Review

Cardiac tamponade, a clinical challenge

Ahmed Abuzaid, MD[1]; Mohamed Elkashab, MD[2]; Marwan Saad, MD[3]

[1]Creighton University, Internal Medicine department, Omaha, NE, USA

[2]John H Stroger Hospital of Cook County, Cardiology department, Chicago, IL, USA

[3]Trinitas Regional Medical Center / Seton Hall University, Internal Medicine department, NJ, USA

Abstract: Pericardial effusion and cardiac tamponade can develop in patients with virtually any condition that affects the pericardium, including post-pericarditis, malignancies, chronic renal failure, thyroid disease, autoimmune disease, traumatic and idiopathic causes. Symptoms and signs lack both sensitivity and specificity. Appropriate diagnostic arcades can minimize the associated morbidity and mortality. Cardiologist should be aware of the physiological and clinical aspects of the disease spectrum.
Key Words: Pericardial effusion, Cardiac Tamponade, Pulse, Echocardiography

Introduction

Cardiac tamponade is a sort of cardiogenic shock and a medical emergency. A high index of suspicion with proper diagnostic arcades lessens the concomitant morbidity and mortality. The science behind the disease spectrum, diagnostic consideration, and relevant management will be discussed in the following review.

Definition, disease spectrum and pathophysiological consideration

Acute pericarditis is a clinical syndrome caused by inflammation of the pericardium and is associated with chest pain, a friction rub, and characteristic electrocardiographic changes. It appears to be more common in men and in young adults.[1] Pericardial effusion is a fluid collection in the pericardial space. Depending on the underlying etiology and rate of accumulation, the clinical presentation may range from being asymptomatic to life-threatening compromise.[1,2,3] There are a large number of potential etiologies for pericardial effusion. In practice, these are classified into idiopathic, infectious, inflammatory, metabolic, post-myocardial infarction, neoplastic, traumatic, radiation, dissection thoracic aneurysm, and certain drugs.[1,4,5]

The normal pericardium permits an unimpeded expansion of the ventricles during diastole[6]. Large effusion may be unexpectedly found without a significant elevation of intra-pericardial pressure and are usually asymptomatic, whereas rapidly accumulating small effusion may result in compressive physiology and hence tamponade, a condition involving an increase in the intra-pericardial pressure together with an elevation in intra-cardiac pressure and the progressive limitation

of ventricular filling during diastole and a reduction in cardiac output.[2,3] Pericardial compliance and the rate of fluid accumulation constitute a major role in determining the degree of diastolic filling compromise.[2,3,7] The cardiac output is initially maintained by a heightened adrenergic drive resulting in sinus tachycardia and peripheral vasoconstriction.[1] In severe cases, the compensatory mechanisms fail, resulting in a decreased cardiac output, reduced coronary perfusion, and ultimately the equalization of filling pressures between the cardiac chambers and surrounding stretched pericardium.[2,3,7,8] The absence of significant stretch of the compressed myocardium together with proportionate coronary mismatch explain the absence of elevated atrial naturetic peptides and actual acute unstable atherosclerotic coronary plaque inducing ischemic events, respectively.[9,10]

During respiration, reciprocal changes occur in the ventricles and are suggestive of ventricular interdependence. Increased venous return to the right ventricle together with limited ventricular expansion forcing the interventricular septum to bulge into the left ventricular chamber.[11]

Acute cardiac tamponade can occur within minutes due to trauma, rupture of the heart or great vessels, or as a complication of an invasive procedure, resembling cardiogenic shock that requires urgent drainage.[2,3,8] On the other hand, subacute cardiac variants are usually less dramatic and occur over days to weeks and usually due to nontraumatic causes.[1,2,3] In conditions with significant hypovolemia or over diuresis, low pressures (occult) tamponade occurs; a subset of subacute tamponade with good response to fluid challenge therapy.[12] The predominance of adrenergic drive with classic tamponade features characterizes the hypertensive variant.[13] Regional cardiac tamponade occurs when an eccentric effusion or localized hematoma produces regional cardiac compromise in which only selected chambers are affected, particularly in the postinfarction, essentially the right ventricle, or postcardiac surgery recovery periods.[14] Localized right atrial tamponade may also cause right to left shunting through a patent foramen ovale or an atrial septal defect resulting in significant acute hypoxia.[15]

