Management of Cardiac Tamponade

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Case Presentation

A 64-year-old man with a remote history of stage IIIa adenocarcinoma of the lung treated with chemotherapy and radiation presented to the emergency department complaining of left-sided chest pain and dyspnea. He had been diagnosed with a pulmonary embolism 2 weeks prior and was started on warfarin at that time. He felt well until the night before his current presentation when he became acutely dyspneic while lying in bed. His triage vital signs were notable for a heart rate of 106 beats per minute (bpm), blood pressure of 92/70 mmHg, and respiratory rate of 28 breaths per minute. A pulsus paradoxus of 18 mmHg was measured by manual sphygmomanometer. A 12-lead ECG showed sinus tachycardia with low-normal QRS voltages (Fig. 14.1). A chest X-ray showed stable reticular opacities in the right middle and lower lobes at the sites of prior radiation treatment, as well as a prominent cardiomediastinal silhouette. A bedside echocardiogram was performed which showed a large circumferential pericardial effusion with early right ventricular (RV) diastolic collapse (Fig. 14.2) and exaggerated reciprocal respiratory variation in mitral and tricuspid early-diastolic inflow velocities (Fig. 14.3 and Supplementary Video 14.1).

Question

What is the appropriate next step in the management of the patient's pericardial effusion?

Answer With few exceptions, patients with clinical and supportive echocardiographic evidence of cardiac tamponade should undergo emergent drainage of the pericardial effusion by percutaneous needle pericardiocentesis. Isotonic fluids can modestly increase cardiac output and mean arterial pressure in about half of patients with tamponade [1] but the results are generally transient, and this intervention should not substitute for or delay pericardiocentesis.

In this case, the patient was given a 500 mL bolus of normal saline over 10 min with transient improvement in his systolic blood pressure. The cardiac catheterization laboratory was activated and the patient was given two units of fresh frozen plasma to reverse a supratherapeutic INR of 4.2. The pericardial space was accessed through a subxiphoid approach using echocardiographic and fluoroscopic guidance. The pericardial pressure was measured at 24 mmHg. A pericardial drain was placed, 850 mL of bloody fluid was removed, and the pericardial pressure was reduced to 0 mmHg. His symptoms dramatically improved and his blood pressure increased to 132/78 mmHg. He was admitted to the cardiac intensive care unit for ongoing monitoring, and over the ensuing 48 h, the output from his pericardial drain tapered off to zero. A repeat transthoracic echocardiogram showed a small residual pericardial effusion without evidence of tamponade, and the drain was removed. Pericardial fluid analysis revealed a markedly elevated red blood cell count (2 million/μL) with negative culture and cytology. The clinical picture was felt to be consistent with a hemorrhagic pericardial effusion due to excessive anticoagulation in the setting of subclinical radiation-induced pericardial disease.

Principles of Management

Hemodynamic Derangements

Cardiac tamponade occurs when fluid accumulates in the intrapericardial space, increasing intrapericardial pressure

