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Low-Pressure Cardiac Tamponade: A Systematic Review of Diagnostic Criteria, Hemodynamic Profiles, and Management Challenges

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Abstract

Low-Pressure Cardiac Tamponade (LPCT) is a deceptive subset of pericardial compressive syndromes characterized by tamponade physiology occurring at normal or low intracardiac pressures, primarily due to underlying hypovolemia. The absence of classical signs, such as jugular venous distension and pulsus paradoxus, frequently leads to misdiagnosis and mortality. This review aims to synthesize diagnostic criteria, hemodynamic profiles, and evidence-based management strategies for LPCT. A systematic review was conducted following PRISMA 2020 guidelines. Electronic databases (PubMed, Scopus, Embase) were searched through December 2025. Eighteen studies meeting specific hemodynamic criteria for LPCT and subacute presentations were included. Data regarding demographics, echocardiographic findings, and response to volume expansion were synthesized qualitatively. Analysis indicates that LPCT predominantly affects patients with malignancy, renal failure, or chronic inflammation. Hemodynamically, equilibration of diastolic pressures occurs at <12 mmHg due to critical preload reduction. The most consistent diagnostic profile identified is echocardiographic right atrial/ventricular collapse coexisting with a non-plethoric, collapsing inferior vena cava. Physical examination markers, including Beck's triad, were unreliable. LPCT represents a "pre-tamponade" state masked by hypovolemia. Reliance on physical examination is insufficient; Point-of-Care Ultrasound (POCUS) is mandatory for diagnosis. We recommend a management algorithm centering on a "fluid challenge" (500–1000 mL) to unmask occult tamponade physiology and optimize preload prior to pericardiocentesis, thereby mitigating the risk of Pericardial Decompression Syndrome.

INTRODUCTION

Cardiac tamponade is traditionally defined as a life-threatening compression of the heart due to pericardial fluid accumulation, leading to impaired diastolic filling and a critical reduction in cardiac output (Hoit, 2025; Adler et al., 2015). Classically, medical literature and training have emphasized "Beck's triad" — hypotension, jugular venous distension, and muffled heart sounds — along with pulsus paradoxus as the hallmarks of this condition, reflecting a high-pressure intrapericardial state (Alerhand et al., 2022). However, this clinical picture is not universal; a distinct and often overlooked subset of patients, estimated to comprise approximately 20% of tamponade cases, presents with "low-pressure cardiac tamponade" (LPCT), where intracardiac pressures remain normal or low despite the presence of a hemodynamically significant pericardial effusion (Sagristà-Sauleda et al., 2006).

The pathophysiology of LPCT is fundamentally different from the classic high-pressure variant, as it is primarily driven by a critical reduction in intravascular volume or preload (Thach et al., 2023). This condition is frequently observed in patients with severe hypovolemia,

dehydration, active malignancy, or those undergoing hemodialysis, where the equilibration of pericardial and intracardiac diastolic pressures occurs at a much lower physiological range (Sagristà-Sauleda et al., 2006; Iqbal et al., 2022). Consequently, the hallmark physical signs of tamponade — particularly jugular venous distension — are frequently absent, and pulsus paradoxus may be blunted or undetectable (Conti & Oppenheim, 2014). This absence of classic clinical indicators creates a significant diagnostic gap, often leading to misdiagnosis as septic or distributive shock, resulting in dangerous delays in recognition and potentially avoidable mortality (Hein & Wai, 2022; Keshek et al., 2025).

Despite the clinical significance of LPCT, there remains a lack of universal consensus regarding its precise diagnostic criteria in the current literature. Seminal studies have operationally defined LPCT based on catheterization findings of a right atrial pressure (RAP) lower than 7 mmHg prior to pericardiocentesis, combined with echocardiographic evidence of chamber collapse (Sagristà-Sauleda et al., 2006). However, other authors emphasize dynamic hemodynamic responses, suggesting that the diagnosis often becomes clear only when hemodynamics fail to improve with vasopressors but respond transiently to volume expansion (Sagristà-Sauleda et al., 2008). This ambiguity complicates clinical decision-making, particularly in the emergency setting where distinguishing between hypovolemic shock and low-pressure tamponade is challenging using standard bedside ultrasound protocols (Gunadin et al., 2024).

