

Pericardial effusion and developing tamponade in a 30-year-old man

The cause sometimes remains unclear, but cardiac tamponade is potentially fatal. A rapid diagnosis and appropriate treatment are essential to saving the patient.

Kristen Frank, BS; Fred Weber, MD, JD

CASE

A 30-year-old white male arrived at the emergency department complaining of chest discomfort of 2 weeks' duration. He described the discomfort as a constant, retrosternal, left-sided pressure that had begun gradually and worsened over time. In the past 2 days, it had progressed to a constant, sharp, left-sided chest pain that radiated to his left shoulder. He denied alleviating or aggravating factors, fever, chills, changes in weight, bruising, or bleeding.

The patient had a significant medical and traumatic history. In a motor vehicle accident 15 months previously, he sustained a right hemothorax with pulmonary contusion, multiple rib fractures, grade 5 liver laceration, right renal injury, and L1 and L2 anterior body fractures. He had a large ventral hernia due to abdominal compartment syndrome at the time of the trauma, with subsequent inability to close the abdomen.

The patient was not taking any medications and had no known allergies to medications or dyes. He was employed as a bartender and lived with a friend. He denied use of cigarettes. He was admitted for investigation of his chest pain.

Physical examination On admission, the patient was awake and in moderate distress. BP was 120/70 mm Hg with a 10-mm Hg paradoxical pulse. Heart rate was 70 beats per minute (bpm); respirations, 15 breaths per minute; and temperature, 98°F. The external jugular veins were minimally dilated and did not collapse on deep inspiration. The pericardium was quiet, with no rubs, murmurs, or gallops. The lungs were clear. There was a large, protruding ventral hernia from the xiphoid to below the umbilicus.

Testing The results of a CBC and electrolytes testing were normal. Creatine kinase MB-fraction, and troponin levels were normal. The result of a D-dimer test was high at 2,059 μ /L. The ECG revealed normal sinus rhythm with left atrial enlargement. There were no significant QRS complex or T-wave changes. CT showed a pericardial effusion, and echocardiography confirmed that a pericardial effusion completely surrounded the heart (see Figure 1).

Preoperative course On the second hospital day, the patient became agitated and dyspneic. His chest pain continued to increase and was worse in a supine position and when he breathed deeply. BP was 128/78 mm Hg. Heart rate was 110 bpm. The neck veins were full and did not collapse on deep inspiration. Repeat echocardiography showed that the pericardial effusion was becoming larger, with collapse of the right atrium. A thoracic surgeon was consulted and made a diagnosis of pericardial effusion with tamponade.

Surgery Because of the patient's previous open abdomen with midline fascial dehiscence, the pericardium was approached via a thoracotomy at the left anterior sixth inter-space. The pericardium was thickened, and 600 cc of serosanguinous fluid was present in the pericardial space. The epicardium was erythematous with a thick fibrinous exudate. A pericardial window of 4 × 4 cm was created by excising the parietal pericardium. The pericardial and pleural spaces were drained with thoracic catheters.

Postoperative course On the first postoperative day, the patient was awake and alert. A pericardial friction rub was present in the immediate postoperative period but disap-

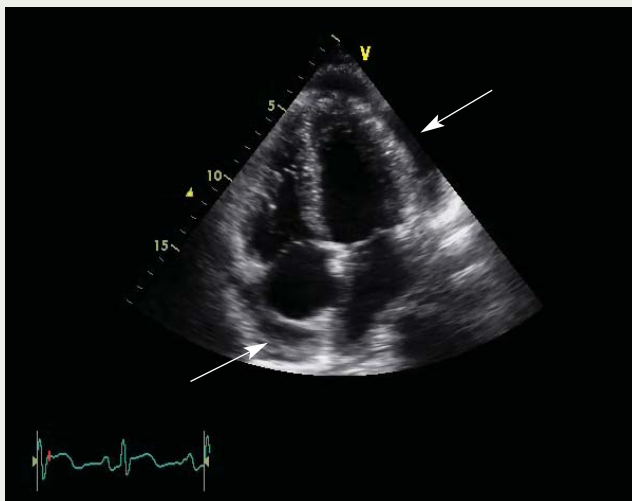


FIGURE 1. Pericardial effusion as seen on an echocardiogram

CASE REPORT | Cardiac tamponade

peared within 12 hours. The patient reported that his chest pain had decreased significantly and he was no longer short of breath. A chest film showed appropriate chest tube placement. Pleural and pericardial tubes each drained 300 cc of fluid.