Clinical presentations

Although cardiac tamponade is considered a clinical diagnosis, clinical findings of hypotension, tachycardia, elevated jugular venous pressure, and pulsus paradoxus are known to have a limited sensitivity and specificity.[2,3] The clinical presentations of cardiac tamponade usually reflects a state of low cardiac output state such as restlessness, drowsiness, decreased urine output, syncope, or near syncope.[10] Every effort is made to narrow the wide range of possible etiologies, particularly the acute, life-threatening tamponade, a variant of cardiogenic shock.[16] Constant dull aches or chest pressure may be confused with other chest pain syndromes. Pressure induced symptoms on surrounding thoracic structures, such as dysphagia, dyspnea, hiccups, nausea and abdominal fullness, can be misleading with the inappropriate deferral of care in the acute settings.[1,2,3]

Large pericardial effusion may be associated with muffled heart sounds and rales in the lung fields secondary to fluid compression.[1] Dullness to percussion and bronchial breathing below the left scapular angle is rarely appreciated (Ewart's sign). Sinus tachycardia and hypotension are signs of hemodynamic compromise. Tachycardia is usually absent in patients with hypothyroid and uremia. In advanced cases, pulseless electrical activity cardiac arrest can be a challenging scenario, where urgent pericadiocentesis can be life-saving where external chest compression is effortless.[7,17] A

careful examination of the peripheral pulse characteristics may anticipate the presence of pulses paradoxus with >10 mmHg drop in systolic pressure because of the impairment of left ventricular filling by the displaced septum during right ventricular filling and then a drop in systolic pressure readings, particularly in patients with tamponade, a phenomenon known by ventricular interdependence.[1] The assessment of pulsus paradoxus should always be performed during normal respiration because deep inspiration may result in false-positive readings.[8] Pulsus paradoxus is not specific for cardiac tamponade.[1,2,3,18] The most frequent causes of pulsus paradoxus without pericardial effusion or cardiac tamponade are bronchial asthma and chronic obstructive pulmonary disease, where the respiratory variation in intrathoracic pressure is greatly intensified.[8] Physicians should be aware of other conditions in which pulsus paradoxus may be present without cardiac tamponade that include obstructive sleep apnea, marked obesity, massive pulmonary embolism, profound hypovolemic shock, severe pectus excavatum, bilateral pleural effusion, right atrial mass, right ventricular myocardial infarction, tension pneumothorax, restrictive cardiomyopathy, and extrinsic cardiac compression.[2,3] Pulsus paradoxus may be absent during low pressure tamponade, as in the presence of dehydration and hypovolemia, and in conditions with raised ventricular diastolic pressure, as chronic hypertension, coexisting atrial septa defect, or significant aortic regurgitation.[4,18] Beck's triad describes the combination of venous distension, distant heart sounds, and absolute or relative hypotension. Pericardial rub may be audible, particularly in inflammatory pericarditis,[8] and cardiac apical impulse could be reduced or absent.[1]

The classic electrocardiogram finding in large effusive state consists of low voltage tracing.[19] Electrical alternans is a marker of massive effusion because of the swinging of heart along both axes.[19,20] Combined P and QRS waves alternation is fairly specific for pericardial tamponade.[21] Different stages of pericarditis can be found with diffuse ST elevation secondary to epicardial injury together with PR segment depression in the limb and chest leads with AVR lead being an exception.[1,19] For cardiomegaly to be evident in chest radiography a minimum of 200–250 mL of fluid has to be accumulated in the pericardial sac. Cardiomegaly with prominent superior vena cava and decreased pulmonary vascularity should suggest the diagnosis of pericardial effusion where pulmonary venous congestion is generally not observed in patients with significant effusion. If possible, lateral chest films can be used to detect large effusions.[12]

