Case Report

# Cardiac Tamponade Associated with Human Immunodeficiency Virus-Associated Immune Complex Kidney Disease

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## Abstract

Cardiac tamponade is a life-threatening emergency, characterized by rapid accumulation of pericardial fluid. There are multiple risk factors for cardiac tamponade, nephrotic syndrome is an uncommon one, especially in adults. Herein, we are reporting a 35-year-old African American woman with membranoproliferative glomerulonephritis secondary to human immunodeficiency virus-associated immune complex kidney disease (HIVICK), who presented with cardiac tamponade. The patient had pericardiocentesis and was discharged, with outpatient follow-up with cardiology, nephrology, and infectious disease. To the best of our knowledge, this is the first report of HIVICK nephrotic syndrome associated with cardiac tamponade.

**Keywords:** Cardiac tamponade, case report, human immunodeficiency virus-associated immune complex kidney disease, human immunodeficiency virus, membranoproliferative glomerulonephritis

## Introduction

Cardiac tamponade is a life-threatening emergency, characterized by rapid accumulation of pericardial fluid either from a transudate, an exudate, or blood, leading to cardiac chambers compression, with a decreased in venous return, and ventricular filling ultimately leading to decreased cardiac output.[1] There are different etiologies that can lead to cardiac tamponade including trauma, infections, autoimmune diseases, neoplasms, uremia, and other inflammatory diseases.[1] To the best of our knowledge, there are only a few cases in the literature describing cardiac tamponade secondary to nephrotic syndrome,[2] they are mainly in children, and none have been reported in an adult human immunodeficiency virus (HIV)-infected patient with HIV-associated immune complex kidney disease (HIVICK).

## Case Report

A 35-year-old African American female with medical history of HIV, off antiretroviral therapy (ART) for over a year, chronic kidney disease Stage 3b, membranoproliferative glomerulonephritis secondary to HIVICK that was recently diagnosed by biopsy, and pulmonary hypertension presented to the emergency department complaining of burning sensation in her feet, bilateral swelling of her legs along with difficulty walking and shortness of breath with exertion. She denied any cough, chest pain, palpitations, fever, or sick contacts. Vital signs on admission showed a temperature of 36°C, heart rate of 95/min, blood pressure of 150/100 mmHg, respiratory rate of 18/min, and oxygen saturation of 100% on 2 L/min of oxygen through nasal cannula. Physical examination was significant for engorged neck veins, elevated jugular venous pressure, distant heart sounds, decreased air entry bilaterally with basal crackles, and 3+ lower extremity pitting edema up to the knees. Laboratory workup showed bicarbonate level of 17 mmol/L (20-31 mmol/L), blood urea nitrogen of 26 mg/dL (6–24 mg/dL), serum creatinine 1.7 mg/dL (0.5–1 mg/dL), and estimated glomerular filtration rate of 34

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mL/minute/1.73m 2. Cardiac markers such as brain natriuretic peptide was 507 pg/mL (0–100 pg/mL) and initial troponin I 0.485 ng/mL (0–0.45 ng/mL). Inflammatory markers such as D-dimer were elevated, 2300 ng/mL (0–500 ng/mL), but C-reactive protein and procalcitonin were within normal limits. CD4 count was 173/uL (359–1519/uL) with viral load of 30,780 copies/mL. She had negative Hepatitis B surface antigen, and negative Hepatitis C antibody; her blood cultures were negative. Urine analysis showed nephrotic range proteinuria (+3). Chest X-ray (CXR) showed small bilateral pleural effusions with enlarged cardiac silhouette with "3 bottle" configuration suggestive of pericardial effusion [Figure 1]. Echocardiogram showed significant pericardial effusion with signs of tamponade [Figure 2], right ventricular systolic pressure was elevated at 64 mmHg (normal range: 15–25 mmHg), for which she was transferred to cardiac critical care unit for close monitoring. The next day, she underwent an emergent pericardiocentesis with pericardial window, 800 cc of serosanguinous fluid was drained from the pericardium. A chest tube was also inserted to drain the right pleural effusion with an output of 1000 cc. The pericardial fluid analysis revealed transudative nature, cultures were negative and acid-fast bacillus test was negative too. Cytology showed no evidence of malignancy and the pericardial tissue pathology showed mild focal inflammation with no evidence of malignancy. Afterward, the patient was monitored in the CCU for a few days, there were no complications of the procedure, repeat CXR showed resolution of the pericardial effusion and improvement in the left pleural effusion [Figure 3]. The patient was discharged, with outpatient follow-up with cardiology, nephrology, and infectious disease departments.

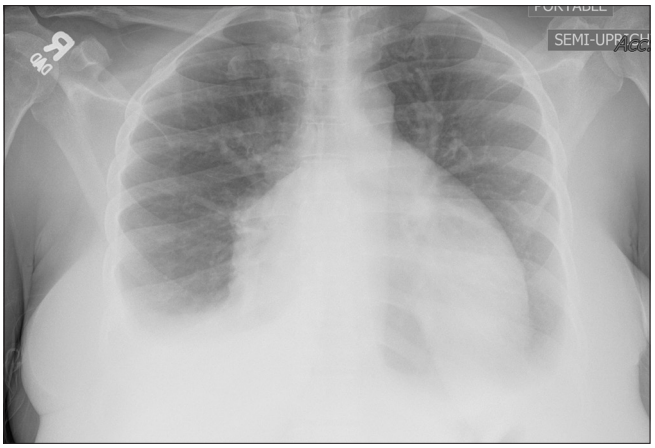

**Figure 1:** Chest X-ray showed small bilateral pleural effusions with enlarged cardiac silhouette with "3 bottle" configuration suggestive of pericardial effusion