On the second postoperative day, an additional 100 cc of fluid was collected via each chest tube. Heart rate was 94 bpm; respiratory rate, 16 breaths per minute; and temperature, 98°F. An ECG showed normal sinus rhythm. Auscultation revealed normal heart sounds without friction rub. Bowel sounds were slightly increased. On the third day, drainage was less than 40 cc for both tubes, and they were removed. The heart sounds continued to be normal. The abdomen was mildly distended at the ventral hernia. The patient was discharged home on the fourth day and continues to do well.

A clinical diagnosis of fibrinous pericarditis was made. The pathologist reported that the specimen was consistent with fibrinous pericarditis with no evidence of malignancy. All bacterial cultures were sterile.

DISCUSSION

Pericardial effusion is the accumulation of fluid within the pericardial sac surrounding the heart. If the fluid collects rapidly or reaches a large volume, the venae cavae and atria will be compressed and restrict cardiac inflow. Cardiac tamponade, manifested by hypotension followed by circulatory collapse, will result. The diagnosis and treatment of pericardial effusion and cardiac tamponade require a high index of suspicion and understanding of the underlying physiology.

The first description of fibrinous pericarditis is attributed to Antonio Benivieni, a 15th century Florentine physician.¹ Laennec described pericarditis in 1822 as “inflammation of the serous membrane which lines the fibrous sac of the pericardium, the heart and the great vessels. ... marked by redness ... and a sero-purulent effusion.”² Laennec also described pericardial effusion under the heading of “hydro-pericardium,” where the “effused serum is sometimes colourless [sic], but more commonly is yellowish, brownish, or reddish....”²

In 1919, Sir William Osler described pericarditis with effusion. He correctly noted the signs of pericardial rub, distended neck veins, and pulsus paradoxus; however, he could not elu-

cidate the physiology. There was a mention of “paracentesis of the pericardium,” but the results have “so far not been satisfactory.”³ Lewis in 1930 advocated a left parasternal incision with resection of the costal cartilages for pericardial drainage.⁴

In 1937, when Beck described the physiology of pericardial effusion leading to pericardial tamponade, now called *cardiac tamponade*, he ushered in a new understanding and treatment of this condition.⁵ Beck found that the heart discharges only the quantity of blood that is returned to the right atrium and that flow in the venae cavae is restricted when a pericardial effusion is present. He correctly reasoned that the “compression [within the pericardium] prohibits the heart from receiving its normal quota of blood. In as much as it receives a

“Pericardial effusion may develop in many diseases and settings, and the etiology may be determined in most but not all patients.”

subnormal quota of blood, it actually pumps out a subnormal quantity of blood....”⁵ Beck is remembered for his diagnostic triad for acute compression: (1) a small, quiet heart; (2) venous hypertension; and (3) arterial hypotension.⁵

Etiology Pericardial effusion may develop in a variety of diseases and settings, and the etiology may be determined in most but not all patients. In a 2004 report, 30% of effusions were due to metastatic cancer, 22% to chronic renal failure, 10% to viral pericarditis, 4% to Dressler’s syndrome, and one case each to tuberculosis, blunt chest trauma, and purulent pericarditis.⁶ Viral pericarditis was suspected in our patient. In 20% of patients, a definite etiology could not be found.⁶ The diagnosis of viral pericarditis was suspected in patients who had a recent history of respiratory infection together with signs of inflammation and an exudative effusion.⁶ Other rare causes of pericardial effusion include HIV infection, recent or remote mediastinal irradiation, and hypothyroidism.⁷

Diagnosis Symptoms of pericardial effusion may initially be mild and nonspecific. As the fluid increases, dyspnea, chest

TEACHING POINTS

- Pericardial effusion and tamponade require a high index of suspicion for diagnosis, along with an understanding of the underlying physiology.
- Both the quantity of fluid and the rate of accumulation are important in the development and timing of cardiac tamponade. If the fluid collects rapidly or reaches a large volume, the venae cavae and atria will be compressed and restrict cardiac inflow. Cardiac tamponade, manifested by hypotension followed by circulatory collapse, will result.
- Treatment consists of drainage of the pericardium. Both the subxiphoid pericardial window and thoracotomy with pericardial window are safe and effective treatments for this disease.
- The patient in this case had fibrinous pericarditis resulting in cardiac tamponade. Treatment by anterior thoracotomy with pericardial window was appropriate, given the size of the effusion and the presence of a ventral hernia.

