Case Presentation
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Twin For Bad, Pericardial Effusion and Cardiac Tamponade, A Dangerous Combination

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Abstract

Pericardial effusion and cardiac tamponade represent a continuum of potentially life-threatening cardiac emergencies, often presenting with subtle or non-specific clinical features. This article presents a case of subacute, occult cardiac tamponade in a 56-year-old male, incidentally, discovered during cardiology follow-up. Despite a large volume of pericardial fluid, the patient remained asymptomatic due to gradual accumulation, allowing pericardial stretch. He underwent successful pericardiocentesis and stabilization. The article discusses the diverse etiologies, pathophysiology, diagnostic modalities, and therapeutic strategies for managing pericardial effusion and tamponade. Timely diagnosis, high clinical suspicion, and appropriate intervention are crucial for optimal outcomes, particularly in atypical or occult presentations.

Keywords: ***Cardiac tamponade, Echocardiography, Electrocardiography, Infectious, Inflammation, Interventricular interdependence, Pericardiocentesis, Pericardial effusion, Pericardial window, Pulsus paradoxus, Raised JVP.***

Introduction

The pericardium is a fibroelastic coat covering for the heart with an intervening thin film of fluid between them. Pericardial effusion is said to occur when accumulated fluid within the sac exceeds the physiologic amount of about 15 to 50 mL [1-3].

The causes of excess fluid accumulation i.e. pericardial effusion in the potential space of the pericardium can be broadly divided into idiopathic, infectious and noninfectious causes [1-3].

In the case of idiopathic cause, no identifiable reason for the pericardial effusion while infectious causes range from viruses like: Coxsackievirus, Echovirus, Adenovirus, Epstein-Barr virus, Cytomegalovirus, Influenza, Varicella, Rubella, HIV, Hepatitis B, Mumps, Parvovirus B19, Smallpox vaccine and SARS-CoV-2; Bacterial such as: Mycobacterium tuberculosis, Staphylococcus, Streptococcus, Hemophilus, Neisseria, Chlamydia, Legionella, Salmonella, Borrelia burgdorferi, Mycoplasma, Actinomyces, Nocardia, Tropheryma whippelii, Treponema and Rickettsia, Fungal like: Histoplasma, Aspergillus, Blastomyces, Coccidioides, Candida and lastly Parasitic infections like: Echinococcus, Amebic and Toxoplasma [1-3].

The noninfectious causes range from autoimmune disorders like lupus, rheumatoid arthritis, scleroderma, Sjögren syndrome, vasculitis, mixed connective disease.

Autoinflammatory diseases especially familial Mediterranean fever, tumor necrosis factor associated periodic syndrome [TRAPS], and IgG4-related disease have also been implicated. Others in this group includes Granulomatosis with polyangiitis, polyarteritis nodosa, sarcoidosis, inflammatory bowel disease (Crohn, ulcerative colitis), giant cell arteritis, Behçet syndrome, and vaccines.

Neoplasm (primary, metastatic and paraneoplastic), cardiac (infarction, myocarditis and aortic dissection), metabolic like uremia, hypothyroidism, radiation, drug induce lupus and postcardiac injury syndrome complete this list [1-3].

When pericardial fluid accumulates rapidly and in sufficient amount, the heart can be compressed when the fibroelastic pericardium exceeds its elastic limit, thereby compromising venous return, leading to a reduction in stroke volume, and ultimately, hemodynamic collapse. This condition is called cardiac tamponade, a medical emergency.

Case Presentation

A 56-year-old male with a past medical history of culture-negative mitral endocarditis s/p MVR (#33 Mitris bioprosthetic valve with 8-week course of IV vancomycin/Rocephin), embolic CVA, splenic infarct, left A2 aneurysm s/p flow diverter placement, non-insulin dependent type 2 diabetic mellitus (A1c 13.3%), HTN, HLD, CKD stage II-IIIa (baseline creatinine 1.5-1.7), and normocytic anemia, came to the emergency department after an echo showed a large pericardial effusion and possible aortic dissection. He follows with cardiology and had an appointment earlier that day from where he was sent to the ED.