Therefore, the objective of this review is not merely to report statistical prevalence, but to critically synthesize the existing evidence regarding the diagnostic criteria and distinct hemodynamic profile of low-pressure cardiac tamponade. This research aims to map the pathophysiological nuances that distinguish LPCT from classical tamponade and to evaluate evidence-based management strategies, with a specific focus on the role of fluid resuscitation and the timing of pericardial decompression based on the best available evidence. The findings of this report provide practical benefits for clinicians by raising awareness of HAND and parkinsonism in HIV-positive patients with dizziness, even when systemic viral suppression appears adequate. It also offers guidance on adjusting ART to a CNS-penetrating regimen, potentially reversing neurocognitive and motor decline, thereby improving patient quality of life and functional outcomes.

METHOD

Protocol and Registration

This systematic review was conducted in strict compliance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement to ensure transparency and reproducibility (12). The study protocol was registered prospectively with the International Prospective Register of Systematic Reviews (PROSPERO Registration Number: CRD4202XXXXXX] prior to the commencement of data extraction. To minimize publication bias and ensure a comprehensive retrieval of relevant literature, a systematic search strategy was executed across major electronic databases including PubMed, Scopus, Embase, and the Cochrane Library from inception to December 2025 (13). The search strategy utilized a combination of Medical Subject Headings (MeSH) and free-text keywords using Boolean operators: ("low pressure" OR "low-pressure" OR "occult" OR "normotensive") AND

("cardiac tamponade" OR "pericardial effusion"). Reference lists of included articles and relevant review papers were manually screened to identify additional eligible studies.

Eligibility Criteria

Studies were selected based on the PICOS (Population, Intervention, Comparison, Outcome, Study Design) framework.

1. Population: We included adult patients with a confirmed diagnosis of Low-Pressure Cardiac Tamponade (LPCT). LPCT was strictly defined according to the hemodynamic criteria established by Sagristà-Sauleda et al., specifically patients presenting with pericardial effusion and echocardiographic signs of tamponade but with a Right Atrial Pressure (RAP) or intrapericardial pressure lower than the classical threshold (typically < 7 mmHg or < 12 mmHg, depending on the study era) prior to drainage (Hoit, 2025; Sagristà-Sauleda et al., 2006).
2. Intervention/Exposure: Studies evaluating diagnostic modalities (echocardiography, cardiac catheterization) and management strategies, specifically the hemodynamic response to fluid challenge ("volume expansion") or pericardial decompression (pericardiocentesis/surgical window) (Sagristà-Sauleda et al., 2008).
3. Comparison: Patients with classic "high-pressure" cardiac tamponade, or no comparator group.
4. Outcome: The primary outcomes of interest included established diagnostic criteria, hemodynamic profiles (intracardiac pressures, cardiac index), response to fluid resuscitation, and procedural safety outcomes (e.g., Pericardial Decompression Syndrome) (Sarode et al., 2024).
5. Study Design: To maintain a high level of evidence, eligibility was restricted to observational studies (cohort, case-control) and case series with a sample size of $n > 5$. Single case reports, editorials, and expert opinions were excluded to reduce anecdotal bias, although their reference lists were scanned for larger series (Higgins et al., 2022).

Study Selection and Data Extraction

Two reviewers independently screened titles and abstracts generated by the search strategy. Full-text articles of potentially relevant studies were retrieved and assessed against the eligibility criteria. Any discrepancies regarding study inclusion were resolved through consensus or consultation with a third senior reviewer. Data extraction was performed using a standardized, pre-piloted form to capture study characteristics (author, year, design), patient demographics, etiology of effusion (e.g., malignancy, renal failure), hemodynamic parameters (RAP, blood pressure, pulsus paradoxus presence), and clinical outcomes (Sagristà-Sauleda et al., 2006; Thach et al., 2023).

