# Cardiac Arrest in a Patient with Mild Bilateral Pleural Effusion: A Case Report

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## ABSTRACT

**Introduction: Cardiopulmonary arrest is the termination of blood circulation caused by a diversity of causes, some of which have recently been identified, such as opioid overdose and congenital heart abnormalities. Etiology of the condition includes both in-hospital and out-of- hospital causes, such as myocardial infarction, cardiac arrythmias, pulmonary embolism, massive pleural effusion, cardiac tamponade, hypoxia, and others. Patients are found unresponsive, and the specific cardiopulmonary resuscitation pathways are initiated according to whether the patients display ventricular fibrillation/ventricular tachycardia rhythm or pulseless electrical activity/asystole rhythm. In many cases, cardiac arrest is a preventable condition, when reversible causes such as hypoxia, acidosis, thrombosis, and hypothermia have been avoided.**

**Our case study will serve to present the first case of mild bilateral pleural effusion in a patient with no other predisposing reversible causes of cardiac arrest.**

**Case: Our case serves to describe and analyze the etiologies of cardiopulmonary arrest in a patient with long standing, bilateral pleural effusion. Being the first case in the literature to describe such a correlation, our case study aims to exclude and provide a connection between mild effusion and cardiac arrest, while demonstrating that reversible causes of cardiac arrest have been excluded. The case further demonstrates the clinical and radiological evidence and highlights the literature studies that have described effusions that have led to cardiac arrest. This will further add to the existing literature by making researchers consider future risks associated with untreated mild bilateral effusion.**

**Conclusion: Although cardiopulmonary arrest is frequent, its association with bilateral pleural effusion has not yet been evaluated. Our case shows a potential etiology that must be considered when explaining complications of untreated pleural effusion to the patient and serves as an opportunity for future research to evaluate the potentiality of mild pleural effusion in causing cardiac arrest.**

**Keywords: Cardiopulmonary arrest, Pleural effusion**

## INTRODUCTION

Cardiac arrest is condition characterized by the termination of circulation due to a diversity of causes, both cardiac and noncardiac[1]. Cardiac arrests are also categorized into in- hospital-cardiac arrest (IHCA), which occur at an incidence of approximately 1 to 1.5 per 1000 hospital admissions, and out-of-hospital cardiac arrest (OHCA), which occur in 55 per 100,000 inhabitants[2]. Within IHCA, a recent cohort study demonstrated that noncardiac causes, which have recently been termed as "presumed cardiac causes", only make up a minority of cardiac arrests, and include acute coronary syndrome and ventricular arrythmias[1]. According to guidelines, management of cardiac arrest starts with cardiopulmonary resuscitation, which follows a specific algorithm. Defibrillation is only given when the heart rhythm is shockable, such as in ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT), whereas only epinephrine boluses and cardiopulmonary resuscitation is done when the rhythm is unshockable, like asystole or pulseless electrical activity (PEA)[3]. In all cases, treatment of cardiac arrest should target the underlying etiology, hence which the patient outcomes will be more favorable[1]. In our case we present a case of "presumed cardiac arrest" in a patient with long standing, untreated, minor bilateral effusion and chronic kidney disease undergoing successful dialysis sessions through a permcath. We aim to demonstrate the etiologies of cardiac arrest, signifying that minor pleural effusion might be a potential cause in cardiac arrest, which prioritizes further future research.

## CASE PRESENTATION

A 58-year-old female, known case of Type 2 Diabetes Mellitus (T2DM), Hypertension (HTN), chronic kidney disease (CKD) stage 3 b, hyperlipidemia, squamous cell carcinoma in the perineum (with previous chemoradiotherapy in 2019 in and was advised surgery but refused) presented to the emergency department, with progressively worsening symmetrical bilateral lower limb pitting edema, abdominal distension, and chronic shortness of breath. She had recurrent

