Case Report

Unilateral pulmonary oedema - A case report

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Abstract

Pulmonary oedema usually occurs bilaterally in a symmetrical pattern, but unilateral lobar pulmonary edema (UPE) can also be encountered in clinical practice, that is often misdiagnosed at the initial stages. Acute unilateral pulmonary edema is an unusual clinical presentation occurring in many rare causes. But the commonest is the presence of mitral regurgitation (MR). Due to its rare presentation, a high index of suspicion is required, and correct management is necessary to reduce the morbidity and mortality. We describe a case report of unilateral pulmonary oedema in a patient of mitral regurgitation.

Key words

Unilateral Pulmonary Edema, Mitral Regurgitation.

Introduction

Acute unilateral pulmonary edema is an unusual clinical condition. Most cases reported in the literature occur at the upper right side and are caused by severe mitral regurgitation (MR) [1-3].

A diuretic responsive lung opacification is most likely to be pulmonary oedema. A common presentation such as pulmonary oedema is normally bilateral. However, rarely, it may be unilateral. Unilateral pulmonary oedema is most commonly due to severe mitral regurgitation. Sometimes it may occur in acute coronary syndromes also [4]. Atypical accumulation of fluid in the lung can produce unilateral lobar pulmonary edema. Two such Mechanisms have been attributed to various causes [5].

Cardiogenic unilateral pulmonary edema (UPE) is a rare clinical entity that presents diagnostic challenges and is associated with an independent increased risk of mortality. That is why the prompt recognition is necessary to avoid delay in treatment [6]. Most cases of UPE reported in the literature occur on the upper right side and are

caused by severe mitral regurgitation (MR) [7]. We herewith report a case of right sided unilateral pulmonary oedema due to mitral regurgitation. The case was successfully treated with decongestive therapy which included loop diuretics.

Case report

A 32-year-old male patient, presented to the emergency department with sudden onset of breathlessness, palpitations and chest pain. He was a known case of Chronic Rheumatic Heart Disease. Not a known diabetic or hypertensive. No history of CAD, smoking, any lung ailment, respiratory infection, TB or COPD. There were no similar complaints in the past.

On examination

Patient was conscious, coherent, answering questions properly. He was moderately built and nourished, no pallor, no icterus, no clubbing, no koilonychias, and no enlarged lymph nodes. Temperature was 95 ^{0}F, PR 120/min regular, BP 110/70 mmHg, Respiratory Rate 20 breaths per minute. Cardiovascular system examination - JVP – not raised, revealed cardiomegaly left ventricular type, forcible, S1 and S2 heard. There was mid diastolic murmur heard at the apex and there was a pansystolic murmur heard at the apex and radiating to axilla. Respiratory system examination - There were fine crepitations on right lung. Other systems, like CNS, GIT, hemopoitic, renal systems were normal.

Lab Investigations were as follow: Hb: 11gm%, WBC: 6.500 percumm, N - 75, L - 23, B - 0, E - 2, M - 0; Platelets: 2.50 lac/cumm, ESR: 06 mm, Bleeding Time - 4 mts, Clotting Time – 6 mts, Prothrombin Time- 12 seconds, RBS: 124 mg/dl, Complete Urine Examination: Normal, Serum Electrolytes: Serum sodium-128 mmol/L, Serum Potassium 6.9 mmol/L, Blood Urea – 20 mg/L, Serum Creatinine - 0.8 mg/dl, Sputum for c/s – Sterile, Sputum for Fungus – Sterile, Sputum for AFB, Sputum for CB NAT – negative, ASO Titre – 200 Todds units, Blood Culture – no organisms grown. The chest X-ray showed a pattern of acute pulmonary edema, predominantly right-sided, which resolved after diuretic treatment (**Figure – 1 to 4**). On Day – 4 (**Figure - 4**), the lungs became clear. The electrocardiogram revealed sinus rhythm left atrial overload and left ventricular hypertrophy. 2 D - Echocardiography demonstrated a moderately impaired left ventricular systolic function with an estimated ejection fraction of 50% and moderate mitral regurgitation with mild mitral stenosis and no thrombus in ventricular cavity. Spo2 - 96% at room oxygen.

Figure – 1: Day 1.