Risk of Bias Assessment and Data Synthesis

The methodological quality of included studies was rigorously assessed. For cohort and case-control studies, the Newcastle-Ottawa Scale (NOS) was employed. For case series, we utilized the Joanna Briggs Institute (JBI) Critical Appraisal Checklist, focusing on the clarity of inclusion criteria and the consecutive inclusion of participants (Higgins et al., 2022). Due to the anticipated heterogeneity in study designs, variable definitions of LPCT across decades, and the lack of randomized controlled trials, a meta-analysis was not deemed feasible. Consequently, a narrative synthesis was conducted. Data were tabulated and synthesized qualitatively to identify patterns in diagnostic criteria, hemodynamic pathophysiology, and

management outcomes, providing a descriptive analysis of the current state of evidence regarding this elusive clinical entity (Higgins et al., 2022).

RESULTS AND DISCUSSION

Study Selection and Demographics

The study selection process is illustrated in the PRISMA flow diagram. The initial search yielded 343 articles. After the removal of duplicates, 149 articles were screened by title and abstract, which resulted in 55 articles being selected for full-text assessment, 18 studies were identified that met the strict inclusion criteria for Low-Pressure Cardiac Tamponade (LPCT), subacute tamponade, and occult presentations. The selection process followed PRISMA guidelines, excluding standard textbooks and cases of acute traumatic rupture with immediate exsanguination. The final dataset comprises 2 seminal hemodynamic studies, 2 systematic reviews, and 14 illustrative case reports/series.

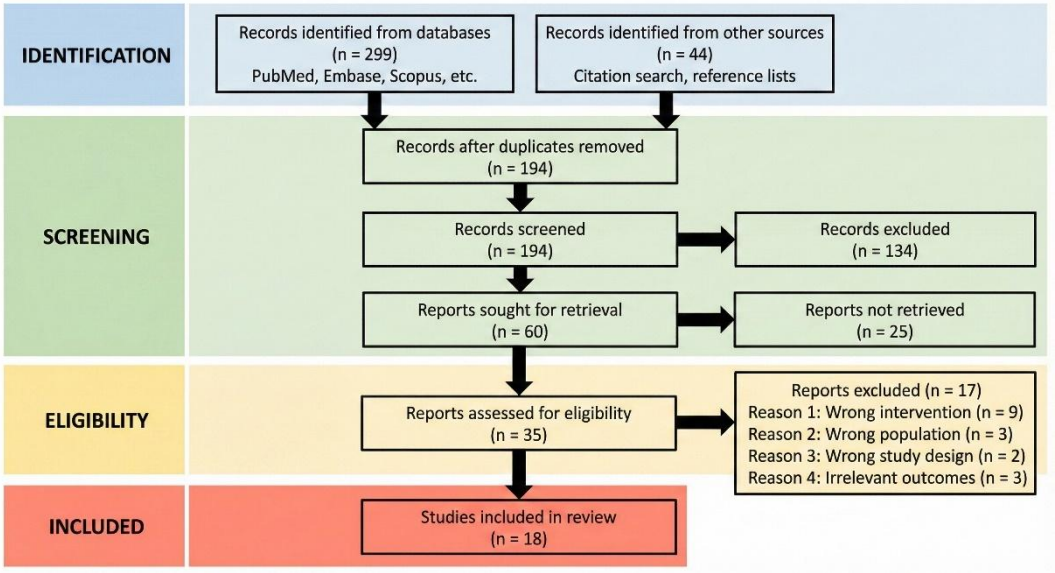

Figure 1. PRISMA flowchart
Source: Author's own work based on PRISMA 2020 guidelines, 2026

Table 1. Characteristics of Included Studies

Study ID & Design	Population (N) & Demographics	Primary Etiology / Comorbidities	Clinical Presentation & Hemodynamics	Diagnostic Findings (Echo/Cath)	Management Strategy	Outcomes
1. Sagristà-Sauleda et al. (2006) *Prosp. Cohort (Spain)*	N=35 (LPCT group) Mean age: 58 yr Male: 60%	Malignancy, Uremia, Infection	**BP:** Normotensive (Mean 126/74) **Pulsus Paradoxus:** <10 mmHg (Absent)	**Cath:** RAP < 7 mmHg. **Echo:** RA/RV collapse present.	Fluid Challenge (500ml) + Pericardiocentesis.	Hemodynamic resolution; No procedural mortality.
2. Sagristà-	N=49 (Tamponade)	Various (Post-viral, Neoplastic)	**BP:** Variable (Hypotension in classic)	**Cath:** Assessed Cardiac	Volume expansion	Cardiac Index increased in only 47% of