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admissions, with the same presentation of dyspnea, and was offered thoracocentesis her bilateral pleural effusion for diagnostic and therapeutic reasons, however she refused, and asked for conservative medical therapy. However, in this admission, the patient was admitted directly to the intensive care unit (ICU), in view of her presentation of acute dyspnea and hypertensive pulmonary edema, pleural effusion, and worsening CKD. She was subsequently initiated on Furosemide and nitroglycerin (GTN) infusion. she was found to have increase in systolic pulmonary artery pressure (SPAP) on Echocardiography (ECHO), and Domain-dimer (D-dimer) was 1.49. At the time, ultrasound doppler of the lower limbs was negative for deep vein thrombosis (DVT). Upon hemodynamics stabilization, since the blood pressure was maintained after stopping intravenous antihypertensives and maintained only on oral antihypertensive agents, she was shifted back to the medical ward. In the medical ward, however, the patient started to progressively produce low amounts of urine with rising creatinine levels.

Once she was hemodynamically, clinically, and radiologically stable, she was shifted to the medical ward, shortly after which she continued to have oliguric Acute Kidney Injury (AKI) on top of her CKD, with volume overload not responding to high dose diuretics. Hence, the patient was shifted to ICU for Continuous Renal Replacement Therapy (CRRT) via a Vascath.

Hence, our colleague nephrologists have recommended to continue hemodialysis and to insert Permacath, both of which were subsequently done. She improved clinically and radiologically, and creatinine levels decreased to baseline following regular hemodialysis sessions. She remained clinically stable for one week within the medical ward, and she underwent hemodialysis through a patent Permcath uneventfully the day before discharge. Additionally, the patient had no complaints and no chest pain and was not breathless.

As the patient was hemodynamically and clinically stable, and laboratory investigations have returned to her baseline, she was planned to be discharged home.

On the day of her discharge, the patient was seen in morning rounds, she was conscious, alert, oriented and vitally stable. Her heart rate and blood pressure were maintained round 60 to 70 beats per minute (bpm) and 120-135 millimeters mercury (mmHg), respectively. During morning rounds, the patient was seen with her son, and was communicating freely, not in distress and had no complaints. She had finished her breakfast early in the morning and was going to take a nap. Shortly after morning rounds, she was found unresponsive by the nursing staff and cardiopulmonary resuscitation (CPR) was immediately started. The patient underwent eleven cycles of CPR, and the electrical monitor one rhythm of pulseless electrical activity (PEA) and all other rhythms demonstrated asystole. She received a total of 5 milligrams (mg) of epinephrine, after which Return to Spontaneous Circulation (ROSC) was achieved, she was intubated, and was shifted to ICU, and started on heparin continuous infusion and vasopressors. ECG post ROSC showed normal sinus rhythm and no ST T wave changes.

Upon investigation of the etiology of her cardiac arrest, Computer Tomography of the Pulmonary Artery (CTPA), as seen in Figures 1 and 2, done ruled out Pulmonary Embolism (PE) and Computer Tomography (CT) of the brain showed no acute ischemic or hemorrhagic insult. Laboratory investigations showed increased creatinine (200umol/L from baseline 170), mild hypokalemia 3.3 mmol/L, coagulation studies were normal, and white cell count (WCC) was elevated at 16.62 x 10^9 per Liter. Hemoglobin (Hb) was stable 8.7 (baseline). Blood cultures from left and right arms and Permcath vein and urine cultures showed no organisms, and sputum culture showed normal respiratory flora, endotracheal aspirates showed suppressed normal flora with predominance of Candida tropicalis. ECHO showed normal ejection fraction (EF) 60% with no regional wall motion abnormalities (RWMA) and PAP 50 millimeters mercury (mmHg). Blood sugar readings were controlled.