COMPETENCIES

- Medical knowledge
- Interpersonal & communication skills
- Patient care
- Professionalism
- Practice-based learning and improvement
- Systems-based practice

pain, chest tightness, or chest fullness may develop. Non-specific symptoms such as lethargy, fever, cough, weakness, fatigue, anorexia, and palpitations may be present.⁸ Patients are frequently admitted with these vague complaints, studies are ordered, and the pericardial effusion is subsequently discovered—as happened with the patient in this case.

Although a small pericardial effusion may produce no clinical signs, both the absolute amount of fluid and the rate of accumulation may eventually cause an effusion to become hemodynamically significant; at this point, classic signs of cardiac tamponade appear. These include Beck’s triad, cyanosis, pulsus paradoxus, a positive Kussmaul’s sign, and a pericardial friction rub. Florid tamponade with cyanosis, neck vein distension, and hypotension is easily recognizable.

Pulsus paradoxus is an exaggeration of the normal inspiratory decrease in BP. It can be detected by palpating the radial pulse and noting an inspiratory diminution of the pulse during normal respiration. Calculation of the paradox requires measuring the systemic BP with a sphygmomanometer or radial artery line. Most textbooks define *pulsus paradoxus* as a greater than 10-mm Hg difference between the initial detection of sounds on expiration and the constant presence of sounds with each heartbeat through the respiratory cycle.⁹

Patients with pericardial effusion and especially cardiac tamponade have dilatation of the external jugular veins. When the patient takes a deep breath, the neck veins collapse and drain. The failure of the veins to collapse upon deep inspiration is a positive Kussmaul’s sign and reflects compression of the superior and inferior venae cavae within the pericardium. Venous distention can occur in other settings such as superior vena caval syndrome.

In patients with fibrinous pericarditis, a pericardial friction rub may be present. This rub typically waxes and wanes, and on auscultation, the sound varies from scraping to grating.¹ A friction rub is present in the immediate period following pericardial window and usually disappears within 12 hours. The appearance of the friction rub may be attributed to the fluid removal, allowing the visceral and parietal pericardium to rub against each other.

TABLE 1. Treatment options for pericardial effusion and cardiac tamponade

Small effusion
Observation
Pericardiocentesis (with echocardiographic or fluoroscopic guidance)
Moderate to large effusion and tamponade
Open thoracotomy with pericardial window
Pericardiocentesis (with echocardiographic or fluoroscopic guidance)
Subxiphoid incision with pericardial window
Video-assisted thorascopic pericardiectomy (VATS)

The diagnosis of cardiac tamponade is suspected on clinical grounds and is confirmed with echocardiography. Echocardiographic features of cardiac tamponade include right atrial and right ventricular collapse, dilated inferior cava with a lack of inspiratory collapse, and in extreme cases, a swinging heart (in which an extremely large effusion causes the heart to oscillate within the pericardium).¹⁰ Many effusions are detected first on CT, and a chest film may show the characteristic “water-bottle heart,” resembling a flask with equal and symmetrical rounded flanges at both the right and left cardiac borders. This cardiac shadow has also been described as “globular.”⁸

Treatment The amount of hemodynamic compromise, the clinical assessment, and serial echocardiograms determine the treatment for pericardial effusion (see Table 1). Small effu-

“The procedure of choice for patients with cardiac tamponade has been subxiphoid incision with pericardial window.”

sions may require only observation and careful monitoring, with treatment directed to the underlying cause (such as dialysis for uremic effusions or discontinuance of minoxidil in drug-related cases).

When the effusion progresses to tamponade with hemodynamic compromise, urgent surgical removal of the pericardial fluid is indicated. Treatments include pericardiocentesis, subxiphoid pericardial window, left anterior thoracotomy with pericardial window, video-assisted thorascopic pericardiectomy (VATS), and median sternotomy with complete pericardiectomy.¹¹

Subxiphoid incision with pericardial window has been the procedure of choice for patients with cardiac tamponade because it is accomplished quickly, is associated with minimal morbidity, and prevents recurrent tamponade in 97% of patients.¹² A small incision is made on the upper chest centered on the xiphoid cartilage. The midline fascia is opened and the peritoneum is not opened. The retrosternal plane is developed, and the pericardium is visualized. A small pericardiectomy releases the pericardial pressure. A 4 × 4-cm opening in the pericardium is fashioned, and the specimen is submitted for pathologic examination. One or two pericardial tubes are placed in the pericardial well. The drainage tubes are not removed until the drainage is less than 20 to 50 cc per day.