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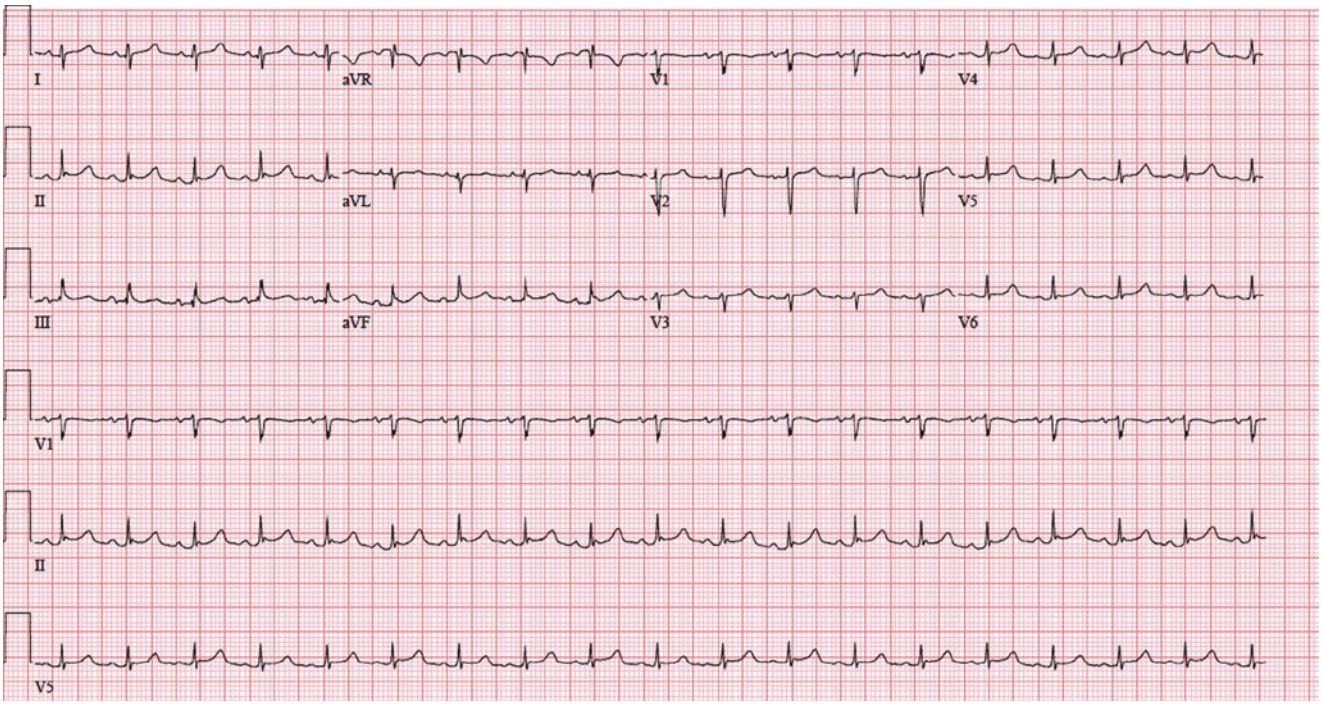

Fig. 14.1 Admission ECG

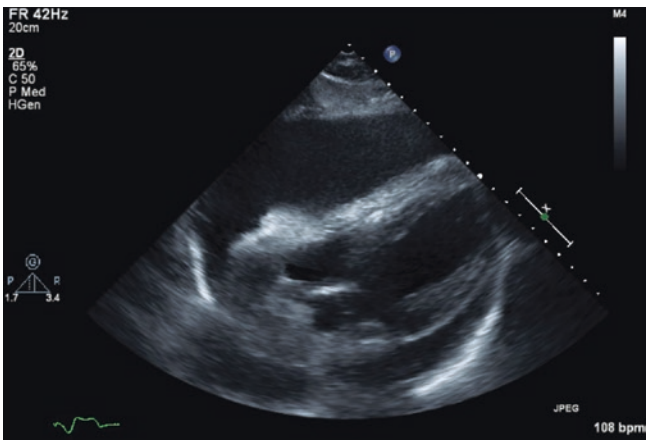

Fig. 14.2 Subcostal view showing large circumferential pericardial effusion with early right ventricular diastolic collapse

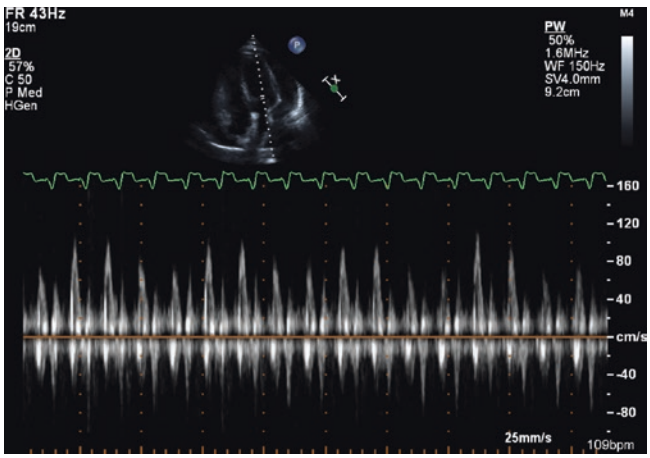

Fig. 14.3 Pulse-wave Doppler of mitral inflow in the apical four chamber view, showing >25% respirophasic variation in diastolic inflow velocities