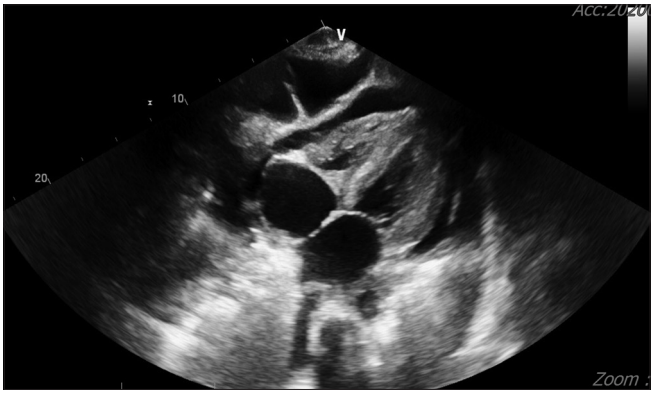

**Figure 2:** Echocardiogram showed significant pericardial effusion with signs of tamponade

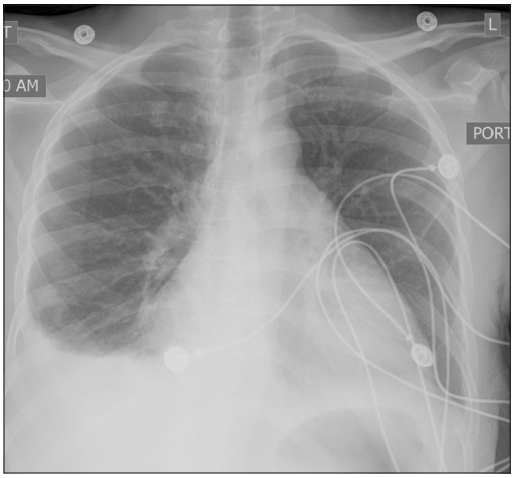

**Figure 3:** Repeat chest X-ray showed resolution of the pericardial effusion and improvement in the left pleural effusion after pericardiocentesis and chest tube placement

## Discussion

Cardiac tamponade is a life-threatening condition characterized by the rapid accumulation of pericardial fluid where the intra-pericardial pressure exceeds the pressure within the cardiac chambers, resulting in inadequate cardiac filling, decreased cardiac output, and ultimately, cardiogenic shock and death if not rapidly identified and appropriately treated.[3] The incidence of cardiac tamponade in the US is estimated to be about 2-5 cases per 10,000.[4]

Renal disease with nephrotic syndrome is considered a rare cause of pericardial effusion and cardiac tamponade with unclear incidence in the literature.[5] Göbel *et al*., in their study, suggested that hydropericardium is rare in nephrotic patients and that an inflammatory or other secondary cause should be considered when pericardial effusion complicates nephrotic syndrome.[5]

HIV-associated immune complex (IC) kidney disease is characterized by the presence of glomerular IC deposition on a renal biopsy of HIV-positive patients.[6] It is more predominant in African American patients with advanced HIV disease.[7] Risk factors for the development of HIVICK included HIV RNA levels >400 copies/ml, diabetes, hypertension, and hepatitis C co-infection.[7] Patients with HIVICK may present with nephrotic syndrome complicated by pleural effusion and very rarely pericardial effusion.[8] There are only a few cases reported in literature of cardiac tamponade associated with

nephrotic syndrome.[2] Our patient is an African American woman with a history of poorly controlled HIV, viral load was around 30K copies/mL and CD4 count of 173 at the time of diagnosis.

The diagnosis of cardiac tamponade can be suspected by history and physical exam findings. Electrocardiogram may be helpful, especially if it shows low voltages or electrical alternans, due to the swinging of the heart within the pericardium that is filled with fluid.[9] Echocardiography remains the gold standard in the diagnosis of pericardial effusion and pericardial tamponade.[9]

Because of the uncommonness of this presentation, there are no clear guidelines for the management and treatment. Initially, where the patients are hemodynamically compromised, the main goal of the treatment will focus on decompression of the pericardium by the removal of pericardial fluid to help relieve the pressure surrounding the heart.[10]

The next step in the treatment after stabilizing the patient is preventing the fluid re-accumulation in the pericardial space by managing the nephrotic syndrome and controlling the HIV. Studies regarding the optimal treatment of patients with HIVICK are limited, and the clinical effect of ART in patients with HIVICK is unclear. Foy *et al*., described a direct relationship between the viral load and the findings on the renal biopsy.[7] The initiation of ART and subsequent suppression of HIV RNA, in addition to the general measures by controlling blood pressure and starting renin–angiotensin inhibitors, were associated with improvement of renal function in patients with HIVICK resulting in improvements in the GFR and proteinuria, and at the same time, it will prevent the fluid accumulation in the pericardium.[7]

## Conclusion

Cardiac tamponade has multiple causes, it is uncommon to have nephrotic syndrome as a cause of tamponade in adult population. In this case, we present an association of cardiac tamponade secondary to the nephrotic syndrome in a patient who has HIVICK on renal biopsy, highlighting the potential unique feature of HIVICK nephrotic syndrome. This case report hopefully can increase awareness among cardiologists and nephrologist of the possible association of cardiac tamponade in patients with HIVICK nephrotic syndrome.

### Statement of ethics

Patients have given the written informed consent to publish the cases including publication of images.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

### Research quality and ethics statement

The authors followed applicable EQUATOR Network ("http://www.equator-network.org/) guidelines, notably the CARE guideline, during the conduct of this report.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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