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Evolving Tamponade Physiology Without Right Ventricular Collapse: The Case for Early Diagnostic and Therapeutic Pericardiocentesis

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Abstract

Cardiac tamponade is a clinical syndrome that may occur without classic echocardiographic right ventricular diastolic collapse. We describe a 48-year-old man with recent pneumococcal bacteremia, severe pulmonary disease, and a large purulent pericardial effusion. Formal echocardiography showed a circumferential effusion measuring up to 4.2 cm, inferior vena cava plethora with 11% inspiratory collapse, respiratory mitral inflow variation, hepatic venous expiratory flow reversal, and inspiratory septal shift, but no right-sided chamber compression. Estimated right atrial pressure was 20 mmHg, and pulmonary artery systolic pressure was 52 mmHg. The patient deteriorated until urgent pericardiocentesis with drainage of 800 mL of purulent fluid resulted in documented hemodynamic improvement and reduced vasopressor requirements. Pericardial Gram stain demonstrated heavy polymorphonuclear leukocytes without visible organisms, and culture showed no growth. This case illustrates the limitations of relying on chamber collapse alone and suggests a practical extended emergency physician point-of-care ultrasonography (POCUS) assessment incorporating inferior vena cava evaluation, right atrial motion, and respiratory septal movement, interpreted alongside clinical instability. This case also highlights tamponade as a hemodynamic continuum and supports early diagnostic and therapeutic pericardial drainage when physiologic compromise is present.

Categories: Cardiology, Emergency Medicine, Infectious Disease
Keywords: cardiac tamponade, echocardiography, hepatic vein doppler, pericardial effusion, pericardiocentesis, point-of-care ultrasound, pulmonary hypertension, purulent pericarditis

Introduction

Cardiac tamponade is a life-threatening hemodynamic syndrome resulting from impaired cardiac filling due to elevated intrapericardial pressure. Although right ventricular (RV) diastolic collapse is frequently described as a hallmark echocardiographic finding, tamponade represents a physiologic continuum, and the absence of an individual echocardiographic sign does not exclude clinically significant hemodynamic compromise [1-4].

A pericardial effusion is an anatomical finding, whereas clinical tamponade reflects pressure-dependent impairment of cardiac filling and circulation. Ventricular interdependence describes the reciprocal changes in ventricular filling imposed by a relatively fixed total cardiac volume; in tamponade, respiratory changes in right-sided filling can shift the interventricular septum and further compromise left ventricular filling [3,5,6].

Right-sided chamber collapse is dependent on the relationship between intracardiac and intrapericardial pressures. Consequently, elevated right-sided filling pressures may alter the pressure gradient responsible for chamber collapse and reduce the sensitivity of RV collapse as an isolated marker of tamponade [3,5,7]. Positive-pressure ventilation and changes in intrathoracic pressure can further modify ventricular filling and Doppler findings [8].

Purulent pericarditis is an uncommon but potentially life-threatening infection of the pericardial space. Although relatively rare in the antibiotic era, it remains associated with significant morbidity and mortality and may progress to cardiac tamponade, systemic infection, and constrictive pericardial disease [1,9-11]. Early drainage combined with appropriate antimicrobial therapy is central to management, with drainage also providing diagnostic material for microbiologic evaluation [1,9-11].

This case focuses on a diagnostic dilemma: severe pulmonary disease and elevated right-sided pressures coexisted with a large effusion and multiple supportive echocardiographic findings, yet classic chamber collapse was absent. We additionally discuss feasible bedside ultrasound observations for emergency physicians when advanced Doppler measurements are unavailable.

Case Presentation

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A 48-year-old man with substance use disorder, chronic hepatitis C, and a previously documented diagnosis of perimyocarditis presented with progressive dyspnea, hypoxemia, and altered mental status. Approximately two weeks earlier, he had left the hospital against medical advice with a mild pericardial effusion after treatment for *Streptococcus pneumoniae* bacteremia.

On arrival, his temperature was 36.8°C, blood pressure was 149/105 mmHg, heart rate was 97 beats/minute, respiratory rate was 25 breaths/minute, and oxygen saturation was 91% on a non-rebreather mask. Jugular venous distension was noted. Laboratory evaluation demonstrated leukocytosis and metabolic acidosis; lactate was 3.3 mmol/L and arterial blood gas pH was 7.30. Chest radiography revealed marked rightward mediastinal shift with near-total opacification of the left lung field (Figure *1*).

