## CLINICAL VIGNETTE

# Palpitations with a Cause

Michael Estes, MD and Melkon Hacobian, MD

***Case***

A 44-year-old healthy female presented to internal medicine complaining of new onset palpitations. The symptoms occurred while she was in an exercise class. She took a brief break and the sensations resolved after a few minutes. She denied any associated shortness of breath, chest pain or dizziness. She had no history of syncope or family history of sudden death. She recalled past episodes of palpitations that were briefer with a few "heavier feeling" beats. An EKG in the office was normal and recent labs ruled out thyroid disease, renal disease or electrolyte disturbances. Given the exertional nature, she was sent for further cardiac evaluation.

The initial work up included a 48 hour holter monitor. She was predominantly in sinus rhythm and had rare PACs/PVCs. She had no sustained ventricular or supraventricular arrhythmias. A 2D echocardiogram, discovered a large circumferential pericardial effusion and evidence of tamponade. The interpreting cardiologist was able to urgently evaluate the patient and she was admitted to the hospital for treatment.

She was hemodynamically stable and given intravenous fluid to maintain her blood pressure. She underwent pericardiocentesis with 450cc of serosanguinous fluid removed and sent for analysis. Bacterial, fungal and acid fast cultures of the fluid were negative. Cytology was negative for malignant cells. Typical causes including thyroid derangement, autoimmune disease, and HIV were also ruled out. The pericardial drain was left in place for three days until it was no longer actively draining fluid and the day of discharge echocardiogram did not demonstrate any residual effusion. She was sent home on colchicine for three months for a presumed acute, inflammatory process of unknown etiology. Follow up echocardiograms after discharge demonstrated a small reaccumulated pericardial effusion and she was readmitted for definitive surgery. CT scans of the chest, abdomen and pelvis did not find evidence of occult malignancy. A pericardial window was performed with cardiothoracic surgery and she did well. Final pathology showed normal pericardial tissue without inflammation or neoplasm with reactive changes in the fluid.

***Discussion***

A small amount of fluid often exists in the space between the visceral and parietal layers of the pericardium, but an accumulation of fluid known as an effusion, can result from several situations. Acute pericarditis is the result of inflammation of the pericardium and can present as fever, chest pain and effusion. Etiologies of pericarditis include viruses (coxsackieviruses, enteroviruses), bacteria (mycobacterium), and idiopathic.[1]

Autoimmune diseases like rheumatoid arthritis and lupus as well as metabolic disturbances of uremia and hypothyroidism are also known causes. Metastasis and rarely, primary malignancy, also cause effusions. One retrospective study found 18% of large effusions were caused by a previously unrecognized malignancy.[2] Rarely, fluid can accumulate after cardiac surgery, myocardial infarction, blunt trauma or aortic dissection.

The clinical presentation can range from asymptomatic, to fevers and chest pain with pericarditis, and heart failure with dyspnea, fatigue and edema from tamponade. The initial evaluation includes laboratory testing with complete blood count, thyroid function, renal function, ANA, electrocardiogram and chest X-ray. Fluid sampling is obtained when the underlying diagnosis is not readily apparent from the laboratory testing. Typically the initial fluid is obtained through pericardiocentesis and sent for bacterial, fungal and AFB cultures and cytology. The treatment depends on the underlying etiology (NSAIDs or colchicine for pericarditis, antimicrobials for infections, chemotherapy for malignancy). Treatment can be urgent, as with our patient, when there is evidence of cardiac tamponade. Additionally, recurrence after treatment or drainage is not uncommon and often requires repeat pericardiocentesis or more definitive surgical pericardiectomy.[3]

Cardiac tamponade is a clinical syndrome caused by an increase in intra pericardial pressure due to the accumulation of blood, other fluid, or gas in the pericardial space. Cardiac tamponade leads to a crisis by decreasing venous return, which impairs diastolic ventricular filling. If this condition is untreated, hemodynamic compromise ensues consequent to diminishing cardiac output. Therefore tamponade is considered a medical emergency. The risk of death, depends on the speed of diagnosis and treatment, the volume and rate of fluid accumulation and the cause of the tamponade.[4]

The common physical findings include attenuated apical heartbeat, distant heart sounds, and narrow pulse pressure. A drop in systolic pressure more than 10 mmHg, also known as pulsus paradoxus, is found in 77% of cardiac tamponade cases although it may be absent in low pressure or right atrial tamponade cases. Jugular venous distention (JVD) is almost always present in tamponade cases. Tachycardia and tachypnea are nonspecific findings.[5] Chest pain, is mostly positional, non-reproducible, and on the left side.