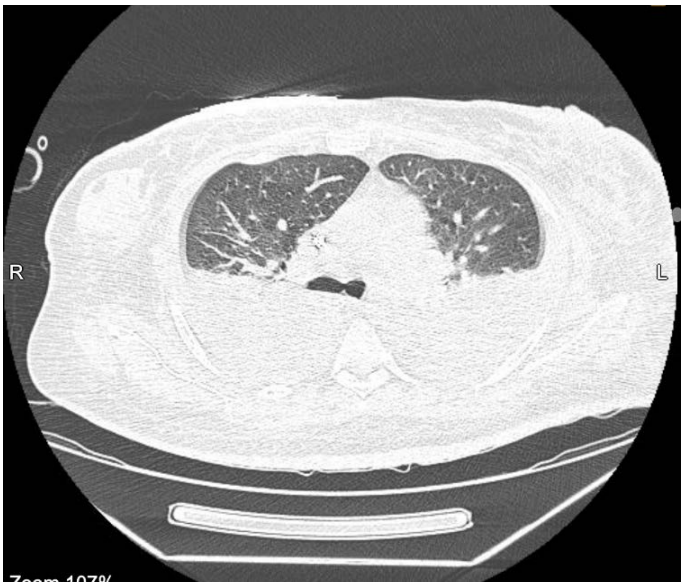


**Figure 1:** Axial CT of Lungs showing Bilateral free-flowing pleural effusions [48 mm in depth] with underlying lower lobes as well as upper lobar posterior segmental compression collapse

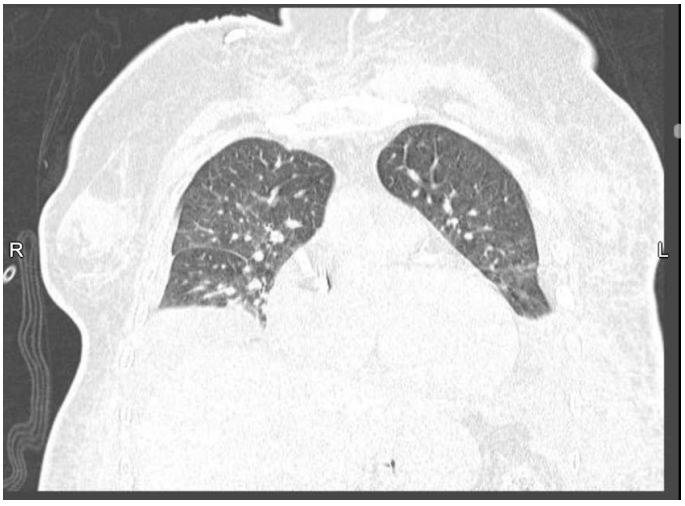


**Figure 2:** Coronal CT chest showing enlarged heart with no aortic aneurysm or dissection flap

While she was planned for discharge, her medications were as follows: Nifedipine prolonged release tablet 90 mg once daily (OD), hydralazine tablets 25 mg three times daily (TDS), Movicol sachets OD, Sodium Bicarbonate 650 mg tablet OD, Heparin prophylaxis subcutaneously, Cholecalciferol 50,000 international unit (IU) capsules once weekly, intravenous Paracetamol 500 mg twice daily when needed, Ferrous Sulphate 80 mg OD, and Omeprazole for gastrointestinal prophylaxis.

In ICU, the patient was gradually weaned of sedation, Glasgow Coma Scale (GCS) remained 3 to 4 points, and repeated CT brain showed global hypoxic brain injury. She was maintaining hemodynamics off vasopressor support, underwent tracheostomy and kept on mechanical

ventilation with fio2 40% 2 hours and CPAP 2 hours t-piece. She continued to undergo dialysis uneventfully. Also kept on Piperacillin-Tazobactam prophylactically while in ICU as per hospital protocol.