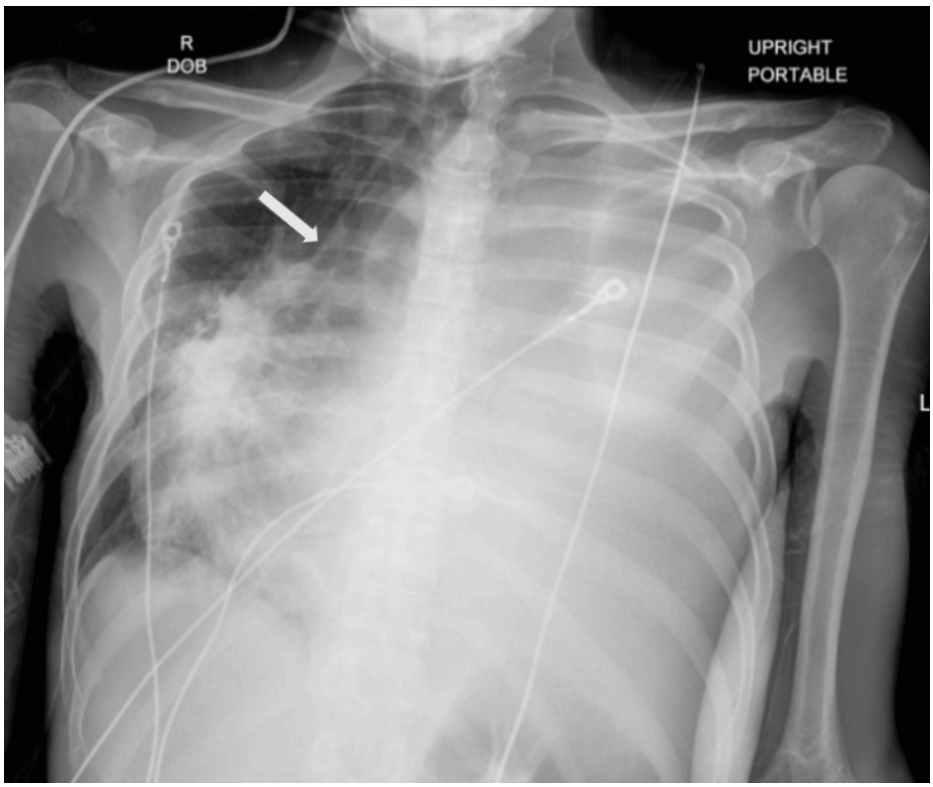


FIGURE 1: Chest X-ray

Initial portable anteroposterior chest radiograph demonstrating near-complete opacification of the left hemithorax with marked rightward tracheal deviation and an enlarged cardiac silhouette consistent with a large pericardial effusion. The arrow indicates the severely deviated trachea.

Emergency department point-of-care ultrasonography (POCUS) demonstrated a large pericardial effusion and inferior vena cava (IVC) plethora (Figure *2*). Formal transthoracic echocardiography demonstrated a large circumferential pericardial effusion measuring approximately 3.8-4.2 cm over the apical/inferolateral and anterolateral left ventricular regions and the RV free wall, and approximately 1.7 cm adjacent to the right atrium. The IVC was plethoric with 11% inspiratory collapse, yielding an echocardiographically estimated right atrial pressure of 20 mmHg. Additional findings included respiratory variation in mitral inflow velocities, expiratory diastolic hepatic venous flow reversal, and inspiratory right-to-left interventricular septal shift. Estimated pulmonary artery systolic pressure was 52 mmHg. The right atrium and RV were normal in size, with low-normal RV systolic function; left ventricular ejection fraction was approximately 50% with mild global hypokinesis. The formal report stated that there was no chamber compression and warned that high venous pressure reduced the sensitivity of chamber-collapse findings, with possible early tamponade and a recommendation for pericardiocentesis. Right atrial pressure was estimated from IVC findings rather than measured invasively, and pulmonary artery systolic pressure was also an echocardiographic estimate.

