Journal of Community Hospital Internal Medicine Perspectives

Volume 14 | Issue 2 Article 9

2024

# Uremic pericarditis and cardiac tamponade resolving with intensive hemodialysis

Mayuri Patel
*Medicine, MedStar Union Memorial Hospital, Baltimore, MD*

Shiavax J. Rao
*Medicine, MedStar Union Memorial Hospital, Baltimore, MD*, shiavax.j.rao@medstar.net

Abhinandan R. Chittal
*Medicine, MedStar Union Memorial Hospital, Baltimore, MD*

Khalid Al-Talib
*Nephrology, MedStar Franklin Square Medical Center, Baltimore, MD*

Sriram Padmanabhan
*Cardiology, MedStar Franklin Square Medical Center, Baltimore, MD*



## Recommended Citation

Patel, Mayuri; Rao, Shiavax J.; Chittal, Abhinandan R.; Al-Talib, Khalid; and Padmanabhan, Sriram (2024) "Uremic pericarditis and cardiac tamponade resolving with intensive hemodialysis," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 14: Iss. 2, Article 9.
DOI: 10.55729/2000-9666.1307
Available at: https://scholarlycommons.gbmc.org/jchimp/vol14/iss2/9

CASE REPORT

# Uremic Pericarditis and Cardiac Tamponade Resolving With Intensive Hemodialysis

Mayuri Patel [a,b], Shiavax J. Rao [a,b,*], Abhinandan R. Chittal [a,b], Khalid Al-Talib [c], Sriram Padmanabhan [d,e]

[a] Medicine, MedStar Union Memorial Hospital, Baltimore, MD, USA
[b] Medicine, MedStar Franklin Square Medical Center, Baltimore, MD, USA
[c] Nephrology, MedStar Franklin Square Medical Center, Baltimore, MD, USA
[d] Cardiology, MedStar Franklin Square Medical Center, Baltimore, MD, USA
[e] MedStar Heart & Vascular Institute, Baltimore, MD, USA

**Abstract**

**We present an interesting and complex case of cardiac tamponade due to uremic pericarditis (UP), resolving with intensive hemodialysis (HD). HD should be considered as first line management for patients with UP and pericardial effusion. Intensification of HD should be considered based on clinical presentation and severity of presentation.**

*Keywords:* Pericardial effusion, Pericarditis, Hemodialysis, End-stage renal disease, Cardiac tamponade, Pericardial tamponade

## 1. Introduction

Pericardial fluid ranges from 15 to 50 ml of plasma ultrafiltrate. When inflamed there is an increase in production of fluid by the pericardium which accumulates. In the acute or subacute setting, accumulation of fluid which exceeds the elasticity of the pericardium causes the heart to compete with the pericardial fluid due to limited intrapericardial volume.[1,2] We present an interesting and complex case of cardiac tamponade due to uremic pericarditis (UP), resolving with intensive hemodialysis (HD).

## 2. Case presentation

A 60-year-old woman presented to the emergency room in the context of chest pain, shortness of breath and generalized weakness of one week duration, following a recent hospitalization for atrial fibrillation. She endorsed sharp chest pain radiating to her back, associated with shortness of breath, dizziness, and palpitations. She denied any such episodes in the past. On initial presentation, she was hypotensive (66/37 mm Hg), tachycardic (134 beats per minute) and she required 4 L of supplemental oxygen via nasal cannula to maintain adequate oxygen saturation. Physical examination was significant for muffled heart sounds, a III/VI systolic ejection murmur in right parasternal 2nd intercostal space and jugular venous distention.

Her past medical history was notable for hypertension, hyperlipidemia, type 1 diabetes mellitus, end-stage renal disease (ESRD) non-compliant with HD (status post two failed kidney transplants) and Marfan syndrome. Given the presenting history, physical examination findings and past medical history, differential considerations included acute coronary syndrome, acute pericarditis, cardiac tamponade, aortic dissection, pulmonary embolism, pulmonary infection, and acute decompensated heart failure/cardiomyopathy.

