Unilateral Pulmonary Edema Seen in Patient with Massive Pleural Effusion and Heart Disease: Case Report

Masif Plevral Effüzyonu ve Kalp Hastalığı Olan Bir Olguda Görülen Tek Taraflı Akciğer Ödemi

ABSTRACT Unilateral pulmonary edema is a rare disorder which can be misdiagnosed with other causes of unilaterally pulmonary infiltrates on chest X-ray. Unilateral pulmonary edema may have cardiac origin and non cardiac origin such as mitral valve disease and reexpansion pulmonary edema respectively, while cardiogenic and noncardiogenic pulmonary edema may be difficult to distinguish because of their resembling clinical manifestations. Reexpansion pulmonary edema is an unusual and a potentially significant cardiopulmonary complication occurring after reinflation of the collapsed lung due to pneumothorax, hidrothorax or atelectazy. Unilateral pulmonary edema with mitral valve disease mostly affects the right lung, but cases in the left lung were also reported. We present an interesting case of reexpansion pulmonary edema seen in a 43 year old woman with massive effusion, mitral valve disease and cardiopulmonary bypass history.

Key Words: Pulmonary edema; pleural effusion; heart; heart valve diseases

ÖZET Tek taraflı akciğer ödemi göğüs röntgeninde diğer tek taraflı infiltrasyon nedenleri ile karışabilen ender bir bozukluktur. Tek taraflı akciğer ödemi mitral kapak hastalığı gibi ya da kalp ve reekspansiyon akciğer ödemi gibi kalp dışı nedenli olabilir, bununla birlikte kalp ve kalp dışı akciğer ödeminin ayırt edilmesi, benzer klinik görünümleri nedeni ile zor olabilir. Reekspansiyon akciğer ödemi, hidrotoraks, pnömotoraks ve atelektaziye bağlı kollabe olmuş akciğerin ekspanse olmasından sonra oluşan, nadir, potansiyel olarak önemli bir kardiyopulmoner komplikasyon nedenidir. Mitral kapak hastalığı ile birlikte olan tek taraflı akciğer ödemi çoğunlukla sağ akciğeri etkilemesine rağmen, sol akciğeri etkileyen olgu raporları da yayınlanmıştır. Biz, kardiyopulmoner baypas, mitral kapak hastalığı hikayesi ve masif plevral effüzyonu olan 43 yaşındaki kadın hastada görülen ilginç reekspansiyon akciğer ödemi olgusunu sunuyoruz.

Anahtar Kelimeler: Pulmoner ödem; plevral effüzyon; kalp; kalp kapağı hastalıkları

Turkiye Klinikleri J Cardiovasc Sci 2012;24(3):291-4

W nilateral pulmonary edema (UPE) is a rare clinical entity. UPE is often confused with infectious diseases, neoplasm and cardiac failure which are other causes of unilaterally pulmonary infiltrates on chest X-Ray.¹⁻³ The etiologies of UPE may be reexpansion pulmonary edema (RPE), obstruction of pulmonary vessels, left-to-right intracranial shunt, atrial tumor, mitral valve disease or heart failure.¹⁻⁵ RPE is an unusual and a potentially significant cardiopulmonary complication occurring after reinflation of the collapsed lung due to pneumothorax, hidrothorax or atelectazy.⁵⁻⁷ Although most cases of UPE with mitral valve disease affect the right lung, cardiogenic UPE in the left lung was also reported.^{1,3,8} Cardiogenic and

Mertay BORAN,^a Ertay BORAN,^b Nesligül YILDIRIM^c

^aClinic of Thoracic Surgery, Çankırı State Hospital, Çankırı ^bClinic of Anesthesiology and Reanimation, İzmir Atatürk Training and Research Hospital, İzmir ^cDepartment of Cardiology, Bülent Ecevit University Faculty of Medicine, Zonguldak

Geliş Tarihi/*Received:* 24.04.2010 Kabul Tarihi/*Accepted:* 16.09.2010

It was presented at the 59th ESCVS International Congress in conjunction with the 6th Congress of Update in Cardiology and Cardiovascular Surgery, Izmir, Turkey, 15-18 April 2010.

Yazışma Adresi/*Correspondence:* Ertay BORAN İzmir Atatürk Training and Research Hospital, Clinic of Anesthesiology and Reanimation, İzmir, TÜRKİYE/TURKEY ertayboran@yahoo.com

Copyright © 2012 by Türkiye Klinikleri

Mertay BORAN ve ark.

non cardiogenic UPE, may be difficult to distinguish because of their resembling clinical findings.⁸ We present an interesting case of RPE seen in a woman with massive pleural effusion, mitral valve disease and cardiopulmonary bypass history.