He had a recent hospitalization for mitral valve replacement and emergency brain aneurysm repair, denies chest pain, shortness

of breath, generalized weakness, blurry vision, and cough. His blood sugar was high at 770 and does not take diabetes medication.

He admitted to poor diet choices during his hospital stay but denies excessive thirst or urination. No tobacco or alcohol use, having stopped smoking marijuana 2 months earlier.

ED Course

1. **Vital Signs:** Blood pressure on arrival 150/84, heart rate 86, respiratory rate 18, saturating well on room air.
2. **Pertinent Labs:** BMP showed sodium of 132, potassium of 4.4, glucose 779, anion gap 8, creatinine 1.50 and calcium 9.7. CBC showed WBC count of 5.9, hemoglobin 12.1 and platelets 160. BNP 112, Troponins 12.
3. **ED Imaging:** CT Angio chest showed there was no evidence of aneurysm or dissection of the thoracic or abdominal aorta, moderate to large pericardial effusion was however seen. Transthoracic echo showed ejection fraction of 55 to 60%, mild concentric hypertrophy. There was a moderate to large circumferential pericardial effusion with a maximum diameter of 2.5 cm. Although there was some tricuspid valve respiratory variation, no definitive tamponade. The ascending aorta was mildly dilated (4.0 cm).
4. **EKG:** was sinus rhythm, Left axis deviation with right bundle branch block and Possible Inferior infarct. Abnormal ECG When compared with ECG four months prior. ECG interpretation was limited by artifact, follow-up ECG was recommended.

Review of Systems

- **Constitutional:** Negative for activity change, appetite change, chills, diaphoresis and fatigue.
- **HENT:** Negative for congestion, facial swelling, rhinorrhea, sinus pressure, sneezing and sore throat.
- **Eyes:** Negative for photophobia.
- **Respiratory:** Negative for apnea, cough, chest tightness, shortness of breath, wheezing and stridor.
- **Cardiovascular:** Negative for chest pain, palpitations and leg swelling.
- **Gastrointestinal:** Negative for abdominal distention, abdominal pain, anal bleeding, blood in stool, diarrhea, nausea, rectal pain and vomiting.
- **Endocrine:** Negative for polydipsia, polyphagia and polyuria.
- **Genitourinary:** Negative for difficulty urinating, flank pain, frequency, genital sores and hematuria.
- **Musculoskeletal:** Negative for arthralgias, joint swelling and neck stiffness.
- **Skin:** Negative for color change and pallor.
- **Neurological:** Negative for dizziness, seizures, syncope, speech difficulty, weakness, light-headedness and headaches.
- **Psychiatric/Behavioral:** Negative for agitation and behavioral problems.

Physical Exam

- **General:** No apparent distress
- **HNT:** NCAT, moist mucous membranes
- **Eyes:** Sclera anicteric, EOMI, PERRL
- **RS:** Clear to auscultation bilaterally, no added sounds
- **CVS:** No JVD, S1 S2 heard, no murmurs, pulse regular rhythm and rate
- **P/A:** Soft, non-tender, non-distended, no evidence of free fluid appreciated, bowel sounds noted
- **GU:** No CVA tenderness, no indwelling catheter - voiding freely
- **Extremities:** Pulses 2+ bilaterally, No edema/cyanosis
- **Neurologic:** Alert/awake/oriented to time, place and person, following verbal commands, grossly no focal neurological deficits appreciated

He was slated for pericardiocentesis two days later and underwent successful echo/fluoroscopy guided pericardiocentesis with drainage of 860 cc of bloody pericardial fluid while samples were sent for analysis. He was then transferred to the ICU for postprocedural monitoring with pericardial drain in place.

ICU Course

He remained stable for the duration of his stay in ICU. Initial output from the drain was approximately 750 mL of serosanguinous fluid. Analysis of the fluid was positive for blood and lymphocytes.