and impairing cardiac filling [2]. Tamponade is a continuum from mild impairment in cardiac filling to complete circulatory collapse [3]. The primary determinant of the hemodynamic significance of a pericardial effusion is the intrapericardial pressure, which is related to the volume of the effusion and the pericardial pressure-volume relationship. The latter is heavily influenced by the chronicity of the effusion, and hence slowly accumulating pericardial fluid can lead to a large effusion without the development of tamponade [4]. As intrapericardial pressure increases, right and left-sided atrial and ventricular pressures also increase to maintain end-diastolic volume. At some point, generally in the range of 20–25 mmHg, the intrapericardial pressure approaches intracavitary pressures with consequent reduction in ventricular transmural pressure and end-diastolic volume [5]. The heart attempts to maintain cardiac output by increasing contractility and heart rate, but these compensatory mechanisms are quickly exhausted and progressive circulatory collapse ensues.

Clinical Findings

The classical findings of cardiac tamponade were reported in 1935 by a thoracic surgeon named Claude Beck, who described the triad of hypotension, elevated jugular venous pressure, and muffled heart sounds in a series of surgical patients with cardiac tamponade due to intrapericardial hemorrhage [6]. Although this constellation of clinical signs has remained the core clinical triad of tamponade, individual components may not be seen in all patients and often do not occur simultaneously within the same patient. A variant form of cardiac tamponade associated with systemic hypertension has also been described [7], and pericardial friction rubs can sometimes be heard in lieu of muffled heart sounds in patients with concomitant pericarditis [8]. Patients with cardiac tamponade often complain of dyspnea and feel more comfortable leaning forward. As they progress along the continuum of hemodynamic derangement, they appear increasingly uncomfortable and have varying manifestations of systemic malperfusion and adrenergic activation (e.g., tachypnea, diaphoresis, altered mental status).

The hallmark of cardiac tamponade is a paradoxical pulse (i.e., pulsus paradoxus), which is defined by a drop in systolic pressure of greater than 10 mmHg during inspiration [2]. This occurs because the total intracardiac volume is relatively fixed due to the elevated intrapericardial pressure. As venous return to the right side of the heart increases with inspiration, the interventricular septum shifts to the left in an exaggerated fashion that further reduces left ventricular (LV) stroke volume. The pulsus paradoxus can be measured by cuff sphygmomanometry as the difference between the systolic pressure when Korokoff sounds first appear and when they appear continuously. In the intensive care unit, it can also be measured by pulse oximetry waveform analysis or arterial waveform analysis when an arterial line is present [9]. The patient should not be asked to breathe deeply during blood pressure measurement since this can falsely exaggerate blood pressure variation over the respiratory cycle. It is important to remember that several other conditions can produce a pulsus paradoxus, including constrictive pericarditis, pulmonary embolism, hypovolemic shock, and severe obstructive lung disease.

Non-invasive Diagnostic Testing

A 12-lead electrocardiogram (ECG) should be obtained in all patients with suspected cardiac tamponade. The characteristic abnormalities seen on ECG are decreased QRS voltage and electrical alternans. Low QRS voltage is a non-specific finding that is also seen in infiltrative myocardial disease, pulmonary disease, and obesity. Electrical alternans, defined as beat-to-beat variation in QRS amplitude related to anterior-posterior swinging of the heart, is not sensitive but is relatively specific for cardiac tamponade [10]. The combination of P wave and QRS alternans further increases specificity [10].

Transthoracic echocardiography is the imaging modality of choice for evaluating the size, location, and degree of hemodynamic impairment caused by a pericardial effusion [11, 12]. Several echocardiographic findings support the diagnosis of tamponade: [12, 13]

1. Right atrial inversion for greater than one-third of systole
2. Right ventricular diastolic collapse (best appreciated in the parasternal long-axis and subcostal views)
3. Reciprocal respiratory variation in RV and LV volumes and consequent septal shifting (best appreciated in the apical four-chamber view)
4. Exaggerated reciprocal respiratory variation (>25%) in mitral and tricuspid early-diastolic inflow velocities (i.e., E velocities)
5. Increase in the flow velocity integral in the pulmonary artery and decrease in the flow velocity integral in the aorta during inspiration (i.e., "echocardiographic pulsus paradoxus")
6. Reduced early-diastolic mitral annular tissue Doppler velocity (i.e., E′ velocity)
7. Severe dilation of the inferior vena cava (IVC) (i.e., IVC plethora)

It is important to remember that cardiac tamponade is a clinical and hemodynamic diagnosis. If the clinical picture is consistent with cardiac tamponade, the most important echocardiographic finding is the presence of a pericardial effusion. In this case, Doppler evaluation should not delay expeditious treatment.