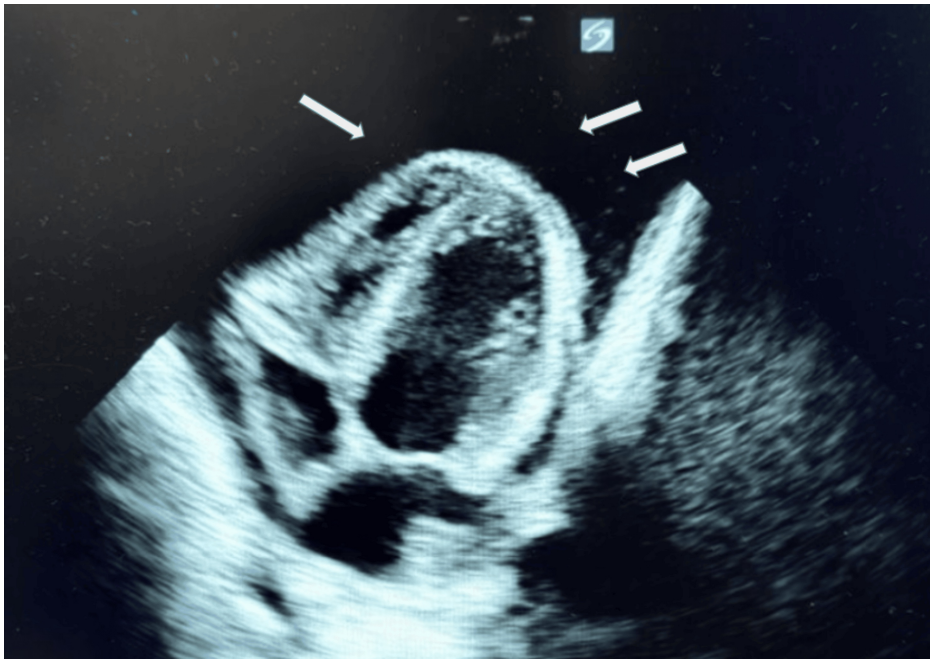


FIGURE 2: POCUS

Parasternal long-axis view point-of-care cardiac ultrasound (POCUS) demonstrating a large circumferential pericardial effusion surrounding the heart at the time of emergency department evaluation. The arrows indicate the hypoechoic area of the pericardial effusion.

Concurrent near-complete left lung opacification and severe hypoxemia provided a competing pulmonary explanation for the patient's deterioration, while absent chamber collapse and elevated right-sided pressures complicated interpretation of the pericardial findings. Drainage was not undertaken at the initial assessment because the severe concurrent pulmonary process provided an alternative explanation for the patient's deterioration, and the absence of end-diastolic RV collapse complicated interpretation of the large pericardial effusion. The patient subsequently required endotracheal intubation and mechanical ventilation. Pulmonology was consulted for bronchoscopy because of worsening oxygenation despite mechanical ventilation and no obvious airway obstruction.

During this time course, nursing documentation recorded heart rates approaching 190 beats/minute with runs described as ventricular tachycardia, accompanied by hypotension. Defibrillator pads were placed, and vasopressor therapy was initiated. Because of continued deterioration despite bronchoscopy, fluid resuscitation, and escalating vasopressor requirements, pericardiocentesis was subsequently performed.

Pericardiocentesis yielded approximately 800 mL of grossly purulent fluid, with documented improvement in hemodynamics and decreased vasopressor requirements following drainage. Exact paired pre- and post-procedure blood pressure, heart rate, oxygenation, and vasopressor doses could not be reliably established from the available record extracts and therefore are not reported as numerical treatment effects.

Pericardial fluid collected September 8, 2025, demonstrated heavy polymorphonuclear leukocytes on Gram stain, no organisms seen, and no growth on culture. Blood cultures from the preceding August admission showed *S. pneumoniae* bacteremia. A subsequent pneumococcal urine antigen was positive, but this does not establish pericardial infection by that organism. Initial blood cultures during the current presentation yielded viridans-group streptococci (*S. mitis, S. oralis, and S. parasanguinis*), and a later culture yielded *Staphylococcus hominis*. These organisms were not used to assign a specific microbiologic etiology to the pericardial infection. The patient was initially treated with broad-spectrum antimicrobial therapy and later required surgical drainage because of reaccumulation. He ultimately stabilized after source control and continued antimicrobial therapy.

Discussion

Tamponade as a hemodynamic continuum

Cardiac tamponade occurs when increased intrapericardial pressure impairs cardiac filling, resulting in reduced ventricular preload and potentially decreased cardiac output [1,3,5]. Classic echocardiographic findings include right atrial systolic collapse, RV diastolic collapse, plethoric IVC, and respiratory variation in transvalvular inflow velocities [1-4,6,8,12].

Echocardiographic signs of increased intrapericardial pressure should be distinguished from the clinical syndrome of cardiac tamponade. In this case, the combination of progressive hemodynamic deterioration requiring vasopressor support, a large circumferential pericardial effusion with multiple findings of impaired filling, and documented hemodynamic improvement with decreased vasopressor requirements after removal of 800 mL of pericardial fluid supported cardiac tamponade with obstructive shock. Concurrent pulmonary infection, respiratory failure, and systemic infection likely contributed to the patient's overall critical illness but do not exclude an obstructive component of shock from tamponade.

