***Case report***

**Recurrent Pulmonary Edema and Cardiovascular Collapse in a Non-Compliant Dialysis Patient with Severe Aortic Regurgitation: A Clinical Challenge**

**Abstract**

 This case report describes a 36-year-old morbidly obese male with chronic kidney disease (CKD) and non-compliance to dialysis presenting with acute-on-chronic heart failure complicated by flash pulmonary edema and subsequent cardiac arrest. Despite aggressive management, including hemodialysis and inotropic support, the patient deteriorated rapidly and expired. The case highlights the complex interplay between CKD, cardiovascular disease, and non-cardiogenic pulmonary complications, underscoring the challenging management decisions in such critically ill patients.

**Introduction**

Chronic kidney disease (CKD) is characterized by the presence of kidney damage or an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m², persisting for 3 months or more, irrespective of the cause. CKD is a state of progressive loss of kidney function, ultimately resulting in the need for renal replacement therapy, such as dialysis or transplantation [1].

Patients with end-stage renal disease (ESRD), but also patients with slightly reduced kidney function, exhibit a high cardiovascular burden, leading to an increased risk of death, major cardiovascular events, and hospitalization. Hereby, a CKD-associated non-traditional risk factor, such as inflammation, is strongly related to a marked increase in cardiovascular mortality [2].

Pulmonary edema is a leading cause of dyspnea and hospital admissions, resulting from fluid accumulation in the alveoli, leading to ventilation-perfusion mismatch and shortness of breath. It is classified into cardiogenic and non-cardiogenic types, with heart disease being the most common cause. Non-cardiogenic pulmonary edema is often linked to conditions like acute respiratory distress syndrome (ARDS) and pulmonary embolism. Additionally, renal failure, particularly in patients noncompliant with hemodialysis, is an emerging cause [3].

**Case presentation**

A 36-year-old morbidly obese male with a known history of Chronic Kidney Disease on dialysis where is advised to do 3 dialysis sessions per week but is non-compliant and performs only 1 session per week when he presents with fluid overload, Type 2 Diabetes Mellitus, Hypertension, and Cerebrovascular accident (TIA in 2019), presented to the hospital on with complaints of sudden onset of precordial chest pain and shortness of breath. ECG showed ST depressions in V4, V5, and V6 with negative cardiac biomarkers. The possible differentials were Acute Coronary Syndrome vs Acute Pericarditis. As a result, he was started on hemodialysis on 2 consecutive days. Over the period, there was a reduction in Creatinine and Urea levels and a decrease in his chest pain. On day 2, the patient developed Atrial Fibrillation with fast ventricular response, for which he was on Amiodarone infusion, and the rhythm was immediately reverted to sinus rhythm. It was then noted that the patient started experiencing an increase in his chest discomfort with orthopnea, and serial cardiac biomarkers were shown to be elevated. Chest X-ray revealed flash pulmonary edema. 2-D ECHO was performed, which showed dilated LVH with anteroseptal hypokinesia, enlarged LA, and Mild mitral stenosis. Severe Aortic Regurgitation. Normal EF. It was then decided that the patient would undergo CT Coronary Angiography, but due to the high calcium score present, CTCA was not completed. Thus, he underwent urgent Coronary Angiography, and before that patient was intubated and sedated and was then shifted to the Cath lab. CAG showed mild coronary artery disease for medical management. Post CAG, the patient underwent hemodialysis with the maximum dose of Inotropes to maintain his blood pressure. Repeated Arterial blood gases show hypoxemia with a high FiO2 setting in the ventilator. Post-dialysis Chest X-ray does not show an improvement in the pulmonary edema. It is also noted that the patient has been having consistently low diastolic blood pressure readings in this admission, compared to his previous blood pressure readings when he would come for his dialysis sessions, which are usually uncontrolled. Thus, considering his low diastolic blood pressure, ECHO findings, and repetitive pulmonary edema, as per the cardiologist, it was advised that the patient should undergo urgent Aortic Valve Replacement to salvage his life.

The patient was on hemodialysis daily, but his oxygen level did not improve significantly, with a recurrence of pulmonary edema. On day 9, despite increasing FiO2 to 100%, the patient’s oxygen saturation was not improving. On chest auscultation, there were reduced breath sounds bilaterally. Blood-tinged sputum was noted in the endotracheal tube, and upon suctioning, red blood secretions were observed. There was a recurrence of massive pulmonary edema. It was decided to reposition and secure the endotracheal tube, but the patient suddenly crashed with desaturation and bradycardia, followed by asystole. Code blue was initiated, and ACLS protocol was followed, but despite high-quality CPR, the patient could not be revived. Death was declared after 26 minutes.

**Discussion**

Cardiogenic Pulmonary Edema (CPE) is defined as the accumulation of excessive fluid in the interstitial and alveolar spaces of the lung as a result of elevated capillary hydrostatic pressure after elevated pulmonary venous pressure, which is brought on by elevated heart filling pressures. CPE can result from any cardiac condition that raises the pressure in the left side of the heart. However, CPE may cause elevated cardiac filling pressures without heart disease due to a variety of other clinical situations of non-cardiological origin, such as severe hypertension, severe renal illness, and primary fluid overload [4]. This condition can quickly worsen and cause serious hypoxia if it is not identified and treated quickly enough [5]. These patients typically have a dismal prognosis, with an in-hospital death rate of 15–20%, a one-year survival rate of 50%, and a six-year follow-up mortality rate of 85%. The prognosis is primarily determined by the underlying medical disorders.