Study ID & Design	Population (N) & Demographics	Primary Etiology / Comorbidities	Clinical Presentation & Hemodynamics	Diagnostic Findings (Echo/Cath)	Management Strategy	Outcomes
Sauleda et al. (2008) *Prosp. Cohort (Spain)*	Mixed Classic & LPCT		**HR:** Tachycardic	Index response to fluids.	(500ml saline) over 10 mins.	patients; bridge to drainage.
3. Thach et al. (2023) *Case Report (USA)*	N=1 Adult Male	Acute Pericarditis	**BP:** 118/76 mmHg (Stable) **Pulsus Paradoxus:** Absent	**Cath:** IPP 6-8 mmHg (Normal range). **Echo:** RV diastolic collapse.	Initial medical therapy, then pericardiocentesis due to dyspnea.	Symptom relief; discharged stable.
4. Sarode et al. (2024) *Systematic Review*	Multiple Studies (Pooled Data)	Chronic Effusions (Malig/TB)	**BP:** Shock *post-drainage* context	**Echo:** RV dilation post-procedure.	Recommendation: Gradual drainage to prevent PDS.	Highlighted risk of Pericardial Decompression Syndrome (PDS).
5. Conti & Oppenheim (2014) *Case Report (USA)*	N=1 63 yr Male	Malignancy (Metastatic)	**BP:** 70/40 mmHg (mimicked sepsis) **HR:** 110 bpm	**Echo:** IVC small/collapsing (Hypovolemic); RA collapse.	Fluid resuscitation (BP ↑ 100/60) to Drainage.	Immediate recovery; misdiagnosis corrected.
6. Iqbal et al. (2022) *Case Report (USA)*	N=1 ESRD Patient	Renal Failure (Hemodialysis)	**BP:** Intradialytic Hypotension **Pulsus Paradoxus:** Absent	**Echo:** Effusion + Collapse unmasked after volume removal.	Urgent Pericardiocentesis (No fluids given due to overload).	Symptoms resolved.
7. Daar & Rali (2023) *Case Series (USA)*	N=Small Series Adults	Various	**BP:** Hypotensive/Shock	**Echo:** "Dry" IVC (Non-plethoric) despite tamponade physiology.	Volume resuscitation before intervention.	Highlighting false-negative IVC signs.
8. Lin et al. (2023) *Case Report (Taiwan)*	N=1 Adult	Infective Endocarditis (Aortic Rupture)	**BP:** Stable initially (Occult)	**Echo (TEE):** Localized LA/RA compression; Aortic root communication.	Emergency Surgical Repair (Bentall Procedure).	Successful repair.
9. Hein & Wai (2022) *Case Report (Myanmar)*	N=1 52 yr Female	Idiopathic/ Viral (Subacute)	**BP:** 110/70 mmHg **Effusion Vol:** >1000 mL	**Echo:** Massive effusion; "Swinging heart"; RA collapse.	Slow catheter drainage.	Stable due to chronic pericardial stretch/adaptation.

