Myth exploded

Unraveling the paradox of cardiac tamponade: case presentation and discussion of physiology

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Summary

A 53-year-old man on warfarin for postoperative pulmonary embolism presented with chest pain and was found to be in cardiac tamponade due to an atraumatic haemopericardium. Findings of tamponade and a novel approach to the pathophysiology of pericardial disease to explain these findings are presented.

BACKGROUND

Illnesses attributable to abnormalities of the pericardium have intrigued and puzzled physicians throughout recorded history. The physical examination findings associated with such disorders are often described as ‘paradoxical,’ suggesting the observations are difficult to reconcile with a historic understanding of the underlying physiology. We present an illustrative case of pericardial tamponade and discuss the associated physiology in a way that may help practicing clinicians unravel the paradoxes of pericardial diseases.

CASE PRESENTATION

A man in his mid 50s came to the emergency room with shortness of breath and chest pain which had developed over about a day’s time. He said it felt nearly identical to

the pulmonary embolism he had experienced 2 weeks prior following a knee replacement surgery for arthritis. His medical history was otherwise significant only for Sjogren’s syndrome and hyperlipidaemia. The medications he was taking included warfarin, aspirin, plaquenil, salsalate and niacin.

INVESTIGATIONS

The patient was afebrile with a heart rate of 80 beats per min and a blood pressure of 90/60 mm Hg with a normal saturation on ambient air. There were crackles at the lung bases bilaterally, and pitting oedema of the left leg. Laying the patient flat to examine him worsened his chest pain. Heart sounds were distant. Laboratory values included an international normalised ratio of 6. The chest radiograph revealed a marked increase in heart size compared with

Figure 1 (A) Chest radiograph immediately prior to surgery. (B) Chest radiograph 2 weeks postop showing enlarged cardiac silhouette with clear lung fields. (C) Baseline ECG. (D) ECG 2 weeks postop demonstrating low voltage.

Figure 2 CT scan demonstrating a large pericardial effusion.

the preoperative image (figure 1 A,B). The EKG showed a significant reduction in voltage compared with the preoperative EKG done 2 weeks prior (figure 1 C,D), and a CT scan demonstrated a large pericardial effusion (figure 2). Echocardiogram re-demonstrated the effusion and allowed observation of diastolic collapse of the right ventricle.

OUTCOME AND FOLLOW-UP

Our patient underwent pericardiocentesis with an initial pericardial pressure measured at 30 mm Hg. After drainage of 2 litres of dark bloody fluid, the pericardial pressure fell below 3 mm Hg which resulted in immediate improvement in systemic blood pressure. The patient died several years later due to unrelated circumstances.

DISCUSSION

The patient was diagnosed with cardiac tamponade. This occurs when rising pericardial pressures limit filling of the heart, leading to impaired cardiac output.¹ The most common causes include idiopathic or viral pericarditis, neoplasm, acute myocardial infarction and iatrogenic myocardial or coronary artery perforation.²

Imaging studies in cardiac tamponade play an important role in the assessment and in directing therapeutic interventions. Chest radiographs typically show cardiomegaly with lung fields that are oligemic in contradistinction to congestive heart failure (figure 1B). The EKG typically shows diffuse low voltage and occasionally electrical alternans (figures 1D and 3). The alternans phenomenon is attributable to the heart twisting back and forth in a large effusion (videos 1 and 2). Echocardiography is the primary diagnostic modality for evaluated pericardial effusions. The characteristic echocardiographic feature of tamponade is right heart chamber diastolic compression.³

The exaggerated respiratory variations in right and left-sided doppler transvalvular inflow velocities (the so called Doppler flow-velocity paradoxus) (figure 4) are also important indicators of a haemodynamically significant effusion, correlate with pulsus paradoxus, and may precede chamber collapse.

Video 1 Apical four-chamber view and rhythm strip demonstrating electrical alternans.10.1136/bcr.09.2011.4792v1

Video 2 Parasternal short-axis view and rhythm strip demonstrating electrical alternans.10.1136/bcr.09.2011.4792v2

Our patient's effusion was found to be a haemopericardium. In 1761, Morgagni described several causes of haemopericardium and is credited with the first scientific description of traumatic pericardial tamponade.⁴ In the absence of trauma, bloody effusions should raise concern for malignancy, aortic dissection, myocardial infarction or postmyocardial (Dressler's) syndrome. Anticoagulants may convert serous or sero-sanguineous effusions into frankly bloody effusions.² A review of several studies estimated the incidence of pericardial bleeding for patients receiving anticoagulation therapy in the non-trauma setting at well under 1%.⁵ Simultaneous presentation of pulmonary embolism and hemopericardium has been previously described in the presence of an underlying carcinoma,⁶ which our patient did not have. We suspect, rather, that our patient's diagnosis of Sjogren's syndrome represented an autoimmune process including an unappreciated inflammatory serositis unmasked by anticoagulation.