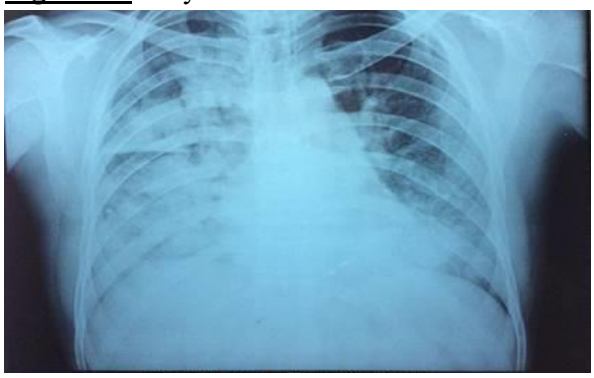


Figure – 2: Day 2.

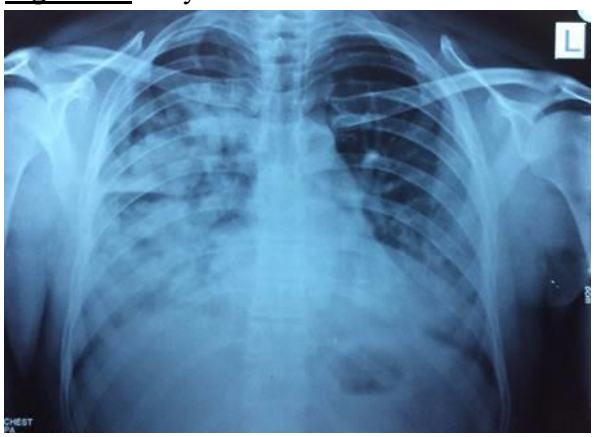


Figure – 3: Day 3.

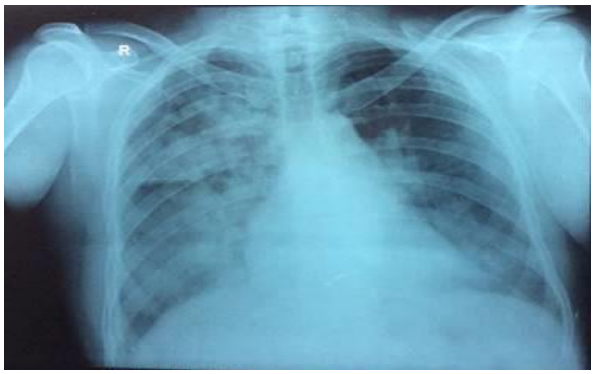
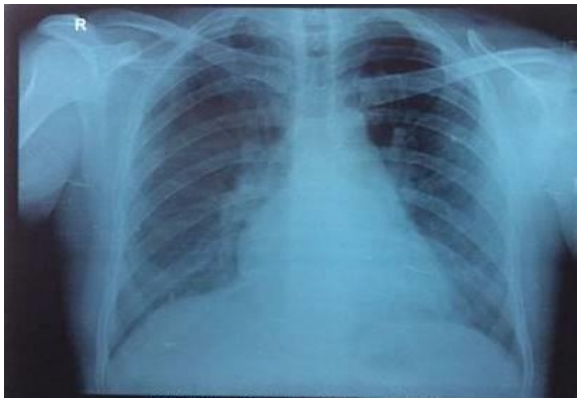


Figure – 4: Day 4.



A diagnosis of Chronic Rheumatic Heart Disease, with Mild Mitral Stenosis and Moderate Mitral Regurgitation, Normal Sinus Rhythm, with Acute Unilateral Right Side Pulmonary Oedema, with No E/O Thromboembolism, No E/O Infective Endocarditis was made.

Treatment given

1). Inj. Furesomide – 100 mg stat
2). Tab. Furosemide - 40 mg daily
3). Tab. Digoxin – 0.25 mg daily 5/7 in a week
4). Liq. Potklor – 1 tsf tid
5). Salt restricted diet
6). Inj. Ampicillin 500 mg qid
7). Inj. Amikacin 500 mg iv bd
8). Inj. Flagyl 500 mg iv tid

Discussion

This was first presentation of underlying cardiac disease in our patient and it was difficult to differentiate from pneumonia. We concluded that unilateral pulmonary edema can mimic as pneumonia. Clinician should be aware of differential diagnosis of pulmonary oedema, otherwise it can lead to unnecessary investigation and delay in starting definitive treatment [5].

Unilateral pulmonary edema represents only 2% of cardiogenic pulmonary edema with predilection for the right upper lobe and is strongly associated with severe mitral regurgitation [6, 8]. It is hypothesized that the regurgitation jet is directed towards the right superior pulmonary vein thus preferentially increasing the hydrostatic pressure in the right upper lobe [9].

