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Case Report / Olgu Sunumu

Non-Cardiogenic Pulmonary Edema and Acute Respiratory Failure after Right Heart Catheterization: A Rare Complication with A Diagnostic Dilemma

Sağ Kalp Kateterizasyonu Sonrası Nadir Bir Komplikasyon; Non-Kardiyojenik Akciğer Ödemine Ikincil Akut Solunum Yetersizliği

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Abstract

Although right heart catheterization (RHC) is generally a safe procedure, serious adverse events can occur during the procedure. We report a case of non-cardiogenic pulmonary edema and respiratory failure after RHC to assess pulmonary arterial hypertension in a patient who was a candidate for lung transplantation and discuss the differential diagnosis of pulmonary edema.

Keywords: Pulmonary edema, respiratory failure, air embolism, right heart catheterization

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Öz

Sağ kalp kateterizasyonu genellikle güvenli bir girişim olsa da nadiren hayatı tehdit edici komplikasyonlar görülebilmektedir. Bu vakada bronşektaziye bağlı solunum yetersizliği nedeniyle akciğer transplantasyonuna hazırlanan 24 yaşında erkek hastada, sağ kalp kateterizasyonu sonrasında gelişen nonkardiyojenik akciğer ödemi tablosu sunulmuş ve ayırıcı tanısı tartışılmıştır.

Anahtar sözcükler: Akciğer ödemi, solunum yetmezliği, hava embolisi, sağ kalp kateterizasyonu

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Introduction

Here, we report a case of non-cardiogenic pulmonary edema (NCPE) and respiratory failure after right heart catheterization (RHC) to assess pulmonary arterial hypertension in a patient who was a candidate for lung transplantation (1).

Although RHC is generally safe, serious adverse events, such as bleeding at the site of vascular access, pneumothorax, arrhythmia, myocardial, vessel, or valve rupture, thromboembolism, infections, and air embolism, can occur during the procedure (2).

Non-cardiogenic pulmonary edema, characterized by protein and fluid accumulation in the alveoli with an increase in capillary membrane permeability and normal pulmonary capillary pressure, is caused by various direct and indirect disorders.

Case Report

A 24-year-old male patient with bronchiectasis was scheduled for lung transplantation due to rapid deterioration of respiratory functions (forced expiratory volume 1: 0.17 l, 17%). RHC was performed by an invasive cardiologist to assess pulmonary hypertension. Before the procedure, the patient had no complaint. His vital signs were all normal [blood pressure (BP): 113/68 mmHg, heart rate (HR): 82 beats/min, and peripheral oxygen saturation: 92% (on room air)]. Figure 1 shows the chest X-ray of the patient. The procedure was performed using a local anesthesia agent (prilocaine 20 mg), and no systemic drug or contrast agent was administered. After the catheterization procedure, the patient experienced acute severe respiratory failure.

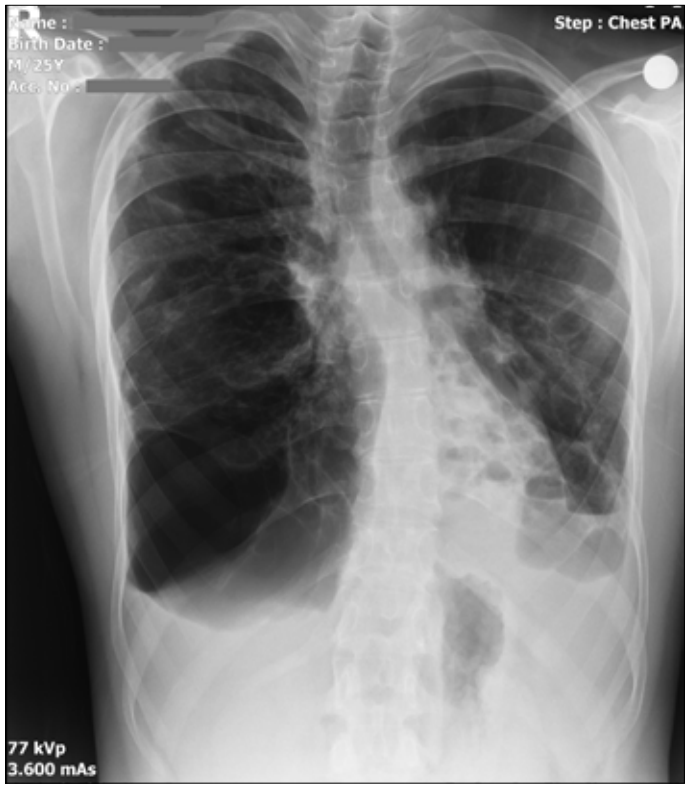


Figure 1. Chest X-ray before the procedure. Air cyst in the outer part of the right lower region, marked bronchiectasis in the left lower region, the left sinus is closed, and pleural thickening on the right side.