## DISCUSSION

Managing the underlying etiology of cardiac arrest is essential for the patient's survival. Firstly, reversible causes are known as: hypoxia, acidosis, hypovolemia, hyper and hypokalemia, and hypothermia, toxins, thrombosis, tension pneumothorax, and tamponade[4]. Hypoxia was ruled out with normal oxygen saturation, and acidosis was ruled out by normal venous blood gas (vbg) parameters. Additionally, hypovolemia was excluded since the patient had no signs of dehydration, was maintaining good oral and fluid intake, with good urine output. There were no electrolyte abnormalities, especially potassium. The patient's temperature was normal at 36.7 to 37 degrees Celsius throughout her stay. Toxins were excluded as a possible cause as the patient's medications were reviewed daily. Venous and pulmonary thrombosis were ruled out by ultrasound doppler of the lower limbs and computed tomography of the pulmonary artery. Chest x-ray and clinical examination revealed no signs of tension pneumothorax nor of tamponade. The only evidence on CTPA was bilateral pleural effusion. Electrocardiography (ECG), although being low voltage since previous admissions, showed no ST or T wave changes, and troponin was mildly elevated at 87 due to type 2 myocardial injury, which had resulted from the hypertensive fluid overload she was admitted with initially. Moreover, MI was ruled out since the patient had no chest pain, no elevation in cardiac markers, and Echocardiography (ECHO) showed no new regional wall motion abnormalities and systolic ejection fraction (EF) was 55%, and pulmonary artery pressure was only mildly increased from 40 to 50 from the previous admission. There was no drop in hemoglobin. The patient is not known case of coronary artery disease and no previous coronary angiography was done. The nurses had confirmed no aspiration episodes, as the patient was not eating or drinking, and airway obstruction was ruled out as the patient was saturating normally and was talking to her brother shortly before she was found unconscious.

After ruling out the reversible causes in our patient, we have concluded that bilateral pleural effusion could have been a potential cause in the cardiac arrest. Pleural effusion is common in routine medical practice and can be due to many different underlying diseases. Precise differential diagnostic categorization is essential, as the treatment and prognosis of pleural effusion largely depends on its cause[5].

Most patients who have a newly detected pleural effusion should undergo diagnostic thoracentesis to determine the nature of the effusion (transudate or exudate) and to identify potential causes. The exceptions are when there is only a small amount of pleural fluid with a secure clinical diagnosis (viral pleurisy), or when there is clinically obvious heart failure without atypical features (fever or bilateral effusions of significantly disparate sizes)[6]. Therapeutic thoracentesis is commonly performed for symptom relief (dyspnea) or if the fluid has imaging characteristics of a complicated pleural effusion, such as loculations suggesting a parapneumonic pleural effusion[7].

Additionally, a tension hydrothorax is defined as a massive pleural effusion presenting with hemodynamic abnormalities secondary to mediastinal compression. In these patients, pleural volume increases intrathoracic pressure to the point of compromising diastolic filling and cardiac output simulating a cardiac tamponade physiology[8]. Tension hydrothorax can result from a number of causes, including trauma, chylothorax, pancreatitis, cirrhosis, parapneumonic effusions, and autoimmune diseases, but by far the most common cause is malignancy[8]. This is an uncommon yet potentially fatal medical emergency, which if left untreated, may progress to cardiac arrest. Hence, early detection and rapid intervention of these patients prevents cardiorespiratory collapse[8].

In our case the CTPA showed bilateral, mild, free-flowing pleural effusions [48 mm in depth], with no filling defects, and ECHO showed no signs of tamponade as well. Henceforth, we conclude that bilateral mild pleural effusions, can be a potential cause leading to cardiac arrest.

## CONCLUSION

**Since reversible causes of cardiac arrest have been addressed and ruled out during resuscitation, our case explains a new possible etiology for cardiac arrest, which is the presence of mild bilateral effusions. Whether unilateral mild effusions might be a potential trigger for cardiac arrest is something that future research is eligible to evaluate. Therefore, our case serves as an opportunity to avoid leaving pleural effusions untreated for the possibility of causing severe outcomes such as cardiac arrest.**

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**Consent:** Patient relatives have given her informed consent for the case to be published, as the patient remains unconscious.

**Clinical Message:** This article is relevant in its confirmation that considering mild effusions as potential causes of cardiac arrest.

**Author's Contribution:** Dr. Nayla Al Khalifa, Dr. Daher Al Arab, and Dr. Hussein Farhoud, and Dr. Walid Assar are satisfied with this research publication. They aimed to demonstrate potentiality of mild effusions in causing cardiac arrest.

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**Potential Conflict of Interest**: None

**Competing Interest**: None

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