains the gold standard for durable MPE management, with recurrence rates consistently reported below 5%. The procedure's efficacy lies in its straightforward and robust mechanical solution to the pathophysiological problem. By surgically excising a generous portion of the pericardium, a permanent, non-obstructable communication is created between the pericardial space and a larger serosal cavity—either the pleural space (via thoracoscopy or thoracotomy) or the peritoneal space (via a subxiphoid approach). This allows any fluid produced by the pericardial tumor to drain freely and continuously away from the heart, where it is easily resorbed by the vast surface area of the pleura or peritoneum. It effectively transforms the pericardium from a closed, high-pressure system into an open, low-pressure system, making recurrent tamponade a mechanical impossibility. The choice of the subxiphoid approach in our patient was particularly astute. It is less physiologically demanding than a formal thoracotomy or thoracoscopy, avoiding the need for single-lung ventilation, and is associated with less postoperative

pain and a quicker recovery.[19,20] It provides excellent surgical access to the anterior pericardium, allows for the creation of a large and durable window, and offers the added diagnostic benefit of obtaining a substantial tissue biopsy for definitive histopathological analysis. While fluid cytology is often positive in MPE, its sensitivity is not 100%, and a tissue diagnosis, as obtained in this case, provides the highest level of diagnostic certainty. The profoundly positive outcome in this case serves as a powerful testament to the value of a well-executed, patient-centered, and multidisciplinary treatment plan. The open pericardiostomy did not merely relieve a symptom; it restored the patient's quality of life. By eliminating the constant threat of suffocation, it enabled her to be discharged home, re-engage with her family, and, crucially, regain the functional status necessary to tolerate further systemic palliative chemotherapy. This underscores the symbiotic relationship between effective local palliation and systemic cancer care. The decision-making process was a model of interdisciplinary collaboration, involving cardiology for the initial diagnosis and intervention, oncology for providing the crucial prognostic context and goals of care, and cardiothoracic surgery for delivering the definitive palliative procedure. This integrated approach, which places the patient's overall well-being and stated wishes at the forefront of the therapeutic strategy, is the hallmark of modern, compassionate cardio-oncology and was the key to the success reported here.

4. Conclusion

This case report offers a definitive clinical narrative on the management of recurrent malignant pericardial effusion secondary to advanced lung adenocarcinoma. It powerfully demonstrates that while pericardiocentesis serves as an indispensable first-line intervention for immediate diagnosis and stabilization, its utility as a long-term solution is severely limited by the high probability of fluid reaccumulation. The rapid recurrence of symptoms in our patient acted as a clear and unambiguous trigger for a necessary escalation in therapeutic strategy. The subsequent implementation of an open subxiphoid pericardiostomy provided a robust, durable, and definitive palliative outcome, completely resolving the patient's debilitating respiratory distress and significantly enhancing her quality of life. The central conclusion drawn from this experience is a compelling clinical imperative: in patients diagnosed with recurrent malignant pericardial effusion who possess a reasonable performance status and a life expectancy that justifies a more invasive procedure, a timely and decisive transition to a surgical pericardial window should be considered the standard of care. This proactive approach stands as the most effective strategy to prevent the catastrophic consequences of cardiac tamponade and to provide meaningful, lasting palliation in a profoundly challenging patient population, thereby upholding the primary goal of palliative care: to improve the quality of life for patients and their families facing the problems associated with life-threatening illness.

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