However, echocardiography should be interpreted in conjunction with the patient's clinical and hemodynamic state rather than as a checklist requiring the presence of a single finding. The echocardiographic literature demonstrates substantial variability in the reported sensitivity and specificity of right atrial and RV collapse [13]. Similarly, a modern emergency medicine review emphasizes that pericardial tamponade is neither solely a clinical nor solely an echocardiographic diagnosis and that multiple findings should be integrated with the clinical presentation [8].

Importantly, a combined echocardiographic and hemodynamic study by Singh et al. demonstrated that RV and right atrial collapse were not universally present in patients with tamponade. In one patient with elevated right-heart pressures and RV hypertrophy, hemodynamic evidence of tamponade was present despite the absence of both RV and right atrial collapse [7]. This observation is particularly relevant to the present case, in which estimated right atrial pressure was 20 mmHg and pulmonary artery systolic pressure was 52 mmHg.

When right-sided filling pressures are elevated, the pressure gradient required to produce right-sided chamber collapse may be altered, potentially reducing the sensitivity of chamber collapse as an isolated marker of tamponade [3,5,7]. Thus, in this patient, the absence of RV diastolic collapse did not exclude clinically significant pericardial constraint. Rather, the elevated right-sided pressures provide a physiologically plausible explanation for why classic right-sided chamber collapse was absent despite clinically significant tamponade.

Echocardiographic markers beyond right ventricular collapse

Multiple echocardiographic findings supported tamponade physiology in this case. A plethoric IVC with minimal respiratory variation is associated with elevated right atrial pressure and is a sensitive echocardiographic marker of tamponade [1,2,8,12]. Respiratory variation in mitral inflow velocities reflects exaggerated ventricular interdependence associated with pericardial constraint [4,8,12]. Inspiratory interventricular septal shift provides an additional indication of altered ventricular filling dynamics [6].

The IVC-derived estimated right atrial pressure and IVC plethora should not be regarded as independent findings because the pressure estimate is derived in part from IVC characteristics. Nevertheless, the markedly reduced IVC inspiratory collapse of 11% provided important evidence of elevated systemic venous pressure when interpreted with the remainder of the examination.

The echocardiographic literature provides particular support for integrating these findings rather than relying on chamber collapse alone. Appleton et al. demonstrated increased respiratory variation in transvalvular flow velocities in patients with cardiac tamponade and evaluated changes before and after pericardiocentesis [4]. A separate echocardiographic review described exaggerated respiratory variation in transvalvular flow and characteristic expiratory hepatic venous flow abnormalities in patients with tamponade [14].

Of particular importance in this patient was expiratory diastolic flow reversal in the hepatic veins. Hepatic venous Doppler abnormalities, particularly expiratory increases in reverse flow, have been described in cardiac tamponade and reflect abnormal right-sided filling associated with ventricular interdependence and pericardial constraint [6,14].

These Doppler findings are supportive rather than individually diagnostic. Respiratory transvalvular variation and hepatic venous flow can be altered by pulmonary hypertension, abnormal intrathoracic pressures, mechanical ventilation, and changing loading conditions [8,14]. Their significance in this case therefore derives from their occurrence in conjunction with the large circumferential effusion, IVC plethora, inspiratory septal shift, clinical deterioration, and subsequent hemodynamic response to drainage.

The constellation of findings in this patient, including a large circumferential pericardial effusion, elevated right atrial pressure, plethoric IVC, respiratory mitral inflow variation, inspiratory septal shift, and hepatic vein expiratory flow reversal, provided multiple indicators of impaired ventricular filling concerning for cardiac tamponade despite the absence of RV diastolic collapse.

A practical POCUS approach for emergency physicians

This case also raises a practical question for emergency physicians: when a large pericardial effusion is

identified on POCUS, but RV diastolic collapse is absent, what additional bedside findings can be sought without relying on advanced Doppler measurements?

Emergency physicians can extend the standard cardiac POCUS examination by assessing IVC size and respiratory variation, observing the right atrial free wall for systolic collapse, and evaluating the interventricular septum over several respiratory cycles for respiratory septal shift or exaggerated ventricular interdependence [6,12]. These observations can be obtained using standard two-dimensional cardiac and IVC views and may therefore be more practical during emergency resuscitation than spectral Doppler measurements of mitral inflow or hepatic venous flow.