About four days later, the fluid output was much reduced, after a TTE, cardiology cleared him for downgrade with removal of the drain.

Floor Course

His problem list was:

- Large pericardial effusion s/p pericardiocentesis and drain placed–now removed.
- Hypertension
- Hyperlipidemia
- T2DM

Cardiology thereafter recommended discharge with outpatient follow-up, to continue Coreg, lisinopril, amlodipine as needed for blood pressure control. Also, to continue atorvastatin, aspirin while his ticagrelor was held.

Diabetic education was provided, and he was started on insulin lantus along with lispro by the endocrinologist.

Epidemiology

In a cohort of 340 patients diagnosed with cardiac tamponade, invasive percutaneous cardiac procedures accounted for a total of 33%, followed by those who developed post pericardiotomy syndrome after 7 days of cardiac surgery; were 23% of the total, those of neoplastic origin was next; 15, then pericarditis due to connective tissue disease, infectious, or noninfectious conditions; constituted 12.5%, closely followed by complications from cardiac surgery at 9%, uremia 2%, and lastly, acute myocardial infarction comprised of 1.5% [4].

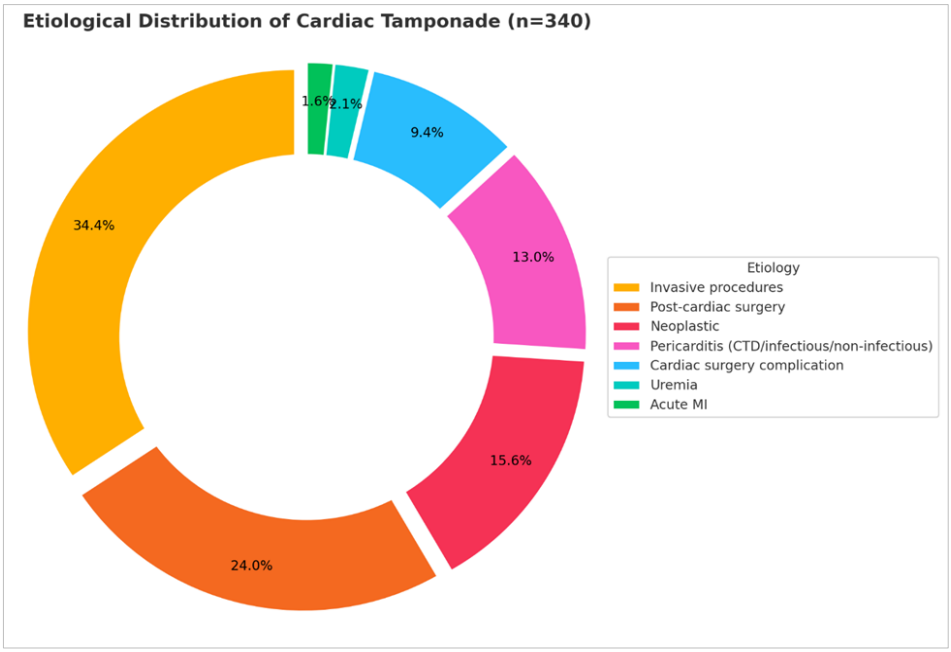

Figure 1: Distribution of Cardiac Tamponade Etiologies

In a systematic review of 34 patients with COVID-19 associated with pericarditis, of the 76% who had effusions, 35% developed tamponade [5].

Also, in a nationwide study done in Sweden, 200 of the 44,497 patients who underwent invasive electrophysiology procedures, developed procedure-related cardiac tamponade [6]. Ensuing five-years follow-up with these patients, showed increased hospitalization from pericarditis without a corresponding increase in adverse events.

A single-center 5-year experience on 5222 procedures for catheter ablations of atrial fibrillation, 51 of which was complicated by cardiac tamponade, amounting to 1% [7] of the procedures. It was least reported for cryoablation than it was for radiofrequency ablation.

