Case Report

Acute Right Ventricular Dysfunction Complicating Prolonged Cardiac Tamponade

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Abstract:

We report a case of transient right ventricular dysfunction associated with prolonged cardiac tamponade, an unusual complication of uncertain etiology. We believe that in this case dynamic coronary flow restriction resulted in ischemic injury and stunning of the right ventricle. Other possible causes are briefly reviewed. Right ventricular failure can easily emerge undetected during tamponade since both disorders exhibit very similar physical findings. This complication should be considered when patients with pericardial effusion and tamponade deteriorate unexpectedly or fail to respond as anticipated following pericardial drainage. Our patient's clinical course identifies a potential adverse event associated with delayed diagnosis and treatment of cardiac tamponade which should encourage prompt pericardial drainage at the earliest clinical sign of cardiac compression.

Keywords: Tamponade, stunning, right ventricular failure

Introduction:

In resource limited healthcare systems, especially those with high burdens of HIV and tuberculosis, pericardial disease and tamponade are common and potentially complicated management problems[1]. Patients with these disorders often present to outlying clinics or hospitals where diagnostic modalities may be unavailable. By the time a definitive diagnosis is made, clinical or subclinical cardiac tamponade may have been present for days to weeks. Even in referral centers the lack of appropriate equipment for pericardial drainage may further delay return of cardiac hemodynamics to normal. A little recognized and seldom reported complication of prolonged cardiac tamponade is acute right ventricular failure. We herein report such a case.

Case Report:

A 58-year old Tanzanian male laborer presented to our referral hospital with an 8 week history of progressive prostration, shortness of breath, chest tightness, and lower limb swelling. He also reported a non-productive cough, intermittent low grade fever and night sweats. Prior to admission he had been treated at an outlying village clinic with diuretics, antibiotics and unspecified over-the-counter medications without improvement. Physical examination revealed an alert male in moderate distress that was dyspneic at rest. Temperature was 36.7 degrees Celsius, pulse 110 beats per minute, respirations 22 cycles per minute and blood pressure 110/85 millimeters of mercury. The neck veins were engorged and there was pulsus paradoxus of 15 millimeters of mercury. The cardiac apex was indistinct and heart tones were distant. The liver was palpable 3 centimeters below the right costal margin and was tender. The lower extremities were edematous. A rapid HIV antibody test was negative. An electrocardiogram showed generalized low voltage and his chest x-ray revealed a markedly enlarged, globular cardiac Asilhouette. Cardiac ultrasound performed using a hand-held device showed a large anterior and posterior

pericardial effusion. Fibrinous strands were present in the pericardial fluid. There were signs of tamponade including low ventricular volumes and diastolic collapse of the right ventricle and right atrium (Fig. 1).