In the present case, emergency department POCUS identified the large pericardial effusion and IVC plethora, while subsequent formal echocardiography demonstrated inspiratory interventricular septal shift in addition to the more advanced Doppler findings. Right atrial systolic collapse was not demonstrated. This suggests a practical bedside approach in which absence of RV diastolic collapse prompts additional assessment rather than terminating the sonographic evaluation.

None of these findings should be used as an isolated indication for pericardiocentesis, nor do we propose a diagnostic POCUS score. Rather, concordant bedside findings should be integrated with the patient's clinical trajectory and hemodynamic status. In a deteriorating patient with a large effusion, IVC plethora and respiratory septal movement may provide additional readily obtainable evidence of pericardial constraint while formal echocardiography and definitive management are pursued [6,12].

Confounding factors in diagnosis

The echocardiographic appearance of tamponade can be influenced by loading conditions and intrathoracic pressure. Elevated right-sided filling pressures may reduce the likelihood of right-sided chamber collapse despite clinically important pericardial constraint [5,7]. Positive-pressure ventilation can also alter intrathoracic pressure, ventricular loading conditions, and cardiac output, complicating interpretation of respiratory Doppler findings [8].

Pulmonary hypertension was particularly important in this patient. With an estimated pulmonary artery systolic pressure of 52 mmHg and echocardiographically estimated right atrial pressure of 20 mmHg, the pressure within the right-sided chambers may have remained sufficiently high to resist external compression. Because chamber collapse occurs when pericardial pressure transiently exceeds intracavitary pressure, elevated right-sided pressures can mask the classic right atrial or RV collapse expected in tamponade [3,5,7].

The coexistence of pulmonary and pericardial pathology further complicated interpretation in this patient. The near-complete opacification of the left hemithorax raised concern for a pulmonary or pleural process that could have contributed to respiratory failure. Bronchoscopy subsequently demonstrated patent airways without obstructive pathology. The patient also subsequently required positive-pressure ventilation, which can decrease venous return and alter the respiratory variation used to assess tamponade [8].

These pulmonary and infectious processes provided legitimate competing explanations for portions of the patient's respiratory and hemodynamic deterioration and contributed to the initial diagnostic uncertainty. However, they do not negate the evidence for obstructive shock. Continued deterioration despite respiratory interventions, followed by documented hemodynamic improvement and decreased vasopressor requirements after removal of approximately 800 mL of pericardial fluid, strongly supports cardiac tamponade as a clinically important driver of the patient's shock.

These competing possibilities emphasize the importance of integrating the clinical course with echocardiographic findings. In an unstable patient, absence of one classic echocardiographic feature should not necessarily outweigh a constellation of findings indicating impaired cardiac filling.

Diagnostic and therapeutic role of pericardiocentesis

Pericardial drainage serves both diagnostic and therapeutic roles. Current European Society of Cardiology (ESC) guidance emphasizes urgent drainage in patients with hemodynamic compromise attributable to cardiac tamponade [1]. Pericardial drainage also provides fluid for microbiologic and biochemical evaluation when bacterial or purulent pericarditis is suspected [1,9,10].

The 2025 ESC Guidelines specifically recognize pericardial drainage as the treatment for cardiac tamponade and emphasize that drainage should not be delayed in unstable patients [1]. This is consistent with prior ESC guidance and with contemporary emergency medicine literature describing hemodynamic instability or impending deterioration as indications for emergent pericardiocentesis [2,8,12].

The hemodynamic literature further supports an integrated approach. Kearns et al. emphasize that echocardiography is central to the evaluation of tamponade but is insufficient by itself to establish the

physiologic diagnosis of hemodynamically significant tamponade [5]. The diagnosis instead requires synthesis of clinical evidence of impaired cardiac output, elevated filling pressures, sympathetic activation, and recognition of alternative contributors to shock [5].

In this case, progressive hemodynamic deterioration requiring vasopressor support, a large circumferential effusion, elevated right-sided filling pressure, and multiple echocardiographic findings of impaired filling supported clinically significant cardiac tamponade despite absent chamber collapse. Most importantly, emergency physician and nursing documentation described improved hemodynamics and decreased vasopressor requirements following drainage of approximately 800 mL of pericardial fluid. Although invasive opening and closing intrapericardial pressures were not obtained and exact paired pre- and post-procedure measurements could not be reconstructed, the clinical response to decompression provides additional evidence that pericardial constraint was producing an important obstructive component of shock.