Cardiac tamponade has also been reported to complicate less than 1% of people treated with fibrinolytic therapy for acute myocardial infarction and the risk may be lower in patients treated with percutaneous coronary intervention [8].

About 19% of patients who develops ascending aortic dissection (type A), would go on to experience cardiac tamponade according to findings from international registry [9].

Pathophysiology

In cardiac tamponade, the pressure of the pathologic accumulated fluid in the pericardial space, exceed the intracardial pressure during diastole, thereby compressing most or all of the heart chambers [10-11]. How fast this condition occurs is contingent on the rate of accumulation of the pericardial fluid with much smaller volume which accumulate rapidly like from cardiac rupture likely to occur quicker than slow accumulation of effusion because of the difference in the time for the fibroelastic sac to adapt to the increase in intrapericardial pressure [10].

Cardiac tamponade can be classified based on the rate of accumulation, whether rapid or gradual. This also determines when symptoms emerge and how soon intervention might be needed [10].

If it occurs rapidly within minutes like in aortic dissection or coronary artery perforation, it is said to be acute which usually requires urgent intervention because of the potential to cause hemodynamic compromise [12].

It is termed subacute if it occurs between days to weeks, seen in conditions like uremia, neoplasm and pericarditis. Symptoms might not be apparent at the beginning until enough fluid accumulate in the pericardial sac to compromise cardiac filling during diastole before the hemodynamic effect is made apparent. In a subset of this group, when the pressure of accumulated fluid is low from severe hypovolemia due to conditions like hemorrhage or over diuresis, jugular venous distention may not be present, this is called occult cardiac tamponade [10]. It can also be referred to as low pressure cardiac tamponade [26].

When the fluid collection is loculated, some of the heart chambers might be compressed, this is likely seen in the case of cardiac surgeries and rupture from myocardial infarction [10-11]. This is termed regional cardiac tamponade.

Diagnosis

This is achieved through history taking, symptoms and signs elicited during physical examination along with relevant investigations.

History would help to identify the likely etiology while symptoms usually range from fatigue to dyspnea and syncope due to hemodynamic instability.

Signs include:

1. **Sinus tachycardia:** indicates hemodynamic instability in the setting of cardiac tamponade.
2. **Raised JVP:** elevated jugular pressure is absent in occult (low pressure) cardiac tamponade.
3. **Pulsus paradoxus:** Exaggerated fall in systolic pressure greater than 10 mmHg during inspiration is largely due to ventricular interdependence. This phenomenon is due to the fact that in cardiac tamponade, the effective compliance of heart is curtailed by the tightly stretched fibroelastic pericardium during inspiration when the intrathoracic pressure falls and blood returns to the right side of the heart and pulmonary circulation. To accommodate this increased volume to the right ventricle, the interventricular septum, bulges into the left ventricle, leading to its relative underfilling during inspiration,

exaggerating the drop in systolic blood pressure. This is not the case under normal condition.

4. **Hemodynamic collapse:** When severe, cardiac tamponade leads to a reduction in venous return due to compression of cardiac chambers, leading to a decline in stroke volume, ultimately, blood pressure drops.
5. **Muffled or distant heart sounds:** the excess accumulated fluid around the heart, makes the distance between the heart and the bell of the stethoscope to be increased.

Investigations

Patients suspected of having cardiac tamponade should be evaluated with an electrocardiogram (ECG), echocardiogram, and chest radiograph; urgent echocardiography may be the initial test in unstable patients with a high suspicion for cardiac tamponade.

ECG typically shows sinus tachycardia, electrical alternans where there is beat to beat changes in the amplitude of the QRS complex. It is due to swinging of the heart in the pericardial fluid [13] and is seen in about 35% of cases of cardiac tamponade [14]. Others are low voltage complexes.