The pericardium consists of an outer fibrosa and an inner serosa. The serosa is a complete closed sac comprised of a thin, elastic inner layer of mesothelial cells intimately applied to the surface of the heart where it constitutes the visceral pericardium or epicardium, with some extensions

Figure 3 Electrical alternans. Beat-to-beat oscillation of QRS complex is attributable to motion of the heart within a large pericardial effusion.

Figure 4 Trans-thoracic echocardiogram with doppler view of flow across the mitral valve. Note the large pericardial effusion and the variation in flow with respiration representing the 'doppler flow-velocity paradoxus.'

to enclose portions of the juxtacardiac great vessels. It reflects back on itself to line the inner aspect of the outer fibrosa together with which it forms the thick, stiff parietal pericardium. The pericardial space normally contains a thin 15–20 ml layer of fluid rich in surface active phospholipids that reduces friction between opposing surfaces. The response to gradual stretch differs from the response to acute stretch such that a sudden increase of as little as 100–200 ml can cause significant elevation of pericardial pressure, whereas an effusion developing slowly over

weeks or months may reach a volume of several litres with only a modest increase in pericardial pressure.

Richard Lower is credited with the first description of pericardial tamponade physiology in 1699: 'It sometimes happens that a profuse effusion oppresses and inundates the heart, the pulse becomes exceedingly small... until finally it becomes utterly suppressed by the great inundation of fluid whence succeed syncope and death itself.'⁷ One can begin to understand the physiology of tamponade through a comparison to constrictive physiology. Both of

Figure 5 Conceptualised pressure-volume curves of tamponade and constrictive pericarditis. Note that the Y axis represents volume in tamponade versus pressure in constrictive pericarditis. The X axis in both represents the period of diastolic filling.

these disorders present problems of cardiac chamber filling during diastole. Filling in tamponade is limited throughout diastole by the abnormal extracardiac pressure within the pericardial space, whereas filling in constriction is limited in late diastole by the volume determined by the non-distensible pericardium. Cardiac filling in these two conditions is conceptualized in figure 5. In tamponade, there is collapse of right-sided cardiac chambers when pressure within those chambers falls below the pericardial pressure. As pressure rises with blood return to the heart in diastole, inflow does not begin until the filling pressure exceeds the abnormally elevated pericardial pressure (figure 5). In constrictive pericarditis, however, early diastolic filling is brisk, but in later diastole when the chamber is filled to the volume at which the stiffened pericardium limits further distention, pressure rises precipitously without further increases in volume (figure 5). A pericardial ‘knock’ can sometimes be heard at the moment when the pericardium limits filling.

We posit that another distinction between tamponade and constrictive pericarditis can be made in terms of afterload. Afterload is a measure of the force a chamber of the heart must generate to eject the blood volume it contains. While arterial pressure resists ejection, thus adding to afterload, a cardiac compressive force assists in ventricular ejection, thereby reducing afterload.⁸ Consequently, the increased pressure in a pericardial effusion theoretically acts in part as a compensatory response to support a failing ventricle, whereas constrictive pericarditis does not affect afterload in this way. The lower right-sided pressures make it likely that right ventricular failure would be particularly influenced by this effect. Supporting this argument, a retrospective study of patients with right heart failure due to pulmonary arterial hypertension who underwent pericardial decompression reported a high rate of decompensation following these procedures.⁹

In healthy individuals, systolic blood pressure declines slightly with inspiration. Exaggeration of this phenomenon is termed pulsus paradoxus and defined as an inspiratory drop in systolic pressure of greater than 10 mm Hg. Although it can be observed in a number of conditions, it is classically associated with cardiac tamponade. The term ‘pulsus paradoxus’ was coined by Kussmaul in 1873 when he described a patient in whom the effect was so severe that the peripheral pulse disappeared during inspiration. The ‘paradox’ was that despite no pulse, the heartbeat

could still be heard.¹⁰ Kussmaul also described a sign that is named for him and can help differentiate between tamponade and constrictive pericarditis by physical examination. Kussmaul’s sign describes a paradoxical inspiratory increase in jugular venous distension. While occasionally observed in constrictive pericarditis, it never occurs in patients with pure pericardial tamponade. This again reflects the fact that the former is a diastolic problem of pressure, whereas the latter is a problem of volume. During inspiration, intrathoracic pressure becomes negative and venous return the right heart is increased. In tamponade, the negative intrathoracic pressures are transmitted to the right atria, and in turn the jugular venous system with a resultant normal decrease with inspiration. Conversely, the ‘paradoxical’ rise in jugular distension observed in constriction results from the inability of the right heart to accommodate to the increased blood volume presented to it during inspiration.

Learning points

- Tamponade can be conceptualised as a problem of diastolic filling limited by pressure, whereas constriction can be conceptualised as a problem of diastolic filling limited by volume.

Competing interests None.

Patient consent Not obtained.

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