Complete blood count (CBC) was unremarkable except for stable anemia secondary to end-stage renal disease (ESRD). Serum chemistries were remarkable for elevated creatinine and blood urea nitrogen (BUN) at 6.5 mg/dL and 48 mg/dL, respectively (reference range of creatinine:

Received 12 July 2023; revised 7 November 2023; accepted 2 January 2024.
Available online 4 March 2024

* Corresponding author at: MedStar Union Memorial Hospital, Department of Medicine, 201 E University Pkwy, Baltimore, MD 21218, USA.
E-mail address: shiavax.j.rao@medstar.net (S.J. Rao).
(S.J. Rao).

https://doi.org/10.55729/2000-9666.1307

0.5–0.8 mg/dL and BUN: 9–23 mg/dL, respectively), hyperkalemia 5.7 mmol/L (reference range: 3.4–4.5 mmol/L) consistent with ESRD. Brain Natriuretic Peptide (BNP) was elevated at 704.7 pg/mL (reference range: 0–99 pg/mL) and troponin was normal at 24 ng/L (reference range: 0–34 ng/L). A 12-lead electrocardiogram (EKG) revealed atrial fibrillation with rapid ventricular response (125 beats per minute), and nonspecific ST and T wave abnormalities in the inferior and lateral leads (Fig. 1). A plain film radiograph of the chest demonstrated cardiomegaly and trace bilateral pleural effusions. Non-contrast computed tomography (CT) of the head was negative for any acute intracranial abnormality. CT angiography of the chest, abdomen and pelvis (with and without contrast) revealed a moderate pericardial effusion with subtle diffuse pericardial enhancement suspicious for pericarditis. A limited transthoracic echocardiogram (TTE) showed normal left ventricular systolic function with an ejection fraction 60–65 %, grade III left ventricular diastolic dysfunction, and a moderate to large circumferential pericardial effusion with inversion of right atrial and right ventricular walls, consistent with cardiac tamponade (Fig. 2).

Considering the patient's significant history of ESRD, she was diagnosed with uremic pericarditis with impending cardiac tamponade and was initiated on intensive daily sessions of HD via her left arm arterio-venous fistula. She received a total of four consecutive HD sessions, with removal of 1.5 L of fluid during each session. Her hospital course was further complicated by recurrent episodes of atrial fibrillation, initially managed with beta-blockers, but ultimately requiring amiodarone. Anticoagulation was held during the hospitalization due to the risk of hemorrhagic conversion of the pericardial effusion. A repeat TTE showed decrease in the size of the pericardial effusion. The patient was eventually weaned off supplemental oxygen and her blood pressure had improved to 117/57 mm Hg prior to discharge.

After being discharged, our patient followed up with cardiology and nephrology. A 1-month interval TTE performed during the outpatient cardiologist visit, showed normal left ventricular systolic

**Abbreviations**

| | |
|---|---|
| AKI | Acute Kidney Injury |
| BUN | Blood Urea Nitrogen |
| CBC | Complete Blood Count; CKD Chronic Kidney Disease |
| CT | Computed Tomography |
| EKG | Electrocardiogram |
| ESRD | End Stage Renal Disease |
| HD | Hemodialysis |
| RRT | Renal Replacement Therapy |
| TTE | Transthoracic Echocardiogram |