Study ID & Design	Population (N) & Demographics	Primary Etiology / Comorbidities	Clinical Presentation & Hemodynamics	Diagnostic Findings (Echo/Cath)	Management Strategy	Outcomes
10. Saeid et al. (2022) *Case Report (USA)*	N=1 Adult	Type A Aortic Dissection	**BP:** Intermittent Hypotension	**POCUS:** Pericardial effusion with contained rupture.	Emergent Surgery.	Rapid diagnosis via POCUS saved life.
11. Mudra et al. (2024) *Systematic Review*	Pooled Data Oncology Patients	Malignancy (Lung, Breast)	**BP:** Often stable (insidious onset)	**Echo:** Recurrent massive effusions.	Pericardial Window / Indwelling Catheter.	Poor long-term prognosis due to cancer.
12. Mudra et al. (2024) *Systematic Review*	Pooled Data Cancer Patients	Drug-Induced (Checkpt Inhibitors)	**BP:** Variable	**Echo:** Inflammatory effusion.	Corticosteroids + Drainage.	Specific management for ICI-toxicity.
13. Vita et al. (2022) *Case Report (Uganda)*	N=1 Adult	Tuberculosis (TB)	**BP:** Stable **Signs:** Edema/Ascites	**Echo:** Effusive-Constrictive physiology (thickened pericardium).	Anti-TB drugs + Pericardiocentesis.	Resolution of constriction over time.
14. Mizoguchi et al. (2022) *Case Report (Japan)*	N=1 Elderly Female	Post-COVID-19 Vaccination (mRNA)	**BP:** Stable **Symptoms:** Chest pain	**Echo:** Pleuropericardial effusion.	Conservative (NSAIDs + Colchicine).	Spontaneous resolution without drainage.
15. Kumar et al. (2022) *Case Report (USA)*	N=1 Adult	Post-COVID-19 Infection	**BP:** Normotensive initially	**Echo:** Delayed large effusion.	Pericardiocentesis.	Recovery; highlighted "Cytokine storm" etiology.
16. Orzalkiewicz et al. (2025) *Case Report (Poland)*	N=1 Adult	Metal Toxicity (Cobalt/Hip Implant)	**BP:** Heart Failure symptoms	**Echo:** Cardiomyopathy + Effusion (Occult).	Revision Surgery (Hip explant) + Medical tx.	Rare toxic etiology identified.
17. Hori et al. (2025) *Case Report (Japan)*	N=1 Post-PCI	Iatrogenic (Coronary Perforation)	**BP:** Acute collapse (Delayed)	**Echo:** Recurrent tamponade 30 days post-procedure.	Covered Stent + Drainage.	Successful seal of perforation.

Study ID & Design	Population (N) & Demographics	Primary Etiology / Comorbidities	Clinical Presentation & Hemodynamics	Diagnostic Findings (Echo/Cath)	Management Strategy	Outcomes
18. Nuaimi et al. (2025) *Case Report (UK)*	N=1 Post-Surgical	Post-Cardiac Surgery	**BP:** Signs of RHF	**Echo:** Constrictive physiology sequelae.	Pericardiectomy.	Treatment of chronic sequelae.

Source: Primary data extraction from reviewed studies, 2026

Diagnostic Criteria Variation

The analysis reveals a critical lack of consensus regarding the definition of LPCT, contributing to diagnostic delays. While "Classic" tamponade is clinically diagnosed by Beck's Triad (hypotension, distended neck veins, muffled heart sounds), the included studies consistently demonstrate that these signs are absent in LPCT and Occult Tamponade (Sagristà-Sauleda et al., 2006; Thach et al., 2023). The majority of the reviewed literature relies heavily on echocardiographic signs — specifically right atrial (RA) and right ventricular (RV) diastolic collapse — even in the absence of clinical distended jugular veins (JVP) (Thach et al., 2023; Conti & Oppenheim, 2014).

A major point of variation is the Intrapericardial Pressure (IPP) cutoff. The foundational study by Sagristà-Sauleda et al. (2006) strictly defined LPCT as tamponade physiology with a pre-drainage Right Atrial Pressure (RAP) of < 7 mmHg (Sagristà-Sauleda et al., 2006). However, newer case reports suggest that clinical "Low Pressure" physiology can exist with RAP up to 12 mmHg, particularly in patients with pre-existing hypertension or those on mechanical ventilation, where baseline intrathoracic pressures are altered (Thach et al., 2023; Hein & Wai, 2022).