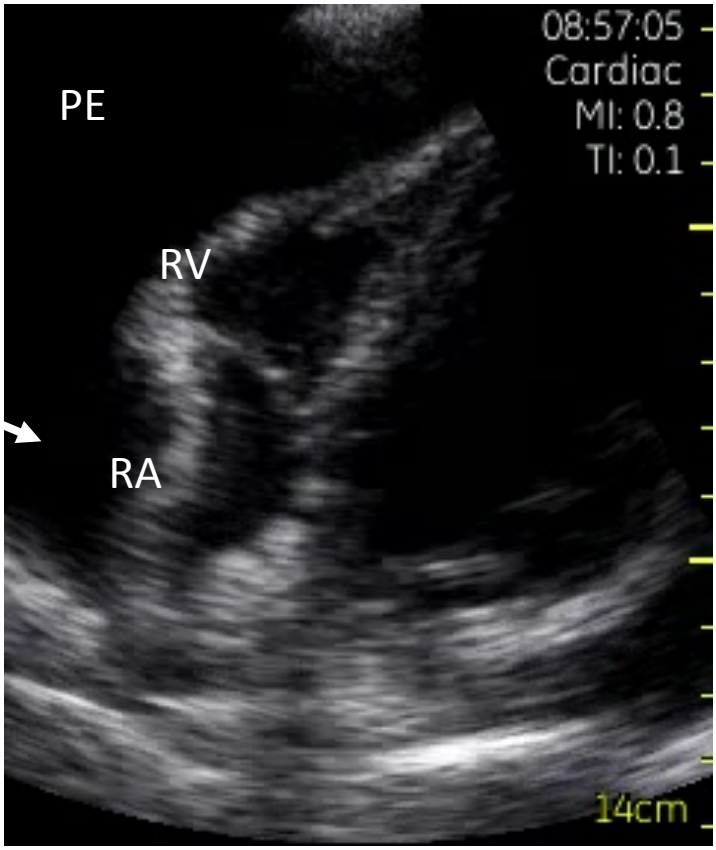

Figure 1.

Urgent pericardiocentesis was performed using a 16 gauge peripheral venous catheter. Approximately 120 mls of sanguineous fluid were removed and the patient reported subjective improvement and appeared more comfortable. Thereafter he was observed and managed conservatively with ibuprofen while diagnostic studies were obtained. By the 15th hospital day the patient's condition had again deteriorated and clinical findings similar to those at admission were observed. An echocardiogram was repeated and showed a

large pericardial effusion. In contrast to the initial study however the right ventricle was markedly dilated and hypocontractile, while left ventricular function appeared normal. The patient underwent urgent surgical drainage of approximately 1000cc of sanguineous fluid and creation of a pericardial window. Hypotension persisted post-operatively but responded well to administration of normal saline and low doses of dobutamine over the ensuing 24 hours. Thereafter he improved rapidly. An echocardiogram performed immediately after surgery showed pericardial thickening, a small residual apical effusion, and normal left ventricular function. The right ventricle remained dilated and hypocontractile, unchanged from the preoperative study (Fig. 2).