Invasive Diagnostic Testing

Invasive hemodynamic monitoring with a pulmonary arterial catheter can provide additional evidence for the diagnosis of cardiac tamponade. Supportive findings include equalization of diastolic pressures between cardiac chambers, which produces a characteristic "blunted" y-descent in the right atrial tracing (Fig. 14.4), and reciprocal respirophasic variation in right and left-sided filling pressures [10]. Invasive monitoring is generally not necessary for diagnosing tamponade and should be reserved for circumstances in which there is diagnostic uncertainty (e.g., hypertensive cardiac tamponade). It is also necessary for the diagnosis of effusive-constrictive pericarditis (see section "Evidence Contour") [14].

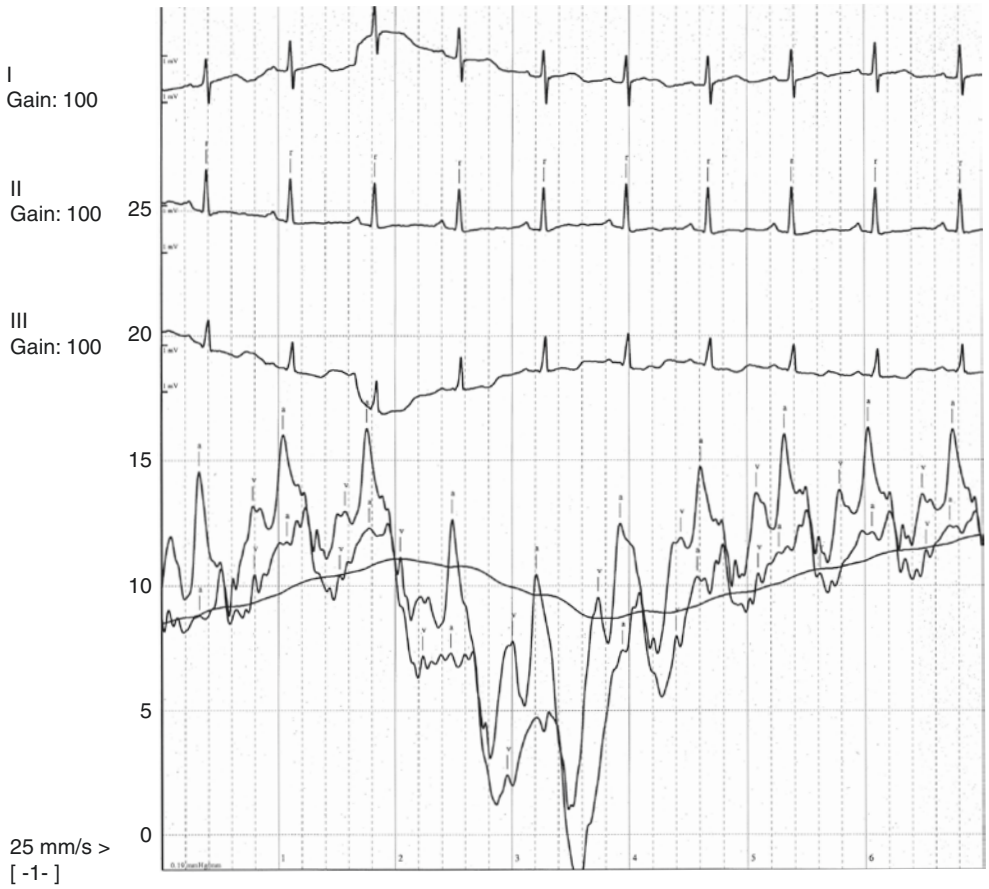

Fig. 14.4 Right atrial tracing demonstrating blunted y-descent in a patient with cardiac tamponade