Both the subxiphoid and anterior thoracotomy approaches have been well tolerated by patients, required short operative times, and resulted in similar rates of overall postoperative complications and length of stay in the ICU and hospital.¹³ Recurrence rates were low with both procedures.¹³

Continued on page 38

“Video-assisted thoracoscopic pericardiectomy had better long-term control of effusion than subxiphoid pericardial window.”

A comparison study between subxiphoid pericardial window and video-assisted thoracoscopic pericardiectomy found that although operative time and minor procedural morbidity are higher with VATS, VATS had better long-term control of effusion than subxiphoid pericardial window.¹⁴ A disadvantage of the VATS procedure is that when tamponade is present, pericardiocentesis must be performed prior to surgery.¹⁴

SUMMARY

Pericardial effusion and tamponade require a high index of suspicion for diagnosis. Both the quantity of fluid and the rate of accumulation are important in the development and timing of cardiac tamponade. Treatment consists of drainage of the pericardium. Both the subxiphoid pericardial window and anterior thoracotomy with pericardial window are safe and effective treatments. Our patient had fibrinous pericarditis resulting in cardiac tamponade. Treatment by anterior thoracotomy with pericardial window was appro-

priate, given the size of the effusion and the presence of a ventral hernia. [JAAPA](#)

Kristen Frank is a senior student in the Drexel University Hahnemann Physician Assistant Program, Philadelphia, Pennsylvania, and a graduate of Villanova University. **Fred Weber** is an assistant professor of surgery at Drexel University and a thoracic surgeon in Somers Point, New Jersey. The authors have indicated no relationships to disclose relating to the content of this article.

REFERENCES

1. Cohen MB. Cross your heart: some historical comments about fibrinous pericarditis. *Hum Pathol.* 2004;35(2):147-149.
2. Laennec RTH. *A Treatise on the Diseases of the Chest.* New York, NY: Library of the New York Academy of Medicine, Hafner Publishing Co; 1962.
3. Osler W. *The Principles and Practice of Medicine.* New York, NY: D. Appleton and Co; 1919.
4. Lewis D. *Practice of Surgery.* Hagerstown, MD: W. F. Prior Co Inc; 1930.
5. Beck CS. Acute and chronic compression of the heart. *Am Heart J.* 1937;14(5):515-525.
6. Kabukeu M, Demireoghi F, Yanik E, et al. Pericardial tamponade and large pericardial effusions. *Tex Heart Inst J.* 2004;31(4):398-403.
7. Ojeda W, Martinez-Toro JA. Diagnosis and management of pericardial effusions. *P R Health Sci J.* 2006;25(3):255-258.
8. Roy CL, Minor MA, Brookhart MA, Choudhry NK. Does this patient with a pericardial effusion have cardiac tamponade? *JAMA.* 2007;297(16):1810-1818.
9. Swami A, Spodick DH. Pulsus paradoxus in cardiac tamponade: a pathophysiologic continuum. *Clin Cardiol.* 2003;26(5):215-217.
10. Fowler NO. Cardiac tamponade: a clinical or an echocardiographic diagnosis. *Circulation.* 1993; 87(5):1738-1741.
11. Guberman BA, Fowler NO, Engel PJ, et al. Cardiac tamponade in medical patients. *Circulation.* 1981;64(3):633-640.
12. Moores DWO, Allen KB, Faber LP, et al. Subxiphoid pericardial drainage for pericardial tamponade. *J Thorac Cardiovasc Surg.* 1995;109(3):546-552.
13. Lieberman M, Labos C, Sampalis JS, et al. Ten-year surgical experience with nontraumatic pericardial effusions. *Arch Surg.* 2005;140(2):191-195.
14. O'Brien PKH, Kucharczuk JC, Marshall MB, et al. Comparative study of subxiphoid versus video-thoracoscopic pericardial "window." *Ann Thorac Surg.* 2005;80(6):2013-2019.

We need peer reviewers!



Join our peer reviewer database, and contribute your expertise to the AAPA's official clinical journal.

Reviewers can expect to receive 3 or 4 manuscripts a year on topics in their area of expertise. We provide a detailed review form along with the manuscript.

If you're interested, please e-mail the editor at: tanya.gregory@haymarketmedical.com.

Include a statement listing your areas of expertise, and attach a current CV to your message.

JAAPA