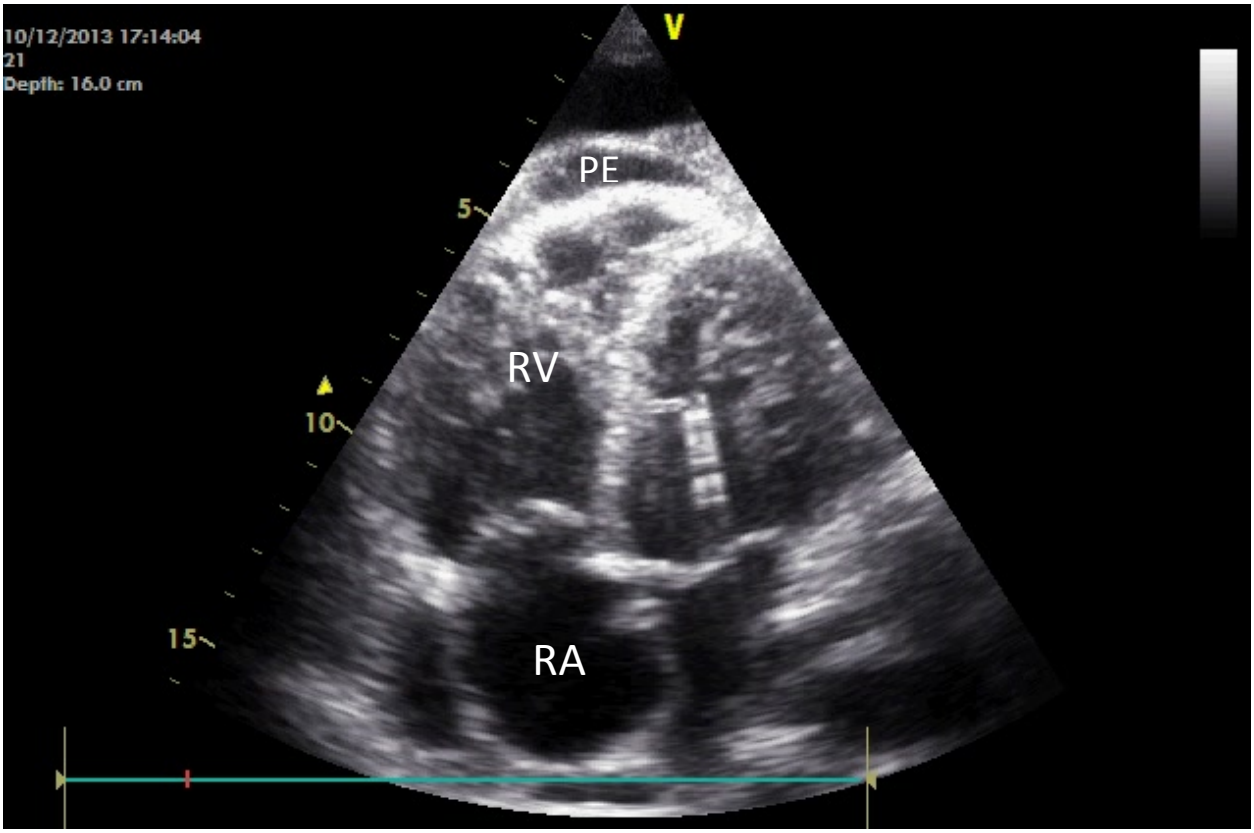

Figure 2

Analysis of pericardial fluid and histologic examination of the pericardial specimen obtained at surgery were consistent with a non-specific inflammatory process. The diagnosis at discharge was idiopathic pericarditis. He was managed with ibuprofen alone. At his 3 month follow-up visit he was

asymptomatic and reported himself returned to his usual state of health. An echocardiogram at that visit showed no residual effusion and return of right ventricular function to normal (Fig. 3). A repeat electrocardiogram was also normal.

