

Case of Cardiogenic Lobar Pulmonary Edema

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ABSTRACT

Introduction: Unilateral pulmonary edema can mimic as pneumonia and is often misdiagnosed.

Case description: We describe a case of cardiogenic unilateral lobar pulmonary edema in a HIV infected and diabetic patient without known history of cardiac disease.

Conclusion: Rarely, unilateral pulmonary edema can present as a lobar consolidation and be confused with pneumonia. High index of suspicion and response to treatment of heart failure can clinch the diagnosis.

Keywords: Edema, Pneumonia, Pulmonary edema.

Journal of Medical Academics (2019); 10.5005/jp-journals-10070-0032

INTRODUCTION

Pulmonary edema in majority of the cases is distributed bilaterally. When it occurs unilaterally or even solely in lobar distribution,¹ it is confused with more common causes of focal lung disease like pneumonia or mass lesion.² Unilateral pulmonary edema presents an interesting and confusing diagnostic problem.^{3,4} Unilateral pulmonary edema represented 2.1% of the cardiogenic pulmonary edema in a contemporary study conducted at cardiology center with 869 patients.⁵ Cardiogenic unilateral pulmonary edema usually involves the right upper lobe.⁶

Lack of awareness of this entity can lead to mistaken diagnosis and delay in definitive treatment. We present an unusual case of cardiogenic lobar pulmonary edema involving right lower lobe.

CASE DESCRIPTION

52-year-old male presented with exertional dyspnea with orthopnea and swelling of feet of one month and fever of 4 days duration. There was no history of chest pain, palpitations, syncope or hemoptysis. He was treated with diuretics and antibiotics over next 5 days in primary care. He presented after 15 days with recurrence of breathlessness and orthopnea. He had past history of type 2 diabetes mellitus (DM) on insulin therapy with poor compliance for 15 years and HIV infection since 20 years not on anti-retroviral treatment with CD4 count of 418 cells/cu mm. On examination, he was afebrile with sinus tachycardia (pulse 108/minute), BP 118/88 mm Hg and tachypnea (RR 28/minute). Arterial saturation by pulse oximetry (SPO₂) was 93% on room air. JVP was raised and he had pitting pedal edema. Fundus examination revealed moderate non proliferative diabetic retinopathy.

Auscultation of chest revealed crackles in right infra scapular and infra axillary area. On cardiovascular examination first heart sound was soft with normal second heart sound and pan systolic murmur at apex with radiation to left axilla suggestive of mitral regurgitation.

His routine investigations were as shown in table below. Chest radiograph showed homogenous opacity in right lower zone with cardiomegaly and minimal bilateral pleural effusion, predominantly on right side. Pleural fluid was transudative. Non contrast CT chest showed moderate pleural effusion (right > left) with thickening of right perihilar interlobular septa. 2D echocardiography revealed

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How to cite this article: Jha DK, Mukherjee PB, Jayaraman M, *et al.* Case of Cardiogenic Lobar Pulmonary Edema. *J Med Acad* 2019;2(1):30–32.

Source of support: Nil

Conflict of interest: None

evidence of a dilated cardiomegaly with LV dysfunction and moderate mitral regurgitation. Gated SPECT confirmed the finding as non-ischemic cardiomyopathy. He had raised NT pro BNP confirming cardiac failure as the underlying condition.

Patient was started on treatment with diuretics and multiple SC insulin injection for glycemic control. Patient responded to therapy and the opacity on chest X-ray cleared in 48 hours.

In view of his lobar pulmonary edema, which rapidly cleared with diuretics, a diagnosis congestive heart failure with moderate mitral regurgitation was made. On follow up, he had marked improvement in his symptoms and effort tolerance with improved LV ejection fraction.

DISCUSSION

Pulmonary edema is a pathological condition in which extravascular fluid accumulates in the lungs. Characteristically intra alveolar pulmonary edema appears as homogeneous, symmetrical density involving the central fields of both lung, giving rise to “batwing” shadow.⁷

Unilateral pulmonary edema (UPE) can be associated with left heart failure.⁸ It is usually seen in the right lung and this is thought to be due to the difference in lymphatic drainage patterns of the two lungs. Lymph from the right lung drains into the right bronchomediastinal trunk which forms the right lymphatic trunk, whereas lymph from the left lung drains into the thoracic duct. Therefore fluids from the left lung drain easily through the larger thoracic duct whereas excess fluid can easily exceed the capacity of the

broncho-mediastinal trunk leading to higher chance of right sided pulmonary edema. In a recent study by Attias et al studied 869 individuals with cardiogenic pulmonary edema and found 2.1% having unilateral pulmonary edema. UPE was right-sided in 89% of cases, and left-sided UPE was infrequent⁹ and represented only 0.2% of all cardiogenic pulmonary edema. Schnyder and coworkers reported a 9% prevalence of predominantly right upper lobe pulmonary edema in cases of severe mitral valve regurgitation.¹⁰ Most of the cases in above study had severe MR and predominantly right-sided UPE.

In severe mitral regurgitation (MR), there is retrograde blood flow directed towards the posterior left atrial wall. Reversal of blood flow in both the pulmonary venous systems causes the bilateral pulmonary edema seen in severe MR. Rarely, the retrograde blood flow is selectively directed towards the right pulmonary venous system. The regurgitant jet usually targets the right superior pulmonary vein, leading to right upper lobe edema which is best visualized by transesophageal echocardiography.¹¹ There are very few reported cases in whom the regurgitant jet targets the right inferior pulmonary vein.¹² Our patient showed edema of the right lower lobe, caused by regurgitation straight into the orifice of the right inferior pulmonary vein.