Pathology

Etiology

1). Unilateral edema with ipsilateral pathology [10]

- severe mitral valve regurgitation
- positioning
- re-expansion pulmonary edema
- pulmonary vein occlusion
- congenital or surgical right-to-left shunt (e.g. Blalock-Taussig shunt)

2).Unilateral edema with perfusion abnormality in the contralateral lung [10]

- unilateral pulmonary embolism
- unilateral hypoplasia of pulmonary artery
- Swyer-James syndrome
- unilateral emphysema/bullae

The differential diagnosis in cases of acute unilateral pulmonary edema should include the other causes of alveolar congestion: unilateral pneumonia, bronchial aspiration, or alveolar hemorrhaging. Most of the cases of acute unilateral pulmonary edema have resulted from severe eccentric MR. A mitral regurgitation jet affecting a pulmonary vein, predominantly on the upper right [1], can lead to a larger increase in mean capillary pressure in the right side and consequently, a greater degree of right acute pulmonary edema [2, 11]. On rare occasions, the asymmetrical increase in capillary pressures is caused by compression of a pulmonary vein outlet by a myxoma or atrial wall hematoma [12]. Perfusion imbalance between the lungs is another source of acute unilateral pulmonary edema. The degree of edema is proportional to the degree of perfusion; hence, in cases of congenital hypoplasia or pulmonary artery agenesis, the less perfused lobes will present a lower degree of edema [13].

Pulmonary edema is rarely unilateral, but may cause confusion and presents diagnostic challenges. The development of pulmonary

edema is the result of complex mechanisms [14]. The force, which tends to push fluid out of the capillaries, is the capillary hydrostatic pressure less the hydrostatic pressure of the interstitial fluid. The force tending to keep fluid in the capillaries is the osmotic pressure of blood proteins less that of the proteins in the interstitial fluid (Starling forces). When the balance of these factors is altered, fluid passes from the pulmonary capillaries to the interstitial spaces – and, when the capacity of lymphatics in the lung interstitium is exceeded, to the alveolar spaces. The pressure of the gas in the alveoli and the presence of surfactant also influence this movement of fluid. Unilateral pulmonary edema can occur when local forces predominate (**Table - 1**) or when the pulmonary vasculature or pulmonary parenchyma is abnormal (**Table - 2**). In all conditions causing contralateral pulmonary edema, there is under perfusion of the abnormal lung, so pulmonary edema affects only the lung with normal vasculature. Another important cause is neurogenic pulmonary edema. Possible mechanism includes increase intracranial pressure and subsequent vascular congestion due to alterations in the sympathetic pathways, resulting in alterations in intravascular hydrostatic pressures as well as myocardial rate and contractility. Unilateral pulmonary edema has been described also in patient with acute multiple sclerosis exacerbation [15] and end stage renal failure [16].

Table - 1: Ipsilateral pulmonary edema.

Condition	**Mechanism**
1. Papillary dysfunction, Mitral valve regurgitation [17, 18]	A retrograde flow of blood directed across the left atrium toward the orifices of the right upper lobe pulmonary veins resulted in a focal increase in pulmonary venous pressure.
2. Dependent lung of unconscious or ventilated patient whose posture is not frequently altered	Contributed by gravity
3. Rapid removal of large amount of fluid or air by thoracocentesis [19, 20]	Swift re-expansion of the decompressed lung causes a change in the hydrostatic pressure in the pulmonary capillaries. The production of surfactant may also have been inhibited while lung was collapsed
4. Aspiration of gastric content	Damage to the surfactant system and irritation of the pulmonary capillaries
5. Unilateral infusion of hypotonic saline through a catheter misplaced in pulmonary artery.	Local decrease in colloid osmotic pressure
6. Bronchial obstruction	Hypoxia damages those alveolar cells which produce surfactant

Table - 2: Contralateral pulmonary edema [21].

Condition	**Mechanism**
- Hypoplasia of the pulmonary artery - Unilateral pulmonary thromboembolism - anastomosis of the left pulmonary artery to descending aorta(Potts procedure)	-Blood flow in the pulmonary capillaries may be reduced
-Primary emphysema -Compensatory emphysema after lobectomy	Pulmonary capillaries may be reduced or damaged.