Echocardiogram can be used to evaluate the amount and features of accumulated fluid in the pericardial sac [15-16]. Evidence of tamponade on echocardiography includes:

1. Chamber compression

The right side of the heart is commonly affected when the transmural pressure gradient is transiently reversed during diastole. The intrapericardial pressure exceeds the pressures in the cardiac chambers, leading to the collapse of the walls of the heart chambers [17]. This is because the right atrium and ventricle are very compliant[18].

Right atrial collapse occurs during end-diastole when atrial relaxation occurs, and the volume of the right atrium is minimal. The higher pressure in the surrounding pericardial space, can compress its walls, leading to its wall collapse. This can be seen in up to one-third of the cardiac cycle in cardiac tamponade while brief episodes can be seen in its absence [19]. Thus, it is both sensitive and specific for cardiac tamponade.

Conversely, right ventricular collapse occurs in early diastole when the volume of the right ventricle is small [17]. Right ventricular collapse is not sensitive for ruling out cardiac tamponade because if its walls are hypertrophied or its diastolic pressure is markedly elevated, it may not occur [18] but is highly specific for cardiac tamponade [20].

Left atrial collapse is seen in about 25 percent of those with cardiac tamponade, who develops hemodynamic instability and is very specific for cardiac tamponade [16,21-22]. Left ventricular collapse is not common because it is more muscular leading to more wall thickness when compared with the other heart chambers, and maybe seen in cases of regional cardiac tamponade [17,22].

2. Respiratory variation in volumes and flows

During inspiration, the atrial and ventricular septa moves leftward and then rightward during expiration. Respiratory variation with flow velocity across mitral and tricuspid flow is increased and out of phase, due to increased ventricular interdependence. Normally, there is no more than 20 to 25 percent variation in the amplitude of inflow and outflow signals across the valves during respiration. However, in cardiac tamponade, there is increased respiratory variation in transvalvular blood flow velocities with inspiration causing a decrease in mitral valve flow and an increase in tricuspid valve flow, a phenomenon called flow velocity paradoxus. During inspiration, mitral flow decreases usually exceeds 30 percent when compared with expiration, and conversely, inspiration causes an increase in tricuspid valve inflow variation usually exceeds 60 percent, when referenced to expiration [23].

3. IVC plethora and hepatic vein flow

Central venous pressure elevation in cardiac tamponade, present as dilation of the inferior vena cava (IVC) and less than 50 percent reduction in diameter during inspiration. In one report, IVC plethora was present in 92 percent of cardiac tamponade patient requiring pericardial drainage [24].

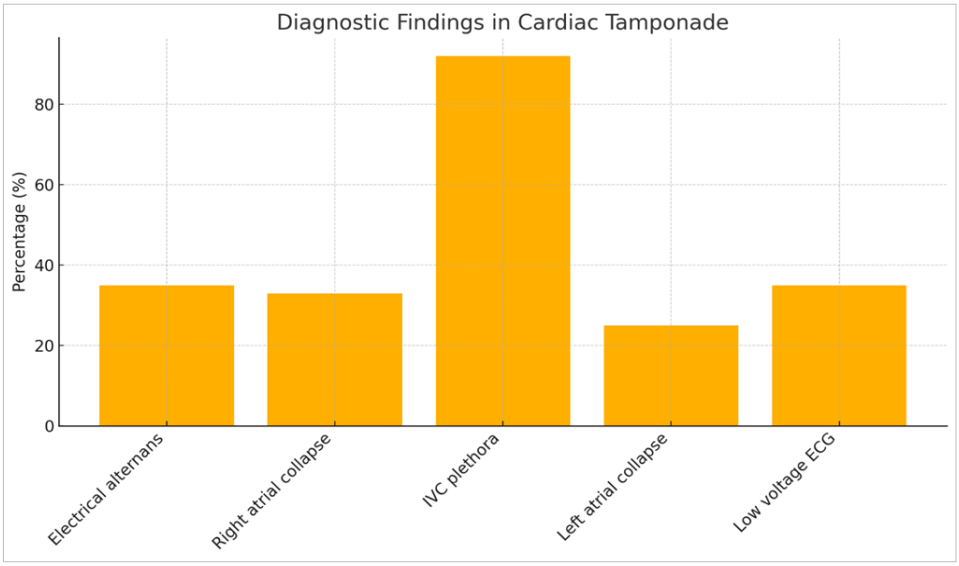

Figure 2: Frequency of Diagnostic Findings in Tamponade

Enlarged cardiac silhouette may be seen on chest X-ray.