CASE REPORT

A 43 year old woman with a history of coronary heart disease, mitral valve disease, type 2 diabetes mellitus and coronary artery bypass graft surgery performed 2 months ago, presented with progressive dyspnea on exertion for 2 weeks. Her medications included spironolactone, metoprolol, acetyl salicylic acid, glimepiride and gliclazide. The echocardiogram performed 2 months before present admission revealed normal left ventricular function, normal pulmonary arterial pressure and grade 2/4 mitral regurgitation. Patient blood pressure was 120/80 mmHg, heart rate was 86 beats/minute, respiratory rate was 25 breaths/min and oxygen saturation without oxygen support was 94%. Relevant physical findings were diminished breath sounds on the left side, pansystolic murmur at the cardiac apex, and bilateral pretibial edema. The laboratory showed decreased hemoglobin (9 g/dl) and hematocrit (27%) and elevated serum glucose level (186 mg/dl). Chest radiograph revealed a large left sided pleural effusion (Figure



FIGURE 1: Chest radiography with massive left sided pleural effusion.

1). A small bore pleural catheter (8 French) was inserted in the left midaxillary line of the patient and within 120 minutes 2 lt exudative effusion was drained spontaneously with relief of symptoms. Cultures and cytology of the fluid were reported later as normal except the rare lymphocytes and polymorphonuclear leukocytes. According to the patient's surgical history, the effusion was thought to be due to post- coronary artery bypass graft surgery effusion.9 The patient complained of increasing nonproductive cough $2^{1/2}$ hour after the insertion of catheter. Repeated radiograph showed resolution of the effusion beside increased opacification of the left hemithorax. Pneumonia, cardiogenic edema and RPE were the presumed diagnoses. Re-examination of the chest revealed unilateral crackles with improved breath sounds on the left side. Patients new vital signs were similar to the firsts'. The electrocardiogram showed sinus tachycardia and cardiac auscultation was unchanged. An echocardiogram revealed normal left ventricular function and moderate mitral regurgitation. The repeated blood tests revealed elevated D dimer (1872 ng/dl) levels and mild elevation of erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) levels without fever or leukocytosis. There was no radiographic evidence of cardiac enlargement, jugular venous distension or other systemic venous distension signs suggesting cardiac failure or volume overload. The patient was transferred to the intensive care unit with the diagnose of RPE and diuretics and nasal oxygen treatment were administered. Subsequently thorax CT also confirmed the diagnosis (Figure 2). Clinical improvement was seen within 24 hours while gradual spontaneous radiological improvement lasted over next 3 days (Figure 3). The total volume of pleural fluid removed after the X-Ray improvement was 1.9 lt. The patient was discharged a week later, without further complications. Patient's informed consent was obtained.

DISCUSSION

Unilateral pulmonary edema is a rare disorder which can be misdiagnosed with other causes of unilateral infiltration.¹⁻³ Some of the etiologies previously described for UPE are RPE, obstruction of



FIGURE 2: Thorax CT with left sided pulmonary edema.



FIGURE 3: Chest radiography with reexpanded left lung and decreased pulmonary edema.

pulmonary vessels, left-to-right intracranial shunt, atrial tumor, acute mitral regurgitation and congestive heart failure.^{1-8,10} RPE was first described by Pinault in 1853 after the removal of pleural effusion.^{5,6} Nevertheless it was described more than 155 years ago, the precise incidence and the exact patophysiology of RPE remains unknown. Ischemia reperfusion injury, leukocytes sequestration and their injure in the microvessels might be some of the complex contributing factors of RPE.^{5,6,11} This theories were supported by the elevated ESR, CRP, D-dimer levels, and also by the leukocytes detected at pleural effusion in our patient. We could not find any human study about such laboratory alterations due to RPE in literature. Nine reports of RPE associated with the removal of pleural effusion (RPEPE) were reported after the first report.^{5,6,11,12} RPE has also been reported after lung reexpansion in pneumothorax, single lung ventilation, relief of bronchial obstruction and thoracoscopic resection of a mediastinal tumor.^{5-7,11-13} As seen in our patient, there were no reported cases of RPE in patient treated with tunneled pleural catheters for pleural effusion.¹¹⁻¹³

RPE has a wide range of clinical presentations. The symptoms start mostly within several hours after reexpansion.^{5,6} Chest discomfort and persistent cough continuing more than 20 min are leading symptoms. The others are dyspnea, tachypnea, respiratory failure and hemodynamic instability. Chest radiograph's or thorax CT's typical findings of RPE are new focal ground-glass opacities in vascular distribution, with no another clinical explanation^{3,5,6,11} while cardiogenic UPE findings, can be also interstitial, alveolar, right or left sided.^{1-4,8} The acute appearance after removal of pleural effusion and rapid clearing of the consolidation over several days without administration of antibiotics, absence of a history of fever or hemoptysis and no evidence of cardiac failure and volume overload, suggested the diagnosis of RPE in our patient.