Diagnosis and workup

Transthoracic echocardiography (TTE) is the modality of choice for diagnosing and quantifying the amount of effusive fluid but is not useful in differentiating among the various etiologies.[4,5] An echo-free space of >50 mL between the two pericardial layers throughout cardiac cycle usually supports the diagnosis.[6] For circumferential pericardial effusions, the common Doppler echo quantitative measurements are as follows: any pericardial effusion with <5 mm of pericardial separation in diastole, corresponding to a fluid volume of 50–100 mL, is defined as minimal; 5–10 mm of separation as small, corresponding to a fluid volume of 100 to 250 mL; 10–20 mm of separation as moderate, corresponding to a fluid volume of 250 to 500 mL; and >20 mm of separation as large, corresponding to a fluid volume >500 mL.[4,5,20]

The echocardiographic findings of tamponade include pericardial effusion, right atrial diastolic collapse (a sensitive sign with 82% specificity),[21,22,23] right ventricular early diastolic

collapse (diastolic inversion which requires more pressure difference between the intrapericardial space and intracardiac chambers than right atrial collapse, imposing more specificity and lesser sensitivity in most current reports),[32] the respiratory variation of tricuspid and mitral valves inflow patterns by Doppler (a decrease in transmitral E wave >25% during inspiration is highly suggestive of cardiac tamponade. Tricuspid and pulmonary flow variation is more exaggerated than mitral and aortic flow initially before significant rise in intracardiac pressures ensue), inferior vena cava plethora (which can also masquerading constrictive physiology), and rarely but highly specific left atrial diastolic collapse (in 25 % of tamponade cases).[23,24,25]

Right ventricular diastolic collapse appears to be of significant importance among the medical patients population rather postoperative groups because of the loculated nature and presence of adhesions with possible hematomas.[26] Transesophegeal echocardiography is highly sensitive in such conditions.[27] Both right atrial and right ventricular collapses are subjected to current perfusion state. Right ventricular hypertrophy such as that in pulmonary hypertension can delay right ventricular collapse until significant intrapericardial pressure is existent.[25]

Cardiac tamponade is not an all-or-none occurrence but is rather a spectrum of findings.[26] Clinicians should correlate the echocardiographic signs of tamponade with the symptoms and clinical findings in each tamponade case.[16,18]

High resolution CT provides an excellent visualization of the pericardium together with the size, distribution, and nature of the accumulated fluid. MRI is usually not required in most settings but is very effective in the detection of loculated fluid and pericardial thickening.[16] However, the clinical utility of CT and MRI is questioned because of the high positive predictive value of echocardiography with potential safety concerns in acutely ill patients.[4,5,17,27,28] The utility of non-echocardiographic modalities can aid in the diagnosis in doubtful severe, complicated, and atypical presentations.[18,28]

A right heart catheterization (RHC) is not necessary in patients where clinical and echocardiographic findings are consistent with tamponade and, in fact, may delay the definitive treatment.[29] Borderline cases with an uncertainty of tamponade physiology could benefit from RHC in which the quantification of the hemodynamic compromise is of benefit.[2,3]

Management

Once the diagnosis of tamponade is confirmed, immediate drainage should be considered.[1] Optimal medical care, including volume expansion, the avoidance of diuretics or vasodilators, and inotropic support, if the patient is hypotensive, is paramount in the acute settings. Volume expansion has been proposed as an alternative; however, the scientific evidence for this recommendation is not widely approved by many institutions in the settings of cardiac tamponade due to poor evidence in practical settings[13]. In fact, excessive fluid administration can impede chambers dynamics with additive forces to an already suffering pericardium.[17] It appears that patients with baseline systolic arterial blood pressure <100 mmHg could benefit from rapid saline infusion to ensure hemodynamic stabilization awaiting pericardiocentesis, the definitive method of management. The watchful and brief use of inotropes is relatively controversial, where a higher resistance in the peripheral circulation is already established in the context of sympathetic overdrive, a condition where known inotropic agents adding arrythmogenicity.[29,30] Early

pericardiocentesis is life saving in patients who have pericardial effusion associated with bacterial pneumonia or empyema because the incidence of bacterial pericarditis is particularly high in this clinical situation to avoid the development of tamponade and subsequent chronic constrictive pericarditis among these population.[1,16,22,28]