CT and cardiovascular magnetic resonance (CMR) imaging is used to further characterize the effusion or other pericardial diseases. It is employed as an adjunct to echocardiography. CT is more ideal for patients with subacute presentation [23].

Cardiac catheterization: If possible, hemodynamics can be assessed through cardiac catheterization during fluid drainage procedure. Right atrial pressure is mostly elevated, and the Y descent is blunted due to a slower than normal fall in right atrial pressure after the tricuspid valve opens. In some patients, the X descent is

prominent. The increased pressure within the pericardium compresses the heart chambers, particularly during diastole when blood is returning to the heart, leading to an increase in the diastolic pressures in all cardiac chambers namely: right atrium, right ventricle, left atrium and left ventricle. As the pericardial pressure increases, the pressure in the chambers of the heart, becomes similar i.e. average intracardiac diastolic pressures equilibration.

Management

Pericardiocentesis is the treatment of choice for most patients with an indication for pericardial fluid drainage. This can be done via percutaneous route with ultrasound guidance by interventional radiologist. If inaccessible by being loculated or coagulated, Surgical drainage with direct or video visualization can be employed. Surgical management is required for aortic or cardiac bleeding like in myocardial rupture, aortic dissection or penetrating chest trauma [25].

Urgent pericardiocentesis is indicated if pericardial effusion is causing hemodynamic compromise i.e. cardiac tamponade. Patients with purulent pericarditis also require urgent pericardial drainage. Samples can be sent for body fluid analysis, to determine the cause of fluid collection, if the etiology is still unknown.

Patients with tamponade can be temporize with volume repletion with saline or blood products while awaiting pericardial drainage.

Pericardiocentesis doesn't lead to complete drainage of the effusion all of the time, fluid can reaccumulate if there is active bleeding or fluid secretion, hence the need for post-procedure care. This includes admission into a unit with telemetry after initial drainage, to monitor cardiac activity and the patency of the pericardial catheter left in the pericardial space that is connected to sealed container with negative pressure. If fluid drainage in a three-to-five-day period is less than 25 to 50 mL/day, catheter can be safely removed but if this is not achieved in which case, the catheter is draining over 50mL/day, a pericardial window can be surgically left in place [10].

Management of underlying pathology helps in preventing recurrence for example, treating inflammatory process like acute pericarditis with NSAIDs and colchicine, leads to both resolution and forestalling of recurrence.

When pericardial effusion is recurrent like in malignant and purulent pericarditis, it is desirable to combine initial drainage with long lasting therapies like; repeated pericardial drainage, leaving a catheter in the pericardial space for prolong period of time and creating a pericardial window through surgical pericardiotomy.

Table 1: Summary Table Diagnosis & Management of Tamponade

DIAGNOSIS AND MANAGEMENT SUMMARY FOR CARDIAC TAMPONADE

CATEGORY	METHOD	DETAILS
Diagnosis	Echocardiogram	Detects chamber collapse, IVC plethora, flow variation
Diagnosis	ECG	Electrical alternans, low voltage, tachycardia
Management	Pericardiocentesis	Ultrasound-guided, indicated for hemodynamic compromise
Management	Surgical Drainage	Used in loculated and acute bleeding from rupture

Discussion

The case discussed above, fit more into the category of an occult form of subacute cardiac tamponade, which explains why he was asymptomatic even while he had large pericardial effusion which was incidentally discovered on imaging while following up with cardiology after mitral valve replacement surgery. The fibroelastic pericardium had time to stretch to accommodate the excess fluid that accumulated in the pericardial.