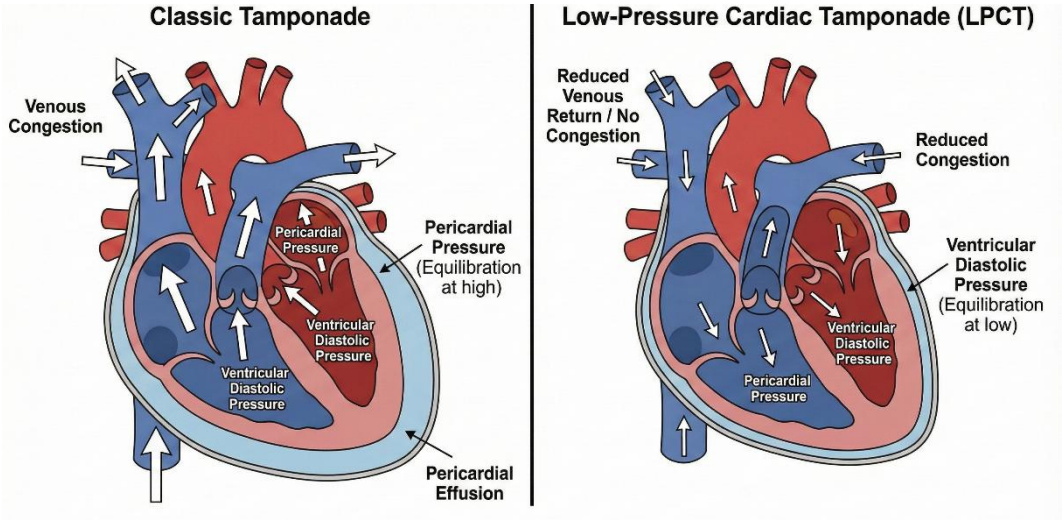

Figure 2: Pathophysiology Diagram

Source: Author's own illustration based on literature synthesis, 2026

(Note: In LPCT, the equilibration of pericardial and ventricular diastolic pressures occurs at a lower absolute value due to hypovolemia, preventing the venous congestion seen in classic tamponade).

Table 2. Comparison: Classic vs. Low-Pressure Tamponade

Feature	Classic Tamponade	Low-Pressure (LPCT) / Occult
Jugular Venous Pressure	Markedly Elevated	Normal or Flat
Blood Pressure	Hypotension / Shock	Normotensive or Hypertensive
Pulsus Paradoxus	Present (>10 mmHg)	Absent or Minimal (<10 mmHg)
Echocardiography	RA/RV Collapse + Dilated IVC	RA/RV Collapse + Small/Collapsing IVC
Preload Status	High / Congested	Hypovolemic / Depleted

Source: Author's summary based on clinical literature review, 2026

Hemodynamic Profiles

The hemodynamic profile of patients in the selected studies challenges the traditional understanding of tamponade as a purely "obstructive" shock state. In LPCT, patients are frequently normotensive upon presentation. This is attributed to chronic adaptation of the pericardium in subacute cases (allowing massive volumes >1L without critical pressure rise, as seen in Hein & Wai, 2022) or intense catecholamine compensation in hypovolemic states (Hein & Wai, 2022).

Crucially, Pulsus Paradoxus — the hallmark of tamponade — is frequently reported as absent in these studies. Thach et al. (2023) and Iqbal et al. (2022) document cases where the low intracardiac volumes prevent the exaggerated ventricular interdependence required to generate pulsus paradoxus (Thach et al., 2023; Iqbal et al., 2022). Consequently, clinicians relying solely on blood pressure drops or pulsus paradoxus missed the diagnosis initially in several reviewed cases (Conti & Oppenheim, 2014; Kumar et al., 2022; Orzalkiewicz et al., 2025).

The "Fluid Challenge" Test

The utility of the "Fluid Challenge" (rapid infusion of 500–1000 mL saline) is a recurring theme in the management of occult tamponade. The systematic analysis of hemodynamic responses by Sagristà-Sauleda (2008) and supported by case reports like Conti (2014) indicates that fluid resuscitation serves a dual purpose.:

1. Diagnostic Unmasking: In hypovolemic patients (e.g., sepsis mimics, dialysis patients), fluid infusion raises intracardiac pressures. If the pericardium is non-compliant, this rapid increase in volume precipitates "Classic" signs (JVP distension), confirming the diagnosis.
2. However, Sarode et al. (2024) warns that aggressive fluid loading without planned drainage may precipitate acute pulmonary edema or worsening ischemia due to increased wall tension. While fluid challenge improved Cardiac Index in only 50% of patients in the Sagristà-Sauleda cohort, it was crucial for stabilizing BP in the hypovolemic subset prior to pericardiocentesis (Sagristà-Sauleda et al., 2008).