The timing and method of drainage ultimately depends on the etiology of effusion, acuity level of patients, and availability of trained physicians. Percutaneous drainage provides a rapid, minimal preparation and less procedural mobidity.[4,5,18] On the other hand, surgical drainage by the creation of a pericardial window by an open surgery or a video-assisted thoracic approach allows complete drainage and permits the direct examination of the pericardium with access to the pericardial tissues for histopathology and microbiologic diagnosis. Percutaneous balloon pericardiotomy, described by Palacios et al.,[33] involves the use of a percutaneous balloon-dilating catheter to form a pericardial window to initially facilitate the malignant effusion drainage. Further studies and clinical report showed substantial success in pediatric population and non-neoplastic disease spectrum.[31] Balloon pericariodotomy appears to be a valid procedure to shorten surgical morbidity and concomitant anesthesia burden in part, and therefore should be considered upon institutional availability giving the low complication rates, similar to treatment by simple drainage.[31,32,33]

Conclusion

Pericardial effusion can develop in patients with virtually any condition that affects the pericardium, including acute pericarditis, subacute pericarditis, malignancies, pulmonary tuberculosis, chronic renal failure, thyroid disease, autoimmune disease, or iatrogenic and idiopathic causes. Symptoms and signs lack both sensitivity and specificity. TTE is the most important tool for diagnosis, grading, drainage, and follow-up. Cardiac tamponade is a sort of cardiogenic shock and a medical emergency. Clinicians should understand the physiology of cardiac tamponade, particularly in cases without large pericardial effusion, and correlate the signs of clinical tamponade together with the echocardiographic findings. The drainage of cardiac tamponade is life saving. A high index of suspicion with proper diagnostic arcades lessens the concomitant morbidity and mortality.

Conflicts of interest

The author has no financial disclosures to declare and no conflicts of interest to report.

Acknowledgment

REFERENCES
1. LeWinter MM. Pericardial diseases in Braunwald's Heart Disease, A Textbook of Cardiovascular Medicine, 8th Edition. Libby P, Bonow RO (eds). Saunders Elsevier, Philadelphia, Pennsylvania: pp. 1829–1854.
2. Spodick DH. Acute Cardiac Tamponade. The New England Journal of Medicine 2003;349(7):684– 690.
3. Spodick DH. How do the Clinical Findings in Patients with Pericardial Effusions Influence the Success of Aspiration? British Heart Journal 1995;74(4):476.
4. Pepi M, Muratori M. Echocardiography in the Diagnosis and Management of Pericardial Disease. Journal of Cardiovascular Medicine 2006;7(7):533–544.
5. Imazio M, Adler Y. Management of Pericardial Effusion." European Heart Journal 2013 34(16):1186–97.
6. Manning WJ. Pericardial disease in Goldman L, Ausiello D, editors. Cecil Medicine. 23rd ed. Philadelphia: Saunders/Elservier; 2008. pp. 548–552.
7. Ariyarajah V, Spodick DH. Cardiac Tamponade Revisited: A Postmortem Look at a Cautionary Case.Texas Heart Institute Journal / from the Texas Heart Institute of St.Luke's Episcopal Hospital, Texas Children's Hospital 2007;34(3):347–351.
8. Sagrista. SJ, Angel J, Sambola A, Permanyer G. Hemodynamic Effects of Volume Expansion in Patients with Cardiac Tamponade. Circulation 2008;117(12):1545–1549.

9. Roy CL, Minor MA, Brookhart MA, Choudhry NK. Does this Patient with a Pericardial Effusion have Cardiac Tamponade? JAMA: The Journal of the American Medical Association 2007;297(16):1810–1818.

10. Abusaid GH, Khalife WI. Reduced Coronary Blood Flow in Cardiac Tamponade: Mystery Solved. The Journal of Invasive Cardiology 2012;24(12):E328–9.

11. Khandaker MH, Espinosa RE, Nishimura RA, Sinak LJ, Hayes SN, Melduni RM, Oh JK. Pericardial Disease: Diagnosis and Management. Mayo Clin Proc 2010;85(6):572–593.

12. Cooper JP, Oliver RM, Currie P, Walker JM, Swanton RH. How do the Clinical Findings in Patients with Pericardial Effusions Influence the Success of Aspiration? British Heart Journal 1995;73(4):351–354.