He didn't endorse the classic findings on physical examination which includes tachycardia, dyspnea, hypotension, raised JVP or pulsus paradoxus.

His EKG also lacked typical findings of low voltage complexes, electrical alternans which is found in about 35% of cases or tachycardia. His echo showed large collection of pericardial fluid with no definitive tamponade with variation of tricuspid flow velocity with respiration.

The etiology wasn't clear, but from past medical history, he had endocarditis which was managed with prolonged course of antibiotics and had his mitral valve replacement surgery along with emergency brain aneurysm repair and mild kidney impairment with no uremia. There was initial thought that he was having dissection of his ascending aorta because of dilation seen on echo but was ruled out on CT Angio chest.

He was managed with the standard therapy of pericardiocentesis by interventional cardiology and a drain was left in place to prevent re-accumulation.

Co-morbid conditions were managed by the various specialist, and he was discharged with instruction to follow outpatient with cardiologist.

Conclusion

Cardiac tamponade is a medical emergency caused by pathologic accumulation of fluid in the pericardial sac due to its potential to cause hemodynamic compromise from compression of cardiac chambers during the filling phase of the cardiac cycle, ultimately leading to a reduction in cardiac output.

It can be caused by various infectious, inflammatory and non-infectious conditions.

How quickly patients come down with symptoms, depends on how rapidly the fluid buildup in the pericardial space.

Diagnosis can be through history and findings on physical examination which includes: The triad of hypotension, elevated JVP and distant heart sound (Beck triad), tachycardia, fatigue, dyspnea

and reduced urine output. Those of infectious origin like acute pericarditis can also have fever and pleuritic chest pain.

Investigations that can help to confirm the diagnosis includes electrocardiogram, echocardiography, CT chest, cardiac MRI and cardiac catheterization.

Treatment of choice is percutaneous pericardiocentesis and if inaccessible due to loculation, surgical pericardiotomy can be done. Drain can be left in place to prevent re-accumulation while recurrence can be prevented by treating the underlying pathology.

Declarations

Informed consent and ethical approval

Taken from The Institutional Ethics Committee

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None

Competing interest

None

Data availability

Available on corresponding author upon responsible request.

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References

[1] LeWinter M. Clinical practice. Acute pericarditis. N Engl J Med 2014; 371:2410.

[2] Imazio M, Gaita F. Diagnosis and treatment of pericarditis. Heart 2015; 101:1159.

[3] Imazio M. Contemporary management of pericardial diseases. Curr Opin Cardiol 2012; 27:308.

[4] Adamczyk M, Wasilewski J, Niedziela JT, et al. Baseline characteristics, management and long-term outcomes of different etiologies of cardiac tamponade evaluated in a cohort of 340 patients. Kardiochir Torakochirurgia Pol 2021; 18:216.

[5] Diaz-Arocutipa C, Saucedo-Chinchay J, Imazio M. Pericarditis in patients with COVID-19: a systematic review. J Cardiovasc Med (Hagerstown) 2021; 22:693.

[6] Von Olshausen G, Tabrizi F, Sigurjónsdóttir R, et al. Cardiac tamponades related to interventional electrophysiology procedures are associated with higher risk of short-term hospitalization for pericarditis but favourable long-term outcome. Europace 2023; 25.

[7] Hamaya R, Miyazaki S, Taniguchi H, et al. Management of cardiac tamponade in catheter ablation of atrial fibrillation: single-centre 15year experience on 5222 procedures. Europace 2018; 20:1776.

[8] Figueras J, Barrabés JA, Serra V, et al. Hospital outcome of moderate to severe pericardial effusion complicating ST-elevation acute myocardial infarction. Circulation 2010; 122:1902.

[9] Gilon D, Mehta RH, Oh JK, et al. Characteristics and in-hospital outcomes of patients with cardiac tamponade complicating type A acute aortic dissection. Am J Cardiol 2009; 103:1029.