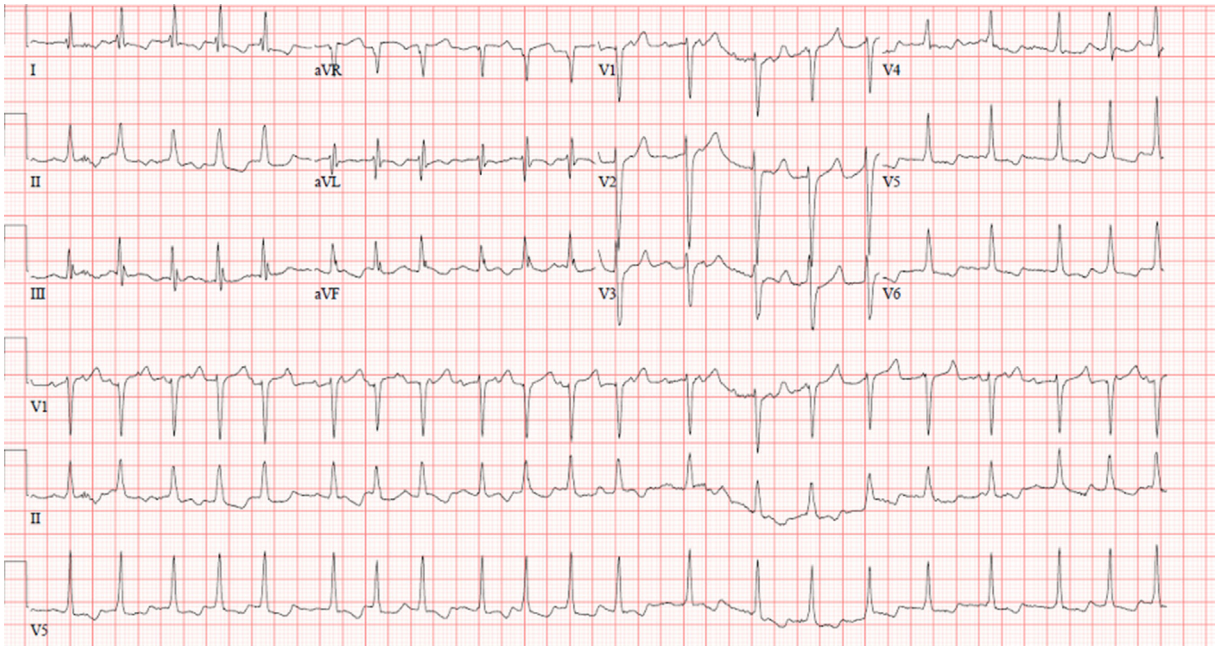


*Fig. 1. 12-lead electrocardiogram revealing atrial fibrillation with rapid ventricular response (125 beats per minute), and nonspecific ST and T wave abnormalities in the inferior and lateral leads.*