Figure 2. Chest X-ray after the procedure. A newly developed bilateral diffuse infiltration.

On physical examination, results were BP: 135/86 mmHg, HR: 186/tachypnea (46 breaths/min) regular, body temperature: 36.9°C, and oxygen saturation: 81% (6 l/min with nasal cannula). Cyanosis, subcutaneous emphysema, or jugular filling was not observed. Heart examination was normal except for tachycardia. Fine crackles were bilaterally heard over the basal lung areas.

Further examination revealed sinus tachycardia on electrocardiography and newly developed bilateral diffuse intensity on chest X-ray.

Table 1 shows the laboratory results as before and after the procedure. The patient had no electrolyte disturbance and abnormality of liver and kidney functions.

On echocardiography, normal left ventricular ejection fraction, normal biventricular contraction, and normal tricuspid valve structure with minimal tricuspid insufficiency were found. No diastolic dysfunction was observed.

On RHC, pre-capillary pulmonary hypertension was observed. Pulmonary capillary wedge pressure was 10 mmHg, and mean pulmonary arterial pressure was 32 mmHg.

Hypervolemia is not an option due to negative fluid balance. There was no decrease in hemoglobin value; no blood product was transfused. Deep vein thrombosis was not detected in the bilateral lower extremity Doppler ultrasonography (USG).

This young adult presented with acute respiratory distress after catheterization, accompanied with hypoxemia, hypercarbia, tachycardia, neutrophil-predominant hyperleukocytosis, and bilateral diffuse intensity on chest X-ray (Figure 2). He was diagnosed with acute mixed-type respiratory failure secondary to NCPE. The patient was admitted to the intensive care unit (ICU) and supported by non-invasive mechanical ventilation (BIPAP-ST 15/5 cm H_2O) bronchodilator and oxygen treat-

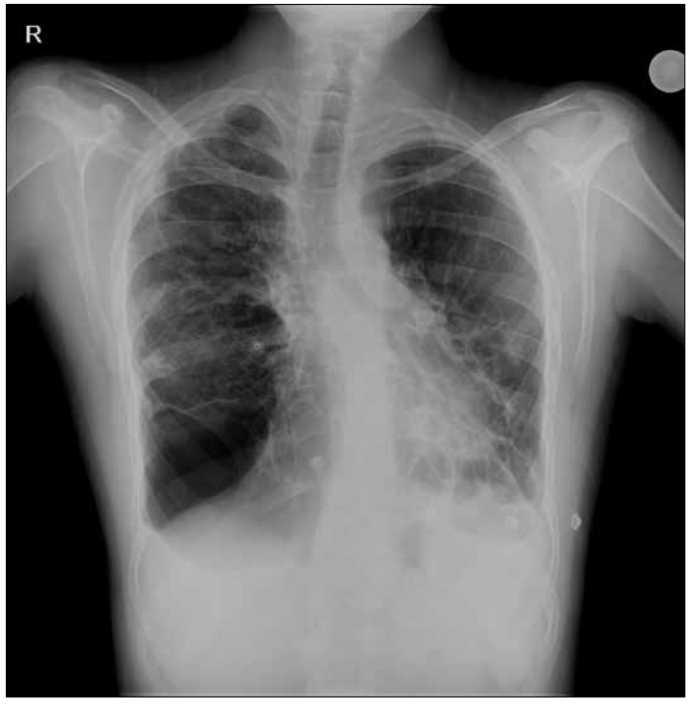


Figure 3. Chest X-ray after discharge from the ICU. Total recovery of pulmonary edema.
ICU: intensive care unit