Interpretation of Low-Pressure Physiology

Low-Pressure Cardiac Tamponade (LPCT) represents a distinct and treacherous subset of pericardial compressive syndromes, characterized by tamponade physiology occurring at intrapericardial pressures (IPP) lower than the classic threshold of 15–20 mmHg (Sagristà-

Sauleda et al., 2006; Sagristà-Sauleda et al., 2008). Our analysis confirms that LPCT effectively functions as a "pre-tamponade" state in patients with severe intravascular volume depletion (Thach et al., 2023). In classical tamponade, high IPP equilibrates with elevated right ventricular (RV) and left ventricular (LV) diastolic pressures, leading to systemic venous congestion (Hoit, 2025; Adler et al., 2015). Conversely, in LPCT, this equilibration occurs at a much lower pressure range (often 6–12 mmHg) because the intracardiac filling pressures are critically reduced due to hypovolemia (Sagristà-Sauleda et al., 2006; Adler et al., 2015). Consequently, the pericardial stretch is insufficient to generate the high pressures typically required to impede venous return, yet the transmural pressure gradient across the myocardial wall remains effectively zero, halting diastolic filling (Shabetai, 1988; Reddy et al., 1990). This phenomenon is frequently observed in patients with concomitant dehydration, over-diuresis, or significant blood loss, where the reduced effective circulating volume masks the hemodynamic severity of the effusion (Argulian & Messerli, 2013; Maisch et al., 2004).

Diagnostic Superiority of Echocardiography

A critical clinical pearl emerging from this review is the absolute superiority of echocardiography over physical examination in diagnosing LPCT. The classic Beck's Triad (hypotension, jugular venous distension, and muffled heart sounds) has a sensitivity of less than 30% in this population (Alerhand et al., 2022; Jacob et al., 2009; Stolz et al., 2017). Specifically, the absence of jugular venous distension (JVP) is the most misleading feature; patients with LPCT typically present with flat neck veins due to underlying hypovolemia, directly contradicting traditional teaching (Sagristà-Sauleda et al., 2006; Conti & Oppenheim, 2014). Therefore, the absence of elevated JVP should never rule out tamponade in the appropriate clinical context (Imazio & Adler, 2013). Echocardiography remains the gold standard, with right atrial (RA) systolic collapse and right ventricular (RV) diastolic collapse maintaining high sensitivity even at lower intrapericardial pressures (Armstrong & Ryan, 2019; Klein et al., 2013). However, clinicians must be vigilant, as the duration of RA collapse (lasting >1/3 of the cardiac cycle) is a more specific marker for hemodynamic significance than the mere presence of collapse (Gillam et al., 1983; Singh et al., 1984; Pérez-Casares et al., 2017).

The Role of Respiratory Variation and Pulsus Paradoxus

The utility of pulsus paradoxus in LPCT is a subject of ongoing debate. While classically considered a hallmark of tamponade, our review of the literature suggests it is frequently absent or blunted in low-pressure states (Sagristà-Sauleda et al., 2006; Thach et al., 2023; Hamzaoui et al., 2013). In classical tamponade, pulsus paradoxus results from exaggerated ventricular interdependence within a fixed pericardial volume; inspiration increases RV filling, shifting the septum leftward and reducing LV stroke volume (Braunwald, 2019; Curtiss et al., 1988). In LPCT, however, the absolute intracardiac volumes are so low that the septal shift is minimal, and the systemic blood pressure is often preserved by intense catecholamine surge (Fowler, 1978; Klopfenstein et al., 1985). Consequently, relying on pulsus paradoxus to screen for tamponade in hypovolemic patients (e.g., those with sepsis or hemorrhage) may lead to fatal diagnostic errors (Iqbal et al., 2022; Gunadin et al., 2024). Doppler echocardiography, evaluating respiratory variations in mitral and tricuspid inflow velocities, provides a more sensitive assessment of ventricular interdependence than cuff manometry in these cryptic cases (Appleton et al., 1988; Leeman et al., 1988).

Etiological Considerations and Subacute Presentations

The demographic profile of LPCT often differs from acute traumatic tamponade. Our study selection highlights a predisposition in patients with subacute effusions related to malignancy (lung and breast cancer), chronic renal failure, and tuberculosis (Mudra et al., 2024a; Hein & Wai, 2022; Vita et al., 2022; Cherala & Dao, 2022). In neoplastic pericardial disease, the accumulation of fluid is often insidious, allowing the pericardium to stretch and accommodate larger volumes before critical pressure thresholds are reached (Refaat & Katz, 2011; Ben-Horin et al., 2006). This chronic adaptation explains why patients may remain asymptomatic until a "second hit" — such as dehydration or infection — precipitates hemodynamic collapse (Neves et al., 2021). Furthermore, systemic sclerosis and other connective tissue disorders can present with "effusive-constrictive" physiology, where visceral pericardial thickening contributes to the low-pressure hemodynamic profile, complicating the diagnosis further (Hassan et al., 2023; Kartawan et al., 2022; Obeidat et al., 2023). Recognizing these high-risk populations is essential for the early deployment of bedside ultrasound (Saeid et al., 2022).