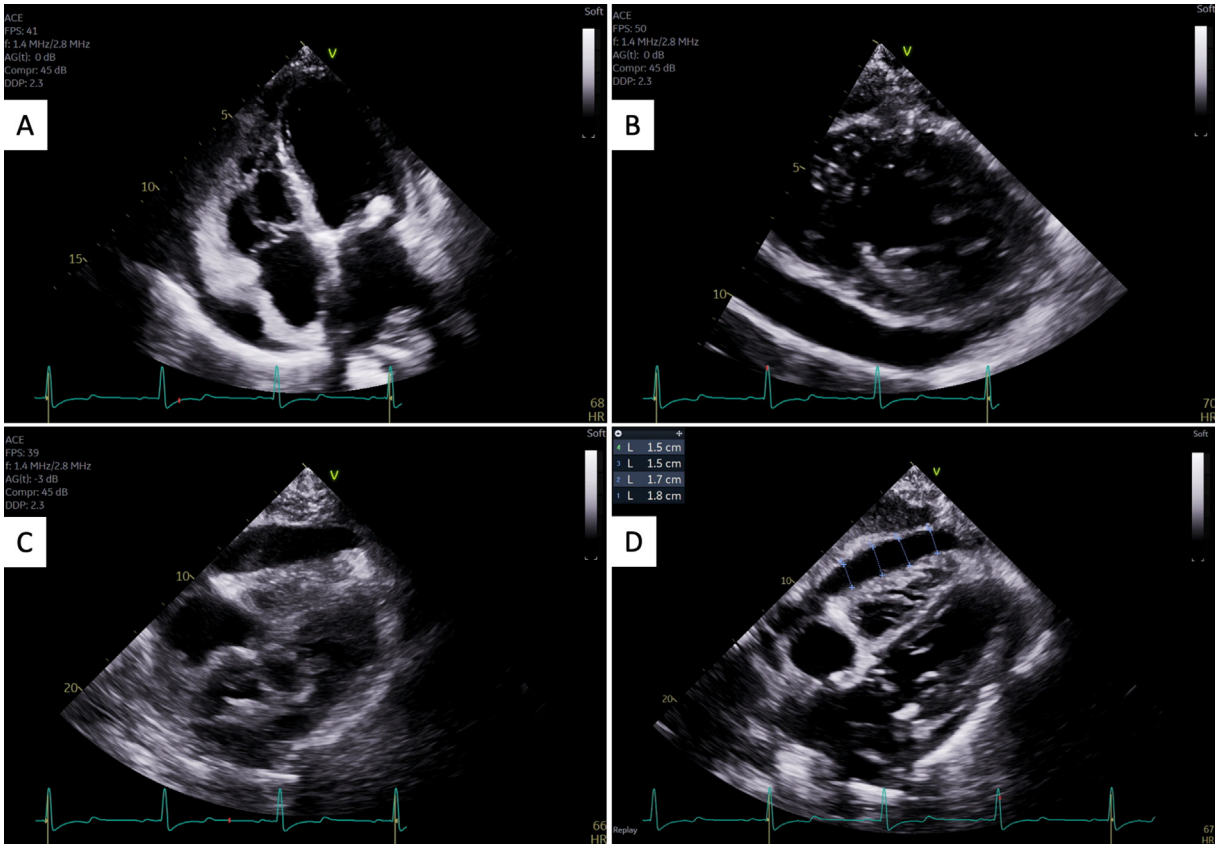


*Fig. 2. Transthoracic echocardiogram showing pericardial effusion with tamponade physiology. (A) 4-chamber view revealing large pericardial effusion with inversion of right atrial wall. (B) Parasternal short-axis view highlighting large pericardial effusion. (C,D) Subcostal view demonstrating large pericardial effusion with inversion of right ventricular wall.*

function with ejection fraction 55–60 %, grade III left ventricular diastolic dysfunction, mildly enlarged right ventricular size, and complete resolution of the previously noted pericardial effusion. Given her overall clinical stability, she was continued on amiodarone and anticoagulation for atrial fibrillation.

## 3. Discussion

The pericardium is a double-walled avascular membrane surrounding the heart, composed of two layers (parietal and visceral pericardium), containing about 50 mL of pericardial fluid. Pericarditis refers to inflammation of this sac, and the accumulation of excess pericardial fluid due to imbalance between its production and absorption is called effusion. Etiologies of pericarditis are wide-ranging, including infection, cardiac insufficiency, surgical intervention, autoimmune processes, metabolic causes, malignancy, and drugs.[1,2] Uremic and dialysis-associated pericarditis constitute the common pericardial diseases among patients with acute kidney injury (AKI), chronic kidney disease (CKD) and ESRD with the full spectrum ranging from pericarditis, to pericardial effusion and cardiac tamponade.[2]

UP generally occurs before or within eight weeks of initiation of renal replacement therapy (RRT), whereas dialysis-associated pericarditis may occur in a dialysis-compliant patient after eight weeks of the initiation of RRT.[1,3] Our patient was diagnosed with UP as she was non-adherent with outpatient HD. The reported prevalence of uremic pericarditis is 2–21 %.[4] The overall incidence of UP has been estimated to be 1.4–29 % per patient-year.[2] In most cases, UP is reported to be fibrous or fibrofibrinous pericarditis with occasional reports of sero-fibrinous and hemorrhagic variants.[2] A higher incidence of pericarditis has been noted in patients undergoing HD versus peritoneal dialysis; however, the difference in the incidence was not clinically significant.[2,4] Risk factors for UP include male sex, advanced age, diabetes, systemic infection, acidosis, serum albumin and phosphocalcic disorders.[3] The definitive pathophysiology of UP is unclear but there have been few hypotheses related to metabolic derangements such as changes in acid-base

homeostasis, abnormal serum calcium levels, hyperparathyroidism and hyperuricemia, and accumulation of toxic metabolites and nitrogenous metabolic end products such as BUN. This ultimately leads to the release of pro-inflammatory markers, and consequent damage to the pericardium causing inflammation as well as increase in permeability of the pericardium resulting in effusion.[3,4] The pericardial effusion in UP is exudative due to infiltration of mononuclear (predominantly lymphocytic) cells and concurrent polymorphonuclear (neutrophilic) cells which may be seen in patients with superimposed bacterial pericarditis.[2,4] The prevalence of asymptomatic pericardial effusion has been reported to be 70–100 % in patients with UP and dialysis-associated pericarditis, which if accompanied by hypotension is clinically suggestive of cardiac tamponade.[4]

The clinical presentation of patients with UP is similar to those with pericarditis due to other etiologies and is characterized by pleuritic chest pain (often exacerbated by lying flat and alleviated by leaning forward), which may or may not be associated with fever, chills, malaise and dyspnea. Small pericardial effusions may remain asymptomatic and detected on routine diagnostic testing. Larger pericardial effusions, however, may present with hemodynamic instability characterized by hypotension and symptoms of hypoperfusion due to decrease in venous return in the setting of impaired compliance. Pertinent physical exam findings include pericardial friction rub, Kussmaul's sign and pulsus paradoxus. Supraventricular tachycardias, particularly atrial fibrillation, are the most common arrhythmias reported in patients with UP.[2,4]

