CASE REPORT

Over-diuresis or cardiac tamponade? An unusual case of acute kidney injury and early closure

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An 84-year-old man with hypertension and a history of deep venous thrombosis (on warfarin) was admitted with shortness of breath presumed to be due to congestive heart failure. Echocardiogram performed the following day showed a low-normal ejection fraction with signs of elevated right-sided pressures but was otherwise normal. He improved with diuretic therapy but after a few days was found to be hypotensive with a concomitant rise in creatinine with decreased urine output. This was felt to be secondary to over-diuresis but he did not respond to small boluses of intravenous fluids as his kidney function continued to worsen and hypotension persisted. He was transferred to the intermediate care unit where a rapid, bedside ultrasound revealed a new, moderate-sized pericardial effusion with tamponade physiology. Pericardiocentesis, with removal of 750 cc of frank blood, led to dramatic improvement in blood pressure, kidney function, and urine output. Here, we demonstrate the utility of point-of-care ultrasound in a community hospital setting where urgent echocardiogram is not routinely available. We also report acute kidney injury due to pericardial tamponade reversed with therapeutic pericardiocentesis.

Keywords: cardiac tamponade; acute kidney injury; bedside ultrasound

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An 84-year-old man with a history of hypertension, hyperlipidemia, esophageal cancer (in remission), and deep venous thrombosis on warfarin presented to the emergency department with shortness of breath. Transthoracic echocardiogram the day following admission revealed a low-normal ejection fraction with left ventricular hypertrophy and signs of elevated right-sided pressures but was otherwise normal. The clinical diagnosis was congestive diastolic heart failure. His initial laboratory studies included a normal creatinine and a therapeutic International normalized ratio (INR) of 2.6. The patient was started on intravenous diuretic therapy with improvement in symptoms after several days of negative fluid balance.

On the fifth day of hospitalization, the patient was found to be hypotensive. Vital signs revealed an oral temperature of 97.9°F, a pulse of 99 beats per minute, a blood pressure of 77/55 mm Hg, respiratory rate of 24 with oxygen saturation of 95% on 2 liters of oxygen by nasal cannula. He was found to be lethargic, having complained of generalized weakness earlier in the day. Jugular venous distension was noted on neck examination with clear lungs and distant heart sounds. Other studies revealed an INR of 3.5 and fractional excretion of sodium of 0.3%. Renal ultrasound was normal and the patient was not on any nephro-toxic medications. The assessment was that of over-diuresis with pre-renal acute kidney injury but he did not respond to small boluses of intravenous fluids as his kidney function continued to deteriorate and hypotension persisted.

Due to worsening decompensation in the evening hours, he was then moved to the intermediate care unit for further management where point-of-care ultrasonography by the internal medicine resident was performed revealing a moderate pericardial effusion with right atrial collapse consistent with tamponade physiology (Fig. 1, Video clip 1 & 2). Pulsus paradoxus was then found with a drop in systolic blood pressure of 20 mmHg with inspiration. Urgent pericardiocentesis was performed with removal of 750 cc of frank blood. Post-procedure, the patient’s urine output and kidney function normalized with creatinine level of 0.8 mg/dl at discharge after a peak of 3.6 mg/dl just prior to pericardial drainage (Fig. 2). Pericardial fluid was negative for infection and malignancy. Given his history of esophageal carcinoma, a Positron emission tomography-computed tomography (PET/CT) was done which was negative for any evidence of cancer. Warfarin was discontinued and a repeat...
Echocardiogram 4 weeks after the procedure did not show any pericardial effusion.

Discussion

Here we present a patient with cardiac tamponade leading to acute kidney injury due to spontaneous hemopericardium in the setting of supratherapeutic anticoagulation. Factors which influence development of cardiac tamponade are the rate of accumulation of pericardial fluid and pericardial membrane stiffness. Rapid accumulation of fluid causes maximum pericardial stretch before compensatory mechanism can be activated thus leading to increased pericardial pressures and tamponade at much lower volumes. In slower accumulation, the compensatory mechanisms have more time to be engaged; as such, much higher volumes are required to cause the tamponade (1).

In our case, hemopericardium was suspected upon discovery of his tamponade physiology as there was no other explanation for such a rapid accumulation of pericardial fluid.

Typical symptoms of cardiac tamponade are weakness and dyspnea related to decreased cardiac output and hypoperfusion (1). Our patient had decreased urine output and pre-renal acute kidney injury. On physical examination, patients can have tachycardia, jugular venous distension, and muffled heart sounds (2). Pulsus paradoxus can also be found, defined as a drop in systolic blood pressure of more than 10 mm Hg during inspiration (3). In addition, phasic respiratory changes in pulse-oximetry waveform amplitude can be helpful in identification of cardiac tamponade; this was present in our patient as well (4). Chest radiography with an enlarged cardiac silhouette and EKG with low voltage (or electrical alternans) are useful diagnostic tools that were subsequently found in our patient but may not be seen in all patients (Figs. 3 and 4) (5). Echocardiogram remains the mainstay in diagnosing pericardial effusions and tamponade. Right atrial inversion during systole and, ultimately, right ventricle collapse during early diastole are typical findings (6).

We reviewed cases of cardiac tamponade secondary to an elevated INR (7–10) and we also reviewed case reports of cardiac tamponade with acute kidney injury (11). To our knowledge not many cases of hemopericardium due to a high INR leading to acute kidney injury have been reported. Our case is unique for a number of reasons. First, the patient’s initial echocardiogram just a few days earlier did not show any pericardial effusion demonstrating that his significant pericardial accumulation had developed acutely during his hospital stay. Second, there was early diagnostic closure as the patient was thought to be hypotensive and to have pre-renal acute kidney injury related to over-diuresis; pericardial tamponade was not considered initially because he had no pericardial effusion just a few days prior. Finally, in a community hospital setting that lacks 24-h intensivist staffing, critical care, or cardiology fellows, and where echocardiography is not readily available in the evening/overnight hours, the use of bedside ultrasound was crucial in the early diagnosis and timely treatment of our patient.

This case also highlights the heart–kidney relationship in an acute setting. The patient went into acute oliguric pre-renal failure which resolved completely after pericardiocentesis. Typically cardio-renal syndrome (CRS) type 1 is described as acute heart dysfunction leading to acute renal failure. This is commonly secondary to acute decompensated heart failure due to cardiogenic shock. Cardiac tamponade is an unusual form of decompensated heart failure and, thus when it leads to acute renal failure due to hypoperfusion, it can be described as an atypical or unusual form of type 1 CRS (12, 13).

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**Fig. 1.** Bedside point-of-care ultrasound. Subxiphoid view showing pericardial effusion with right atrial collapse. PE = pericardial effusion, RA = right atrium, RV = right ventricle, LV = left ventricle.

**Fig. 2.** Serum creatinine (in mg/dl) and urine output (in liters), pre and post pericardiocentesis (Day 5).
Fig. 3. 12 Lead EKG with low voltage.

Fig. 4. (a) CXR at presentation and (b) CXR on Day 5 at time of tamponade diagnosis demonstrating acute expansion of the cardiac silhouette.

Video Clip 1. Sub-Xiphoid view video.

Video Clip 2. Apical 4 chamber view video.
Conclusion
In patients on therapeutic anticoagulation with evidence of hypoperfusion or acute kidney injury, cardiac tamponade should always be kept in mind as a potential cause even if the absence of any such pericardial effusion was recently proven. Bedside, point-of-care ultrasound is a very useful method for early diagnosis and management of cardiac tamponade particularly in a community hospital setting.

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