This case cannot establish that earlier drainage would have prevented intubation or subsequent deterioration. However, it demonstrates why waiting for RV diastolic collapse before intervening in a patient with otherwise compelling clinical evidence of tamponade may be inappropriate. The indication for drainage should arise from the overall evidence of hemodynamic compromise rather than effusion size or any single echocardiographic finding alone.

Implications in purulent pericarditis

Purulent pericarditis is an uncommon but potentially life-threatening condition in the antibiotic era. Delayed source control increases the risk of persistent infection, septic shock, tamponade, and pericardial fibrosis. Sagristà-Sauleda et al. reviewed 33 cases of purulent pericarditis over 20 years and demonstrated that pneumonia was a common source of infection and that tamponade and constrictive pericarditis were important complications [9]. Parikh et al. similarly emphasized the rarity but serious nature of purulent pericarditis in the modern antibiotic era [10].

Pneumococcal infection remains a recognized cause. Hästbacka et al. described life-threatening purulent pericarditis complicating pneumococcal disease despite antimicrobial treatment and emphasized the importance of early detection and treatment [11]. This is relevant to the present case because *S. pneumoniae* bacteremia was microbiologically confirmed during the preceding hospitalization, followed approximately two weeks later by a grossly purulent pericardial effusion. However, the pericardial fluid Gram stain demonstrated heavy polymorphonuclear leukocytes without visible organisms, and the pericardial culture showed no growth. Therefore, pneumococcal involvement of the pericardium remains clinically suspected rather than microbiologically proven. The subsequent positive pneumococcal urinary antigen supports recent pneumococcal infection but does not localize infection to the pericardial space.

Prompt antimicrobial therapy and drainage are central to management of purulent pericarditis [1,9-11]. In addition to relieving pericardial pressure, drainage provides diagnostic material and achieves source control. The 2025 ESC Guidelines emphasize complete drainage of purulent effusions and support surgical drainage when necessary to achieve adequate source control and reduce complications [1]. Thus, surgical drainage should not be viewed solely as rescue therapy after failed pericardiocentesis. In this patient, initial percutaneous drainage provided urgent hemodynamic decompression, while subsequent surgical drainage addressed reaccumulation and provided more definitive source control.

The combination of recent pneumococcal bacteremia, grossly purulent pericardial fluid, persistent large effusion, and progressive hemodynamic instability therefore represented a particularly high-risk clinical scenario in which pericardial drainage had both diagnostic and therapeutic importance.

Lessons for emergency medicine

Emergency physicians frequently evaluate undifferentiated shock in which multiple processes coexist. In patients with a large pericardial effusion, the absence of RV diastolic collapse should not be interpreted in isolation as excluding tamponade, particularly when elevated right-sided pressures may mask chamber compression [3,5,7,13]. Bedside assessment of IVC plethora, right atrial systolic motion, and respiratory interventricular septal movement can supplement standard cardiac POCUS when advanced Doppler is unavailable [6,12]. These findings should be integrated with the patient's hemodynamic trajectory rather than used as isolated criteria for drainage. When pericardiocentesis is indicated, it provides therapeutic decompression and diagnostic sampling and, in purulent pericarditis, contributes to source control [1,9,10].

Conclusions

This case illustrates that cardiac tamponade with obstructive shock may occur without RV diastolic collapse, particularly when elevated right-sided pressures reduce the likelihood of chamber compression. In this patient, a large circumferential effusion, IVC plethora, respiratory mitral inflow variation, hepatic venous expiratory flow reversal, inspiratory septal shift, progressive hemodynamic instability, and documented improvement following drainage of approximately 800 mL of purulent fluid supported tamponade despite absent RV collapse.

For emergency physicians, absence of RV diastolic collapse should prompt continued bedside assessment rather than diagnostic closure. IVC plethora, right atrial systolic motion, and respiratory interventricular septal movement can provide additional information using standard POCUS views when advanced Doppler is unavailable. These findings should be interpreted with the patient's clinical and hemodynamic condition rather than as isolated indications for drainage. In patients with hemodynamic compromise or suspected purulent pericarditis, absence of RV diastolic collapse should not delay appropriate pericardial drainage.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Colby W. McWhorter

Acquisition, analysis, or interpretation of data: Colby W. McWhorter, Timothy R. Havens

Drafting of the manuscript: Colby W. McWhorter

Critical review of the manuscript for important intellectual content: Colby W. McWhorter, Timothy R. Havens

Supervision: Timothy R. Havens

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Saint Louis University Review Board issued approval N/A. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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