The treatment of RPE is usually supportive; including oxygen, diuresis, steroids, inotropic agents and continuous positive airway pressure⁶ however a rapid response to diuretics is typical for cardiogenic UPE.^{1,2,8} The clinic course of RPE can be asymptomatic despite findings at chest radiography so treatment in such patients is unnecessary.^{5,6} Our patient's leading symptom was coughing. Oxygen and diuretics therapy decreased her symptoms.

Reported death rate ranged from 0% to 20% and preexisting disease was thought to be effective over mortality of RPE.^{5,11} Our patient showed a benign clinical course despite her comorbidities (coronary heart disease, moderate mitral regurgitation, type 2 diabetes mellitus, anemia). In the view of the reported RPE cases due to pneumothorax evacuation,^{5-8,11} we thought that the clinical presentation of RPEPE looked more benign. There

are no prospective studies examined the effect of preexisting diseases over RPEPE.

Althout some clinical features of RPE are defined, there are no definitive guidelines for prevention of RPE.^{6,11,12} It is thought that RPE is more likely to occur in a lung that has been collapsed for more than 3 days. The clinic and radiographic RPE seen after large volume thoracentesis was founded to be rare and independent of removed fluid volume but it is also recommended that if pleural pressure is not monitored no more than 1 lt of fluid should be removed. The persistent cough might be a sign of intrapleural pressure reduction. 5,13

In conclusion, RPE is an unusual, potentially significant cardiopulmonary complication occurring after reexpansion of the lung. RPE can be misdiagnosed with other causes of unilateral infiltration. Clinicians should remember the possibility of RPE before evacuation of pleural fluid and for early detection of RPE, should take chest radiograph routinely early after thoracentesis.

REFERENCES

- Lesieur O, Lorillard R, Thi HH, Dudeffant P, Ledain L. Unilateral pulmonary oedema complicating mitral regurgitation: diagnosis and demonstration by transoesophageal echocardiography. Intensive Care Med 2000;26(4): 466-70.
- Tomcsányi J, Arabadzisz H, Bózsik B. Images in cardiology: Left sided unilateral pulmonary oedema. Heart 2005;91(9):1157.
- Gluecker T, Capasso P, Schnyder P, Gudinchet F, Schaller MD, Revelly JP, et al. Clinical and radiologic features of pulmonary edema. Radiographics 1999;19(6):1507-31.
- Nitzan O, Saliba WR, Goldstein LH, Elias MS. Unilateral pulmonary edema: a rare presentation of congestive heart failure. Am J Med Sci 2004;327(6):362-4.

- Mahfood S, Hix WR, Aaron BL, Blaes P, Watson DC. Reexpansion pulmonary edema. Ann Thorac Surg 1988;45(3):340-5.
- Trachiotis GD, Vricella LA, Aaron BL, Hix WR. As originally published in 1988: Reexpansion pulmonary edema. Updated in 1997. Ann Thorac Surg 1997;63(4):1206-7.
- Muz MH, Özer B. [Pneumothorax]. Turkiye Klinikleri J Int Med Sci 2005;32(1):123-37.
- Baldessari EM, Mendez-Villarroel A, Mauriño E, Nachman FD, Veltri IA. Cardiogenic unilateral pulmonary edema: an unreported complication of a digestive endoscopic procedure. Congest Heart Fail 2009;15(5):252-3.
- 9. Light RW. Physiology of pleural fluid production and benign pleural effusion. In: Shields T,

Locicero III J, Rusch VW, eds. General Thorasic Surgery. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2005. p.806-17.

- Ware LB, Matthay MA. Clinical practice. Acute pulmonary edema. N Engl J Med 2005; 353(26):2788-96.
- Sohara Y. Reexpansion pulmonary edema. Ann Thorac Cardiovasc Surg 2008;14(4):205-9.
- Feller-Kopman D, Berkowitz D, Boiselle P, Ernst A. Large-volume thoracentesis and the risk of reexpansion pulmonary edema. Ann Thorac Surg 2007;84(5):1656-61.
- Echevarria C, Twomey D, Dunning J, Chanda B. Does re-expansion pulmonary oedema exist? Interact Cardiovasc Thorac Surg 2008; 7(3):485-9.