Acute cardiogenic pulmonary edema is a critical condition associated with high mortality, and may be caused by a variety of cardiac diseases, including coronary artery disease. The usual radiographic finding in acute cardiogenic pulmonary edema is bilateral symmetrical opacities in the central zones of the lungs, resulting in the classic "butterfly shadow" [23, 24]. UPE is a rare entity that can be mistaken for other causes of unilateral infiltrate on chest radiography, especially pneumonia. UPE has been reported after congestive heart failure, prolonged rest on one side in patients with cardiac decompensation or receiving large amounts of cases of rapid expansion of the lungs after pleural effusion, and pneumothorax [18, 20, 25]. It is also seen in a normal lung in patients with unilateral pulmonary disease such as MacLeod syndrome and unilateral pulmonary artery hypoplasia or agenesis, pulmonary artery compression from aortic dissection or LV pseudo aneurysm, and pulmonary venous obstruction from mediastinal fibrosis [22]. However, it is mainly reported in association with severe MR [18]. Most cases of UPE associated with left-sided heart failure affect the right lung [24]. A possible explanation is the poorer lymphatic drainage of the right lung by the small-caliber right bronchomediastinal trunk in comparison with that of the left lung by the large-caliber thoracic duct. Another explanation relates to the left-sided cardiac enlargement that develops in most patients with heart failure and that may physically impede blood flow in the left pulmonary artery, thereby reducing capillary volume. However, severe MR remains the main cause of UPE [18]. An MR jet affecting predominantly the upper right pulmonary vein, can lead to a larger increase in mean capillary pressure on the right side and, consequently, a greater degree of right acute pulmonary edema. The main mechanism of MR in UPE is mitral leaflet prolapse, but functional MR may also be involved [18, 20].

Conclusion

In our patient, the initial clinical and radiological diagnosis was pneumonia; however the presence of mitral regurgitation and rapid radiological improvement after diuretics raised suspicion of unilateral pulmonary edema. Unilateral lobar pulmonary edema is rare but may present as first manifestation of underlying cardiac disease as in our case. The clinician should be aware that pulmonary edema need not necessarily affect both lungs, otherwise diagnosis may be mistaken and treatment delayed.

Most cases of UPE associated with left heart failure affect the right lung. A possible explanation is the poorer lymphatic drainage of the right lung by the small-caliber right bronchomediastinal trunk in comparison with that of the left lung by the large caliber thoracic duct [27]. Another explanation relates to the left sided cardiac enlargement that develops in most patients with heart failure and that may physically impede blood flow in the left pulmonary artery, thereby reducing capillary volume [28]. Nevertheless, severe MR remains the main cause of UPE. An MR jet affecting predominantly the upper right pulmonary vein, can lead to a larger increase in mean capillary pressure on the right side and, consequently, a greater degree of right acute pulmonary edema. The main mechanism of MR in UPE is mitral leaflet prolapse, but functional MR may also be involved [6, 26].

A diuretic responsive lung opacification is most likely to be pulmonary oedema. A common presentation such as pulmonary oedema is normally bilateral. However, rarely, it may be unilateral. Unilateral pulmonary oedema is most commonly due to severe mitral regurgitation. Nevertheless, acute coronary syndrome may present this way. UPE is a completely reversible condition with good patient outcome if it is suspected early and treated early.