When cardiac tamponade is suspected, 2D echocardiography with Doppler should be obtained emergently. Regardless of

effusion size, cardiac tamponade is potentially lethal. Therefore, the most important echocardiographic findings are the presence of a pericardial effusion, a dilated IVC, hepatic veins indicating that systemic venous pressures are elevated, and a left ventricle that has reduced end-diastolic and end-systolic dimensions, with Doppler evidence of reduced stroke volume and cardiac output. In most cases of cardiac tamponade, other ‘‘classic’’ Doppler echocardiographic findings are also present and confirmatory. These include right heart diastolic chamber collapse when pericardial pressures exceed intra-cardiac pressure, an inspiratory bulge or ‘‘bounce’’ of the interventricular septum into the left ventricle, and characteristic abnormal respiratory changes in Doppler flow velocity recordings.[6]

Pericardial tamponade is a cardiac emergency. When feasible and consistent with the patient's goals of care, the primary treatment is pericardiocentesis. Intravenous fluids and vasopressors can temporarily augment cardiac output and systemic pressure, but these measures are considered temporizing until more definitive treatment with pericardiocentesis is performed. Echocardiographic-guided pericardiocentesis is preferred. Once the pericardial effusion is drained, a catheter is left in situ, until drainage is less than 100 mL over 24 hours. This can lead to a lower recurrence of tamponade. With regard to performing a pericardiocentesis versus an open surgical pericardial window, several issues are important to consider. First, pericardiocentesis can be performed expeditiously at the bedside, and is the procedure of choice in unstable patients, especially because patients with tamponade can deteriorate with induction of anesthesia. However, surgical pericardial window is the method of choice, if the effusion is localized, if safe percutaneous access is not possible, or in patients with recurrent effusions and in patients with malignancy, given that tamponade can reoccur with pericardiocentesis alone. Finally, pericardial biopsies, which can aid in diagnosis, can be performed with surgical procedure.[7]

## REFERENCES


1. **Imazio M, Gaita F, LeWinter M.** Evaluation and Treatment of Pericarditis: A Systematic Review. *JAMA.* 2015 Oct 13;314(14):1498-506. doi:10.1001/jama.2015.12763. Review. Erratum in: *JAMA*. 2015 Nov 10;314(18):1978. *JAMA*. 2016 Jan 5;315(1):90. Dosage error in article text. PubMed PMID: 26461998.
2. **Ben-Horin S, Bank I, Guetta V, Livneh A.** Large symptomatic pericardial effusion as the presentation of unrecognized cancer: a study in 173 consecutive patients undergoing pericardiocentesis. *Medicine (Baltimore).* 2006 Jan;85(1):49-53. Erratum in: *Medicine (Baltimore).* 2006 May;85(3):191. PubMed PMID: 16523053.
3. **Sagristà-Sauleda J, Angel J, Permanyer-Miralda G, Soler-Soler J.** Long-term follow-up of idiopathic chronic pericardial effusion. *N Engl J Med.* 1999 Dec 30;341(27):2054-9. PubMed PMID: 10615077.
4. **Guberman BA, Fowler NO, Engel PJ, Gueron M, Allen JM.** Cardiac tamponade in medical patients. *Circulation.* 1981 Sep;64(3):633-40. PubMed PMID: 6455217.
5. **Shabetai R, Fowler NO, Guntheroth WG.** The hemodynamics of cardiac tamponade and constrictive pericarditis. *Am J Cardiol.* 1970 Nov;26(5):480-9. Pub Med PMID: 5478837.
6. **Klein AL, Abbara S, Agler DA, Appleton CP, Asher CR, Hoit B, Hung J, Garcia MJ, Kronzon I, Oh JK, Rodriguez ER, Schaff HV, Schoenhagen P, Tan CD, White RD.** 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 Sep;26(9):965-1012.e15. doi:10.1016/j.echo.2013.06.023. PubMed PMID: 23998693.
7. **Vakamudi S, Ho N, Cremer PC.** Pericardial Effusions: Causes, Diagnosis, and Management. *Prog Cardiovasc Dis.* 2017 Jan - Feb;59(4):380-388. doi: 10.1016/j.pcad.2016.12.009. Epub 2017 Jan 4. Review. PubMed PMID: 28062268.


*Submitted March 13, 2018*