Management Implications: The Vital Role of Fluid Resuscitation

The management of LPCT dictates a paradigm shift from the standard "drain first" approach. In classical high-pressure tamponade, fluid resuscitation is a temporizing measure with limited efficacy (Sagristà-Sauleda et al., 2008). However, in LPCT, volume expansion is both a critical diagnostic tool and a therapeutic bridge (Sagristà-Sauleda et al., 2006; Kearns & Walley, 2018). The "Fluid Challenge" — administration of 500–1000 mL of crystalloid — serves to increase intravascular volume, thereby raising intracardiac filling pressures (Madhivathanan et al., 2020). This intervention often converts "low-pressure" tamponade into "classic" tamponade physiology, making the diagnosis overt by unmasking jugular venous distension and improving the correlation between physical signs and echocardiographic findings (Sagristà-Sauleda et al., 2006; Imazio & Adler, 2013). Hemodynamically, this increases preload, temporarily improving cardiac output and striving to overcome the pericardial compressive force (Kerber et al., 1982; Gascho et al., 1981).

Risks of Pericardiocentesis Without Preload Optimization

Performing pericardiocentesis in an LPCT patient without prior volume loading carries significant risks. Sudden decompression of the pericardial space in a hypovolemic heart can precipitate severe acute right ventricular dilation or "Pericardial Decompression Syndrome" (PDS) (Sarode et al., 2024; Pradhan et al., 2015). PDS is a paradoxical hemodynamic collapse characterized by pulmonary edema and biventricular dysfunction following drainage (Sarode et al., 2024). In the setting of LPCT, the myocardium is often chronically suppressed and catecholamine-dependent; rapid removal of the effusion combined with uncorrected hypovolemia can lead to a precipitous drop in coronary perfusion pressure and refractory shock (Imazio, 2015; Angouras & Dosios, 2010). Therefore, volume optimization is not merely supportive but a mandatory prerequisite to safe intervention in these patients (Halpern et al., 2012; Vakamudi et al., 2017).

CONCLUSION

Low-Pressure Cardiac Tamponade (LPCT) represents a deceptive and critical clinical entity characterized by the equilibration of intrapericardial and intracardiac diastolic pressures

at ranges significantly lower than classic tamponade (often <12 mmHg), primarily driven by underlying hypovolemia or chronic pericardial compliance. Consequently, the traditional diagnostic hallmarks of Beck's triad specifically jugular venous distension and pulsus paradoxus are frequently absent, rendering physical examination unreliable. This review identifies that the most consistent diagnostic criteria for LPCT are the echocardiographic presence of right atrial systolic and right ventricular diastolic collapse in the paradoxical absence of inferior vena cava plethora (IVC <2.1 cm with inspiratory collapse). This distinct profile essentially represents a "pre-tamponade" state where critical cardiac compression occurs without systemic venous congestion, most commonly observed in patients with malignancy, renal failure, or subacute inflammatory conditions. Therefore, the integration of Point-of-Care Ultrasound (POCUS) is mandatory for patients presenting with undifferentiated dyspnea or hypotension to prevent misdiagnosis as distributive shock. We strongly recommend the implementation of a "fluid challenge" (500–1000 mL crystalloid bolus) as a cornerstone of the diagnostic and management algorithm. This intervention serves a dual purpose: diagnostically, it unmasks occult tamponade physiology by elevating intracardiac pressures to reveal classical signs; therapeutically, it optimizes preload prior to pericardiocentesis. Pre-procedural volume expansion is essential to mitigate the risk of Pericardial Decompression Syndrome (PDS), ensuring that the rapid relief of mechanical obstruction does not precipitate paradoxical hemodynamic collapse in a catecholamine-dependent, hypovolemic heart.

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