Table 1. Laboratory data

Variable	Reference range	Before procedure	After procedure
Hematocrit (%)	41–53	31.6	49
Hemoglobin (g/dL)	13.5–17.5	9.3	13.9
WBC* (/mm^3)	4000–10000	8700	98,800
Neutrophil	40–70	83	97,8
Lymphocytes	22–44	9.9	1
Platelet (/mm^3)	150,000–440000	255000	488000
aPTT** (s)	22–35	29.3	33
INR***	0.9–1.1	1.2	1.19
D-dimer (µg/mL)	0–0.5	0.41	1.39
LDH# (U/l)	0–248	196	270
Troponin I (ng/mL)	<0.01	<0.01	<0.01
Lactate (mmol/l)	0.5–1.6	1.5	3.3
NT-proBNP## (pg/mL)	0–125	93,5	--
CRP### (mg/l)	0–5	<3.14	4.20
Procalcitonin (ng/mL)	0–0.5	0.04	0.35
Arterial blood gases			
pH	7.35–7.45	7.42	7.29
$PaCO_2$ (mmHg)	35–48	42	69
PaO_2 (mmHg)	83–108	100	51
HCO_3 (mmol/l)	21.0–25.0	26.9	31.3
Base excess (mmol/l)		2.7	-1.3
O_2 saturation (%)		100	81
$FiO_2$$ (%)		21	44
PaO_2/FiO_2 (mmHg)		476	116

*White blood count
**Activated partial thromboplastin time
***International normalization ratio
#Lactate dehydrogenase.
##N-terminal pro-B-natriuretic peptide
###C-reactive protein
$Fraction of inspired oxygen

ment. Intravenous methylprednisolone was administered considering a possible allergic reaction. The patient was discharged from the ICU with initial respiratory functions after 3 days. Figure 3 shows the chest X-ray of the patient after discharge from the ICU. Written consent was obtained from the patient for this case report.

Discussion

The most impressive features of the case are newly onset bilateral diffuse consolidation on chest X-ray and hyperleukocytosis. Diffuse consolidation was considered as NCPE due to normal systolic and diastolic functions of the left ventricle on echocardiography. NCPE is caused by various disorders, such as head trauma, sudden airway obstruction, toxic drug intake, blood transfusion, intravenous contrast agent injection, air embolism, pulmonary aspiration, prolonged seizure, and septic shock with factors other than elevated pulmonary capillary pressure. Patient condition rapidly declined and ultimately required mechanical ventilation.

First, as an etiology of pulmonary edema, toxic exposure, and hypersensitivity reaction are unlikely causes because only local prilocaine was administered, and the patient did not respond to systemic steroid therapy. Alveolar hemorrhage can be ruled out with no decrease in hemoglobin level.

Could this patient have had pulmonary leukostasis? Leukostasis refers to hyperleukocytosis combined with organ failure in a patient with extreme blast counts. Respiratory failure due to leukostasis is almost always seen in patients with leukemia.

Pulmonary embolism was also considered in the differential diagnosis with high D-dimer value, but normal bilateral venous Doppler USG of the lower extremities, short recovery period, and normal right heart functions on echocardiography did not support pulmonary embolism.

Air embolism is a rarely diagnosed but potentially life-threatening complication of central venous catheterization. Although air embolism is generally asymptomatic, it can lead to end organ damage with secondary effects (3). Air bubbles reaching the pulmonary microcirculation cause endothelial damage and trigger the inflammatory response. Inflammation further increased with the accumulation of immune cells and mediators. Eventually, accumulation of debris in the alveolar space interferes with the gas exchange and triggers a pulmonary response, including NCPE and serious physiological changes, such as hypoxemia, increased physiological dead space, decreased pulmonary compliance, and increased airway resistance.

Endothelial damage in the pulmonary capillaries activates neutrophils, platelets, and complement system and leads to the release of inflammatory mediators, such as free radicals and cytokines. Hence, hyperleukocytosis can also be accompanied. Extravasation of fluid into the inflamed tissue may contribute to the laboratory findings, such as leukocytosis and thrombocytosis, increasing hemoglobin level consistent with intravascular depletion.

Since other possible etiologies were excluded, air embolism had been considered prior to differential diagnosis. Computed tomography scan, which is the gold standard diagnostic test, could not be performed since the patient was clinically unstable.

Conclusion

Acute respiratory failure due to NCPE secondary to air embolism was resolved with support therapy including positive pressure ventilation and medical therapy. The patient was then discharged from the ICU. Air embolism is a rare complication of catheterization that can have life-threatening consequences, such as systemic inflammatory response syndrome, pulmonary edema, and sudden cardiac death.

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