Excessive fluid buildup in the interstitial and alveolar spaces in the lungs, caused by an imbalance in the "Starling forces,” an increase in the permeability of the alveolar-capillary barrier, or an obstruction of the draining lymphatic system, is said to be the main pathophysiological mechanism [6]. The pulmonary capillary hydrostatic pressure typically falls between 6 and 13 mmHg. Proteins in human plasma determine the oncotic pressure, which is typically 21.1 mmHg for people under 50 and 19.7 mmHg for people 70 to 89 years old. The balance of these forces typically keeps the interstitium "dry." Furthermore, the lymphatic system can counteract an initial increase in the volume of fluid flowing from the pulmonary capillaries to the interstitial spaces. The rise in pulmonary capillary hydrostatic pressure in patients with CPE is caused by an increase in pulmonary venous and left atrial (LA) pressure, which most often results from a higher LV filling pressure. The Starling law states that for edema to develop, the pressure must rise above the plasma colloid osmotic pressure's typical value [4].

Flash pulmonary edema (FPE) is a serious form of CPE that could translate into hypoxemic respiratory failure [7]. Renal artery stenosis predisposes patients to develop FPE via a worsening of diastolic dysfunction and an increase in haemodynamic burden, increased vascular permeability brought on by high levels of angiotensin in the blood, and defective natriuresis. Reduced tubular salt reabsorption and natriuresis result from the sudden rise in blood pressure brought on by a renovascular process [8].

Dyspnoea, bilateral lower-extremity oedema, and chest radiographs displaying bilateral alveolar filling patterns are typical symptoms of PE patients [9]. In order to treat it, supportive methods like mechanical breathing, maintaining a healthy diet, and reducing the pulmonary artery wedge pressure using diuretics, ultrafiltration (during haemodialysis), and fluid restriction are necessary.

Since our case involved a patient who had a known long-standing history of CKD, the incidence of pulmonary complications was almost inevitable. Both renal and non-renal reasons can result in a pleural effusion, which can exacerbate chronic kidney disease. In a cross-sectional study of pleural effusion among cases of CKD, Pleural effusion was seen in 5.9% of postrenal transplant recipients and 6.7% of patients with CKD (stages 3 to 5) [10]. Similarly, CPE could be another culprit. An analysis study emphasised the significance of pulmonary oedema as a solid reason for chronic dialysis (CD) patients to be admitted to intensive care, with a 10% fatality rate. A rich past medical history, dominated by hypertension, previous episodes of PE, and ischaemic heart disease, is characteristic of those impacted. In the study population, the most common cause of PE was cardiovascular diseases. Nonetheless, lung infections and extracellular volume expansion or fluid overload brought on by inadequate diet adherence and inaccurate dry weight assessment were also significant contributors to this condition [11].

An original article by Jimnaz P.A.et al revealed that excess interdialytic weight gain (34%), hypertensive crisis (18%), pulmonary infection (15%), and heart failure (7%) were the causes of acute PE in CD patients. The study included patients with a mean dialysis duration of 3.3 ±1.7 years. Approximately 41% had dialysis for less than 2 years, while 30% had dialysis for 3-4 years. The majority of patients demonstrated compliance to dialysis and medications, while just 4% of patients showed noncompliance to dialysis stoppage drugs, and 2% of patients reported stoppage of antihypertensives [12]. The situation strategically varies in cases of dialysis noncompliance. CKD patients generally consider dialysis sessions as an intrusion to their daily activities and thus lead to their noncompliance with proper treatment. A study showed that during the 347,636 person-years of follow-up, 87 individuals passed away, for a mortality rate of 9.1 per 100 person-years. The average follow-up period was 1493 days, and 54% of deaths were caused by cardiovascular causes, with 14.9% being sudden deaths, 13.8% being acute myocardial infarctions, and 13.8% being strokes; the remaining deaths were caused by acute PE, arrhythmias, or ischaemia. Interdialytic weight gain was an independent predictor of all-cause mortality in haemodialysis patients [13].

There were rare instances where mild noncompliance did not result in direct mortality. In a case study by Natalia Farha et al, the patient had presented with new-onset haemoptysis in addition to the classic symptoms of CPE. It was revealed later on that he had missed a single haemodialysis session. The patient was started on antibiotic prophylaxis and kept on haemodialysis for three consecutive days. Eventually, the patient showed significant improvement clinically [3]. This was unfortunately not the case for our study. A study on an atypical presentation of acute unilateral pulmonary edema (UPE) in a patient with moderate-severe aortic insufficiency during left ventricular assist device (LVAD) support provided recommendations for future treatment. The aortic insufficiency seemed to deteriorate the patient’s condition, indicating the importance of timely diagnosis. The proper evaluation of cardiogenic causes from other causes served as an important tool to necessitate effective management [14]. An RCT showed that frequent sessions of haemodialysis were associated with enhanced hypertension and hyperphosphatemia. Patients subjected to frequent sessions of haemodialysis were more likely to undertake vascular access than those assigned to conventional haemodialysis [15].

**Conclusion**

This case underscores the critical importance of adherence to dialysis in CKD patients to prevent life-threatening complications such as flash pulmonary edema and acute heart failure. Non-compliance with treatment regimens significantly increases the risk of adverse outcomes, including mortality. Early recognition and prompt management of pulmonary edema are crucial in improving patient outcomes, although in severe cases like this, the prognosis remains poor despite aggressive intervention. Future research and clinical efforts should focus on optimizing therapeutic strategies to mitigate the impact of cardiovascular complications in CKD patients.

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