Closed Pericardiocentesis

In most cases, the treatment of cardiac tamponade should be oriented toward emergent drainage of the pericardial effusion by percutaneous needle pericardiocentesis. Intravascular volume expansion with isotonic fluid resuscitation can lead to modest and transient increases in cardiac output and systolic blood pressure in about half of patients. Since volume expansion also acutely increases left ventricular diastolic pressures, it is generally recommended that no more than 500 mL be administered [1, 15]. Positive inotropes are of limited efficacy because endogenous adrenergic activation is generally near maximal [2]. Intubation should be avoided because positive-pressure mechanical ventilation will further reduce ventricular transmural pressure and diastolic filling [10].

Before proceeding with closed pericardiocentesis, it should be confirmed that there is clear clinical evidence of tamponade (including a pulsus paradoxus >10 mmHg), and that the effusion is large enough anteriorly to safely access the fluid via a percutaneous approach. Whenever possible, the procedure should be performed by an experienced provider in the cardiac catheterization laboratory. In the setting of circulatory collapse, a bedside pericardiocentesis may be performed emergently. Real-time transthoracic echocardiographic guidance is often used to identify the optimal percutaneous approach (generally subxiphoid) and has been shown to reduce procedural complications including myocardial puncture [16–18]. When the procedure is done in the cardiac catheterization laboratory, fluoroscopic guidance and invasive hemodynamic monitoring can also be useful. Once the pericardial space has been accessed, a guidewire is passed through the sheath to facilitate introduction of a pigtail catheter [19]. Intrapericardial pressure should be measured prior to fluid removal, and the pericardial fluid analysis should include specific gravity, cell count and differential, total protein content, gram stain and culture for detection of bacteria (including tuberculosis) and fungi, and cytology. When the amount of pericardial fluid drained decreases to less than 50 mL per day, the catheter can generally be removed [10]. In rare cases, paradoxical hemodynamic deterioration and pulmonary edema associated with ventricular dysfunction have been reported after pericardial drainage. Known as pericardial decompression syndrome (PDS), this complication remains poorly understood [20].

Surgical Drainage

Open pericardiocentesis is the preferred approach for treating tamponade that results from intrapericardial bleeding due to myocardial rupture (e.g., post-myocardial infarction) or aortic dissection. Loculated effusions and effusions with excessive fibrinous material (e.g., clotted hemopericardium) may also require a surgical approach. In these cases, surgery is generally performed through a limited subxiphoid incision.

Most malignant pericardial effusions can be treated with closed pericardiocentesis with a low recurrence rate [21, 22]. When hemodynamically significant malignant effusions do recur, they should generally be treated with open pericardiocentesis and creation of a pericardial window [19]. Multiple pericardial biopsies, with or without pericardioscopic guidance, should be obtained at the time of surgery [23].

Evidence Contour

Effusive-Constrictive Pericarditis

Effusive-constrictive pericarditis (ECP) is a clinical syndrome in which constriction of the visceral pericardium occurs in the presence of a tense pericardial effusion. It has been best characterized in patients presenting with cardiac tamponade who have persistently elevated right atrial pressure (i.e., failure to fall by 50% or to a new level below 10 mmHg) after removal of the pericardial fluid [24]. It is estimated that ECP complicates 5–10% of cases of clinical tamponade, though there is significant geographic variation in the prevalence [24]. In most cases, the definitive treatment of ECP is pericardiectomy.

Since invasive hemodynamic monitoring is not routinely needed to diagnose cardiac tamponade, there is interest in developing non-invasive criteria to identify patients at higher risk of ECP who should undergo cardiac catheterization at the time of pericardiocentesis [14]. Echocardiography and cardiac magnetic resonance imaging have been explored but not yet systematically correlated with invasive parameters [14, 25]. In addition, it has been suggested that patients with ECP have a distinct pattern of immune activation when compared with patients who have effusive but non-constrictive pericardial disease [26]. Further investigation into these differences may ultimately lead to the identification of novel serum or pericardial biomarkers for the diagnosis of ECP.

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