[10] Spodick DH. Acute cardiac tamponade. N Engl J Med 2003; 349:684.

[11] Troughton RW, Asher CR, Klein AL. Pericarditis. Lancet 2004; 363:717.

[12] Reddy PS, Curtiss EI, Uretsky BF. Spectrum of hemodynamic changes in cardiac tamponade. Am J Cardiol 1990; 66:1487.

[13] Kudo Y, Yamasaki F, Doi T, et al. Clinical significance of low voltage in asymptomatic patients with pericardial effusion free of heart disease. Chest 2003; 124:2064.

[14] Mathur AP, Saini A, Lucas BP, et al. Diagnostic accuracy retrospectively of electrocardiographic findings and cancer history for tamponade in patients determined to have pericardial effusion by transthoracic echocardiogram. Am J Cardiol 2013; 111:1062.

[15] Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 2015; 36:2921.

[16] Cheitlin MD, Armstrong WF, Aurigemma GP, et al. ACC/AHA/ASE 2003 guideline for the clinical application of echocardiography www.acc.org/qualityandscience/clinical/statements.htm (Accessed on August 24, 2006).

[17] Reydel B, Spodick DH. Frequency and significance of chamber collapses during cardiac tamponade. Am Heart J 1990; 119:1160.

[18] Leimgruber PP, Klopfenstein HS, Wann LS, Brooks HL. The hemodynamic derangement associated with right ventricular diastolic collapse in cardiac tamponade: an experimental echocardiographic study. Circulation 1983; 68:612.

[19] Gillam LD, Guyer DE, Gibson TC, et al. Hydrodynamic compression of the right atrium: a new echocardiographic sign of cardiac tamponade. Circulation 1983; 68:294.

[20] Kerber RE, Gascho JA, Litchfield R, et al. Hemodynamic effects of volume expansion and nitroprusside compared with pericardiocentesis in patients with acute cardiac tamponade. N Engl J Med 1982; 307:929.

[21] Torelli J, Marwick TH, Salcedo EE. Left atrial tamponade: diagnosis by transesophageal echocardiography. J Am Soc Echocardiogr 1991; 4:413.

[22] Fusman B, Schwinger ME, Charney R, et al. Isolated collapse of left-sided heart chambers in cardiac tamponade: demonstration by two-dimensional echocardiography. Am Heart J 1991; 121:613.

[23] Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. J Am Soc Echocardiogr 2013; 26:965.

[24] Himelman RB, Kircher B, Rockey DC, Schiller NB. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. J Am Coll Cardiol 1988; 12:1470.

[25] Arefin, S., & Zannat, N. T. (2024). The ROI of Data Security: How Hospitals and Health Systems Can Turn Compliance into Competitive Advantage. Multidisciplinary Journal of Healthcare (MJH), 1(2), 139-160.

[26] Arefin, N. T. Z. S. (2025). Future-Proofing Healthcare: The Role of AI and Blockchain in Data Security.

[27] Arefin, N. T. Z. S. (2025). AI vs Cyber Threats: Real-World Case Studies on Securing Healthcare Data.

[28] Arefin, S., & Simcox, M. (2024). AI-Driven Solutions for Safeguarding Healthcare Data: Innovations in Cybersecurity. International Business Research, 17(6), 1-74.

[29] Taqwa, M. R. A. (2025). Ethics in Social Science Research: Current Insights and Practical Strategies: Otto Federico von Feigenblatt and M. Rezaul Islam. 2025. Springer Singapore, 263 pp, ISBN 978-981-97-9880-3 (hbk), ISBN 978-981-97-9883-4 (pbk), ISBN 978-981-97-9881-0 (ePDF).

[30] Adler Y, Charron P, Imazio M, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J 2015; 36:2921.

[31] Sagristà-Sauleda J, Angel J, Sambola A, et al. Low-pressure cardiac tamponade: clinical and hemodynamic profile. Circulation 2006; 114:945.