Although TTE helps with definitive diagnosis, other serum laboratory tests including CBC, serum chemistries, inflammatory markers, and cardiac biomarkers along with 12-lead EKG and chest x-ray usually comprise the initial diagnostic testing. Leukocytosis is seen in 40–60 % of patients with uremic pericarditis.[4] Elevated serum levels of erythrocyte sedimentation rate and C-reactive protein are also frequently noted. Cardiac biomarkers such as elevated troponin levels and creatine kinase are related to underlying myocardial inflammation and/or decreased clearance in patients with ESRD.[2,4] A retrospective case control study found that tachycardia (>100 beats per minute), hyperkalemia (>5 mEq/L) and hypocalcemia (corrected calcium <8 mg/dL) were highly specific for pericardial effusion in CKD patients with hypocalcemia alone being predictive of moderate to large pericardial effusion.[5] EKG findings include diffuse PR depressions and ST elevations; however, these may be normal in patients with UP.[2] Chest x-ray is usually significant for enlarged cardiac silhouette with blunting of cardiophrenic angles and obliteration of retrocardiac space in lateral view referred to as water-bottle or flask configuration.[2] TTE is the diagnostic procedure of choice which will demonstrate pericardial effusion and will also help assess alteration in ventricular function. Cardiac magnetic resonance imaging and non-contrast chest CT may be required for complicated or questionable pericarditis.[4]

The important management for patients not on dialysis would be to begin one and intensify dialysis sessions for patients already receiving HD. UP in hemodynamically stable patients should be managed with intensive HD,[2,6] although peritoneal dialysis (PD) can also be used in patients resistant to HD.[2] TTE should be performed to assess the size of the pericardial effusion every 3–5 days.[6] If it is resolving along with clinical improvement, the patient may resume their maintenance dialysis as described in this case. However, if the effusion is increasing in size and there is clinical decompensation, further surgical options with pericardiostomy, pericardial window or pericardiectomy should be considered.[2,6] These options remain as the last resort for patients in UP, as they carry high risk of hemopericardium due to dysfunctional platelets in uremia and loss of antithrombin III in ESRD.[2] In patients with recurrent pericarditis, pericardiectomy is the preferred definitive procedure of choice.[2,4] UP in patients with renal transplant has limited information, however, management of these patients is no different.[6]

## 4. Conclusion

HD should be considered as first line management for patients with uremic pericarditis and pericardial effusion. Intensification of HD should be considered based on clinical presentation and severity of presentation including cardiac tamponade.

## Disclosures

The authors have nothing to disclose.

## Funding

No funding sources.

### Conflict of interest

The authors have no conflicts of interest to disclose.

## References


1. Bentata Y, Hamdi F, Chemlal A, Haddiya I, Ismaili N, El Ouafi N. Uremic pericarditis in patients with end stage renal disease: prevalence, symptoms and outcome in 2017. *Am J Emerg Med*. 2018;36(3):464–466. https://doi.org/10.1016/j.ajem.2017.11.048.
2. Chugh S, Singh J, Kichloo A, Gupta S, Katchi T, Solanki S. Uremic- and dialysis-associated pericarditis. *Cardiol Rev*. 2021; 29(6):310–313. https://doi.org/10.1097/CRD.0000000000000381.
3. Dad T, Sarnak MJ. Pericarditis and pericardial effusions in end-stage renal disease. *Semin Dial*. 2016;29(5):366–373. https://doi.org/10.1111/sdi.12517.
4. Rehman KA, Betancor J, Xu B, et al. Uremic pericarditis, pericardial effusion, and constrictive pericarditis in end-stage renal disease: insights and pathophysiology. *Clin Cardiol*. 2017;40(10):839–846. https://doi.org/10.1002/clc.22770.
5. Ravi V, Iskander F, Saini A, Brecklin C, Doukky R. Clinical predictors and outcomes of patients with pericardial effusion in chronic kidney disease. *Clin Cardiol*. 2018;41(5):660–665. https://doi.org/10.1002/clc.22946.
6. Alpert MA, Ravenscraft MD. Pericardial involvement in end-stage renal disease. *Am J Med Sci*. 2003;325(4):228–236. https://doi.org/10.1097/00000441-200304000-00009.