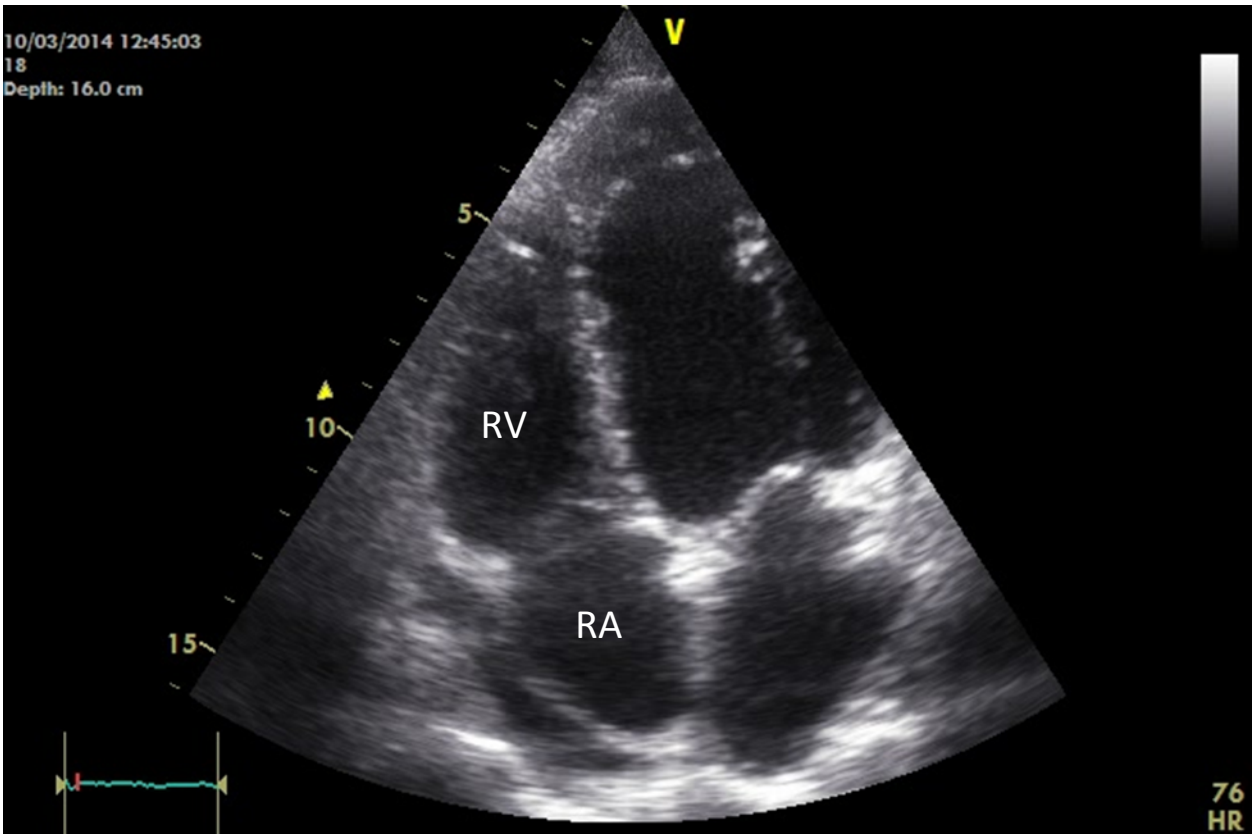

Figure 3

Discussion:

Over the past three decades there have been scattered reports of ventricular dysfunction associated with cardiac tamponade (2-7). In all but one of these cases dysfunction developed after pericardial drainage (2-5, 7). One case of biventricular dysfunction appeared prior to pericardiocentesis (6). With the exception of a single case of right ventricular failure which proved fatal (5), ventricular dysfunction was transient. In our patient isolated right ventricular dysfunction developed prior to definitive pericardial drainage, was likely the cause of his sub-acute deterioration, and resolved within several weeks of the procedure.

Various mechanisms by which ventricular injury might occur during tamponade have been proposed (7). These can be grouped into four general areas: 1. myocardial ischemia, 2. adverse hemodynamics of rapid cardiac refill after pericardial drainage, 3. an infectious process producing combined pericarditis and myocarditis and 4. Acute pulmonary embolism with associated right ventricular injury.

Myocardial ischemia figures prominently as a likely etiology. It has been shown that blood flow in an unobstructed epicardial coronary artery decreases and coronary resistance increases as intra-pericardial pressure rises (8). When pericardial pressures are high enough to produce diastolic right ventricular collapse it may be argued that additional flow limiting compressive forces appear in diastole, further decreasing the time available for effective perfusion. Because the left ventricle is thicker and less compliant than the RV, pressure gradients between the pericardium and the left ventricular chamber may be preserved during diastole both preventing LV diastolic collapse and protecting diastolic coronary filling (10). Tamponade persisting for longer periods is associated with a compensatory rise in blood volume causing atrial and ventricular filling pressures to increase (11). As right atrial pressure raises so also does pressure in the great cardiac vein. Significant increases in great

cardiac vein pressures may also negatively affect coronary flow (12). Finally, tamponade - induced hypotension with low aortic diastolic pressures directly reduces myocardial blood flow. Individually or in aggregate these factors may threaten effective myocardial perfusion.

Vandyke et al (2) proposed that sudden reduction of intrapericardial pressures during pericardial drainage allows unimpeded return of increased volumes of venous blood to the heart, filling the ventricles to high diastolic volumes and pressures and producing abrupt myocycte stretch which, if of sufficient magnitude, may produce ventricular dysfunction. The thin-walled right ventricle may be particularly susceptible to injury by this mechanism. Pericardial drainage also reduces the stimulus for high sympathetic outflow reducing heart rate and inotropism representing an ancillary mechanism by which underlying ventricular dysfunction might become manifest (4).