References

1. Brander L, Kloeter U, Henzen C, Briner V, Stulz P. Right-sided pulmonary oedema. Lancet, 1999; 354: 1440.
2. Roach JM, Stajduhar KC, Torrington KG. Right upper lobe pulmonary oedema caused by acute mitral regurgitation. Diagnosis by transesophageal echocardiography. Chest, 1993; 103: 1286-8.
3. Lesieur O, Lorillard R, Thi HH, Dudeffant P, Ledain L. Unilateral pulmonary oedema complicating mitral regurgitation: diagnosis and demonstration by transoesophageal echocardiography. Intens Care Med., 2000; 26: 466-70.
4. Bhargava R, Cowell R. Unilateral pulmonary oedema. BMJ Case Rep., 2019; 12: e230596. doi:10.1136/bcr-2019- 230596
5. Garvi J Pandya, Amartya Mukhopadhyay, Chua Ai Ping, Tow Keang Lim. Unilateral Lobar Pulmonary Edema. JAPI, 2012; 60: 60 – 62.
6. Attias D, Mansencal N, Auvert B, Vieillard-Baron A, Delos A, Lacombe P, N'Guetta R, Jardin F, Dubourg O. Prevalence, characteristics, and outcomes of patients presenting with cardiogenic unilateral pulmonary edema. Circulation, 2010; 122: 1109-15.
7. Jeong Hun Shin, Seok Hwan Kim, Jinkyu Park, Young-Hyo Lim, Hwan-Cheol Park, Sung Il Choi, Jinho Shin, Kyung-Soo Kim, Soon-Gil Kim, Mun K. Hong, Jae Ung Lee. Unilateral Pulmonary Edema: A Rare Initial Presentation of Cardiogenic Shock due to Acute Myocardial Infarction. J Korean Med Sci., 2012; 27: 211-214.
8. Myrianthefs P, Markou N, Gregorakos L. Rare roentgenologic manifestations of pulmonary edema. Curr Opin Crit Care, 2011; 17(5): 449-53.
9. Miyatake K, Nimura Y, Sakakibara H, et al. Localisation and direction of mitral regurgitant flow in mitral orifice studied with combined use of ultrasonic pulsed Doppler technique and two dimensional echocardiography. Br Heart J., 1982; 48(5): 449-58.
10. Hacking C, Hsu C, et al. Unilateral pulmonary oedema. In: Radiopaedia. 2015. [online] http://radiopaedia.org/articles/unilateral-pulmonary-oedema
11. Lesieur O, Lorillard R, Thi HH, Dudeffant P, Ledain L. Unilateral pulmonary oedema complicating mitral regurgitation: diagnosis and demonstration by transoesophageal echocardiography. Intens Care Med., 2000; 26: 466-70.
12. Álvarez J, Rubio A, Mora MD, Fernández Madero G, Vivancos R, Malpartida F. Hematoma intramural auricular izquierdo diagnosticado por resonancia magnética nuclear. Rev Esp Cardiol., 2002; 55: 872-4.
13. Rios B, Driscoll DJ, McNamara DG. High-altitude pulmonary oedema with absent right pulmonary artery. Pediatrics, 1985; 75: 314-7.
14. Robin ED, Cross CE, Zelis R. Pulmonary oedema New England Journal of Medicine, 1973; 288: 239-46 and 292 – 304.
15. Makaryus JN, Kapphahn S, Makaryus AN. Unilateral neurogenic pulmonary oedema and severe left ventricular dysfunction secondary to acute multiple sclerosis exacerbation. Heart Lung Circ., 2009; 18: 155-8.
16. Agarwal R, Aggarwal AN, Gupta D. Other causes of unilateral pulmonary edema. Am J Emerg Med., 2007; 25: 129-31.
17. Osmundson PJ, Callahan JA, Edwards JE. Mitral insufficiency from ruptured chordae tendineae simulating aortic stenosis. Proc Staff Meet Mayo Clin., 1958; 33: 235-44.
18. Legriel S, Tremey B, Mentec H. Unilateral pulmonary edema related to massive mitral insufficiency. American

Journal of Emergency Medicine, 2006; 24: 372.

19. Trapnell D H, Thurston J G. Unilateral pulmonary oedema after pleural aspiration. Lancet, 1970; 1: 1367-9.
20. Childress ME, Moy G, Mottram M. Unilateral pulmonary edema resulting from treatment of spontaneous pneumothorax. Am Rev Respir Dis., 1971; 104: 119-21.
21. Calenoff L, Kruglik GD, Woodruff A. Unilateral pulmonary edema. Radiology, 1978; 126: 19-24.
22. Shin JH, Kim SH, Park J, et al. Unilateral pulmonary edema: a rare initial presentation of cardiogenic shock due to acute myocardial infarction. J Korean Med Sci., 2012; 27(2): 211–4. https://doi.org/10.3346/jkms.2012.27.2.211.
23. Pandya GJ, Mukhopadhyay A, Ping CA, Lim TK. Unilateral Lobar Pulmonary Edema. JAPI, 2012; 60: 60–62.
24. Nessa CB, Rigler LG. The roentgenological manifestations of pulmonary edema. Radiology, 1941; 37: 35–45.
25. Nitzan O, Saliba WR, Goldstein LH, Elias MS. Unilateral pulmonary edema: a rare presentation of congestive heart failure. Am J Med Sci., 2004 Jun; 327(6): 362–4.
26. Peña C, Jaquet M, Salgado J, Pubul V, Ruibal A, Vázquez B. Asymmetric pulmonary perfusion causing unilateral pulmonary edema as a complication of acute myocardial infarction. Rev Esp Cardiol., 2005; 58: 875-7.
27. Akiyama K, Suetsugu F, Hidai T, Shimamoto K, Takahashi S. Left-sided unilateral pulmonary edema in postinfarction ventricular septal rupture. Chest, 1994; 105: 1264-5.
28. Schnyder PA, Sarraj AM, Duvoisin BE, Kapenberger L, Landry MJ. Pulmonary edema associated with mitral regurgitation: prevalence of predominant involvement of the right upper lobe. AJR Am J Roentgenol., 1993; 161: 33-6.