13. Ramsaran EK, Benotti JR, Spodick DH. Exacerbated Tamponade: Deterioration of Cardiac Function by Lowering Excessive Arterial Pressure in Hypertensive Cardiac Tamponade. Cardiology 1995;86(1):77–79.

14. D'Cruz I, Rehman AU, Hancock HL. Quantitative Echocardiographic Assessment in Pericardial Disease. Echocardiography (Mount Kisco, N.Y.) 1997;14(2):207–214.

15. Varghese V, Mogtader A, George JC. Regional Cardiac Tamponade Resulting in Hypoxia from Acute Right to Left Inter–Atrial Shunting. The Journal of Invasive Cardiology 2011,23(5):E96–8.

16. Sagrista J, Merce AC, Soler J. Diagnosis and Management of Pericardial Effusion. World Journal of Cardiology 2011,3(5):135–143.

17. Hashim R, Frankel H, Tandon M, Rabinovici R. Fluid Resuscitation–Induced Cardiac Tamponade. The Journal of Trauma 2002;53(6):1183–1184.

18. Jung HO. Pericardial Effusion and Pericardiocentesis: Role of Echocardiography. Korean Circulation Journal 2012;42(11):725–734.

19. Spodick DH. Images in Cardiology. Truly Total Electric Alternation of the Heart. Clinical Cardiology 1998;21(6):427–428.

20. Ivens EL, Munt BI, Moss RR. Pericardial Disease: What the General Cardiologist Needs to Know. Heart (British Cardiac Society) 2007;93(8):993–1000.

21. Seidenberg PH, Haynes J. Pericarditis: Diagnosis, Management, and Return to Play. Current Sports Medicine Reports 2006;5(2):74–79.

22. Adler Y. Management of Pericardial Effusion." European Heart Journal 2012;34(16):1186–97.

23. Little WC, Freeman GL. Pericardial Disease. Circulation 2006;113(12):1622–1632.

24. Reydel B, Spodick DH. Frequency and Significance of Chamber Collapses during Cardiac Tamponade. American Heart Journal 1990;119(5):1160–1163.

25. Wann S, Passen E. Echocardiography in pericardial disease. J Am Soc Echocardiogr 2008;21(1):7–13.

26) Holmes DR. Iatrogenic Pericardial Effusion and Tamponade in the Percutaneous Intracardiac Intervention Era. JACC;Cardiovascular Interventions 2009;2(8):705–717.

27. Singh S, Wann LS, Schuchard GH, Klopfenstein HS, Leimgruber PP, Keelan MH, Brooks HL. Right Ventricular and Right Atrial Collapse in Patients with Cardiac Tamponade– a Combined Echocardiographic and Hemodynamic Study. Circulation 1984;70(6):966–971.

28. Verhaert D, Gabriel RS, Johnston D, Lytle BW, Desai M, Klein AL. The Role of Multimodality Imaging in the Management of Pericardial Disease. Circulation.Cardiovascular Imaging 2010;3(3):333–343.

29. Meltser H, Kalaria VG Cardiac Tamponade." Catheterization and Cardiovascular Interventions: Official Journal of the Society for Cardiac Angiography & Interventions 2005;64(2):245–255.

30. Du B, Hu X, Weng L. Comparison of Dopamine and Norepinephrine in Shock. The New England Journal of Medicine 2010,362(24):2330.

31. Martins JB, Manuel WJ, Marcus ML, Kerber RE. Comparative Effects of Catecholamines in Cardiac Tamponade: Experimental and Clinical Studies.The American Journal of Cardiology 1980;46(1):59–66.

32. Swanson N, Mirza I, Wijesinghe N, Devlin G. Primary percutaneous balloon pericardiotomy for malignant pericardial effusion.Catheter Cardiovasc Interv 2008;71(4):504–507.

33. Del Barrio LG, Morales JH, Delgado C, Benito A, Larrache J, Martinez–Cuesta A, Bilbao JI. Percutaneous balloon pericardial window for patients with symptomatic pericardial effusion. Cardiovasc Intervent Radiol 2002;25(5):360–4.