Elevated intrapericardial pressures induce significant venous stasis during tamponade. Prolonged stasis in the venous system invites thrombosis and in turn pulmonary embolization with acute right ventricular dysfunction. Especially in the case of isolated right ventricular dysfunction complicating tamponade, such a mechanism may be operative.

Numerous studies have shown that pericardial inflammation often involves adjacent myocardium producing myocarditis. Although the associated myocarditis is often restricted to epicardial layers, the possibility of a full-blown myocarditis with associated contractile dysfunction should be considered in circumstances of ventricular failure complicating pericardial disease.

In our case acute right ventricular dysfunction was identified prior to definitive pericardial drainage. The echocardiogram at admission demonstrated a large pericardial effusion, a small right ventricle and right atrium with features of right atrial and right ventricular diastolic collapse (Fig. 1). Right ventricular diastolic collapse in tamponade is seen only in the setting of normal ventricular free wall thickness and compliance arguing that in this case there was no

antecedent right ventricular hypertrophy or dysfunction. Our patient had no history of epicardial coronary disease nor did he have a clinical syndrome suggestive of acute myocardial infarction during hospitalization. There was neither electrocardiographic nor echocardiographic evidence of infarction of the inferior left ventricular segment arguing further against RV infarction due to epicardial coronary obstruction. Return of the right ventricle to completely normal function would also be inconsistent with acute right ventricular infarction. The finding that right heart dilatation and dysfunction appeared after admission but prior to surgical pericardial drainage indicates that acute right ventricular injury occurred during the period of increased intrapericardial pressures. This observation would suggest that neither rapid chamber refill nor sympathetic overdrive and masking of underlying ventricular dysfunction were operative in our patient. We could not exclude pulmonary thromboembolism as a cause of this patient's acute right ventricular failure. However, the absence of a sentinel clinical event and his rapid clinical improvement following pericardial drainage without thrombolytic or anticoagulant therapy makes this possibility seem unlikely in our view. An acute myocarditis in association with acute pericarditis as a cause for this patient's clinical syndrome remains possible however the isolated involvement of the right ventricle would be an unusual presentation of myocarditis. It seems most likely that reversible right ventricular dysfunction in this case was the result of dynamic coronary flow restriction, ischemia and stunning due to tamponade. Without question, delays in diagnosis and definitive pericardial drainage common in resource limited healthcare systems contributed to this complication.

Conclusion:

In resource-limited areas with high burdens of HIV, tuberculosis and other infectious disorders pericardial disease complicated by cardiac tamponade is common and significant treatment delays may occur. We report a case of transient right ventricular dysfunction associated with prolonged cardiac

tamponade and discuss possible etiologic mechanisms. Isolated right or bi-ventricular heart failure can easily emerge undetected under the cloak of tamponade since findings on physical examination of both disorders may be very similar. This complication should be considered when patients with pericardial effusion and tamponade deteriorate unexpectedly or fail to respond as anticipated following pericardial drainage. Cardiac ultrasound is the diagnostic modality most likely to render a rapid and accurate diagnosis in this circumstance. In most reported cases improvement of ventricular function after pericardiocentesis has been observed. Our patient's clinical course identifies an adverse event associated with delayed diagnosis and treatment of cardiac tamponade and should encourage prompt pericardial drainage at the earliest clinical sign of cardiac compression.

Legends:

Figure 1: 2-D echocardiogram at admission showing a large pericardial effusion (PE) with right atrial (RA) and right ventricular (RV) diastolic collapse (DC).

Figure 2: 2-D echocardiogram obtained post pericardial drainage showing marked right ventricular (RV) enlargement, large right atrium (RA) and small apical effusion (PE). RV dimensions are unchanged from those observed immediately before pericardial drainage.

Figure 3: 2-D echocardiogram at 3 month follow-up which shows return of right ventricular (RV) and right atrial (RA) dimensions to normal.

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