SUMMARY

Patients having mitral regurgitation may present with UPE. Consideration should be given to cardiogenic causes of unilateral infiltrates on chest X-ray, particularly when the patient's clinical condition is worsening despite what is considered aggressive appropriate treatment for pneumonia.

INVESTIGATIONS

Pleural Fluid Analysis

Cytology—appearance-turbid, WBC-750, predominant cells-lymphocytes, Biochemistry—protein –1.5 (g/dL), LDH-95 (U/L), Glu-259 (mg/dL), ADA-23 U/L, Gram/ZN staining was negative. Sputum for AFB-negative twice 24 hours urinary protein-308 mg, HbsAg/Anti HCV/VDRL—negative.

Chest X-Ray

Figure 1.

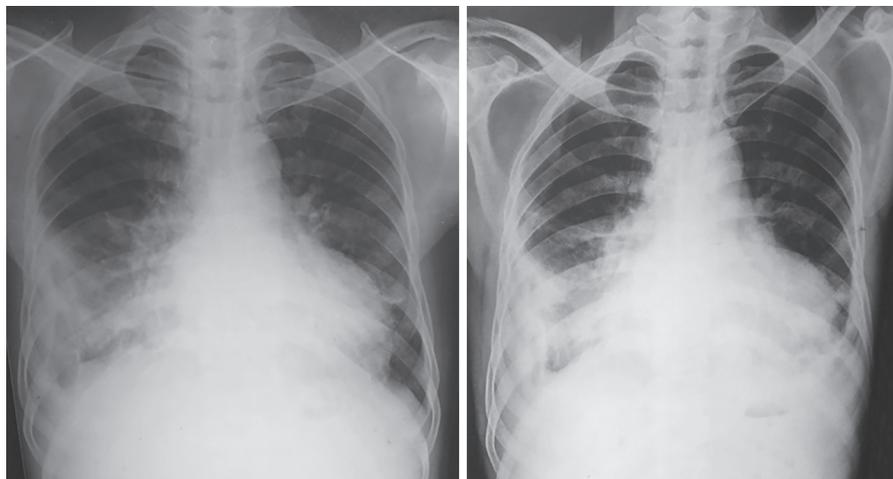


Fig. 1: Chest X-ray

NCCT Chest (20/2/14)

In a K/C/O dilated cardiomyopathy, bilateral moderate pleural effusion (right > left) with thickening of left perihilar interlobular septa suggestive of volume overload.

Laboratory Investigations

Table 1.

Table 1: Investigations

Routine	Values
Hb (g/dL)	13.2
TLC (10^9 /L)	5720
DLC (10^9 /L)	P 58% L 32%
Platelets (10^9 /L)	2.62
Urea (mg/dL)	32
Creatinine (mg/dL)	1.4
Na (meq/dL)	
K (meq/dL)	
Ca (mg/dL)	9.6
PO ₄ (mg/dL)	4.2
Bilirubin (mg/dL) total	0.4
Bilirubin (mg/dL) dir	0.2
AST (U/L)	30
ALT (U/L)	81
ALP (U/L)	104
LDH (U/L)	245
Total protein (g/dL)	4.9
Alb/glob (g/dL)	3.7/1.2
PT	12.5
aPTT	28
INR	1.03
Blood sugar (mg/dL) F/PP	220/345
HbA1c	13.3%
Lipid profile (mg/dL)	
Total chol	183
TG	52
LDL	102
HDL	50
NT-pro BNP (pg/mL)	2102

2D ECHO

EF: 25–30%, LV dilated with global hypokinesia, moderate MR, Imp-dilated cardiomyopathy with severe LV dysfunction.

USG-KUB

USG-KUB- (19/02/14)—right kidney-10.8 cm, left kidney-10 cm.
Liver-12.0 cm, coarse echotexture with B/L pleural effusion.

Myocardial Perfusion Scan-rest Viability Study- (22/2/14) (Fig. 2)

Gated SPECT shows-LVEF <25%, global hypokinesia

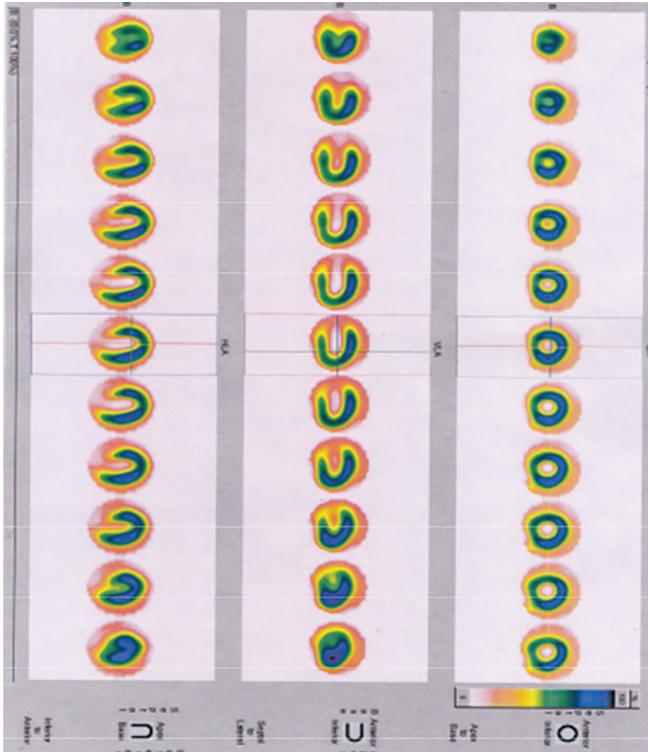


Fig. 2: Thallium scan

Impression

Viable myocardium in all three vascular territories, with features of severe LV systolic dysfunction.

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