LETTER TO THE EDITOR

Reexpansion pulmonary edema after therapeutic thoracentesis

Olívia Meira Dias, Lisete Ribeiro Teixeira, Francisco S. Vargas

Pulmonology Section, Heart Institute (Incor), Hospital das Clinicas da Faculdade de Medicina da Universidade de São Paulo - São Paulo/SP, Brazil.

Email: meiradias@yahoo.com.br
Tel.: 55 11 3069-5000

Reexpansion pulmonary edema is a rare complication resulting from rapid emptying of air or liquid from the pleural cavity performed by either thoracentesis or chest drainage. Despite being infrequent, mortality may occur in up to 20% of cases and is attributed to the abrupt reduction in pleural pressure, especially as a result of extensive pneumothorax drainage or when there is long-term pulmonary collapse.[1,2]

We report the case of a young patient who experienced intense chest discomfort during thoracentesis for relief of dyspnea.

CASE DESCRIPTION

A 40-year-old female from São Paulo (Brazil) was admitted for investigation of dyspnea associated with vespertine fever, loss of weight (4 kg), and arthralgia for 1 month; she denied other symptoms or comorbidities. Upon physical examination, only the absence of thoracic–vocal trill and vesicular breath sounds at the base of the mid-third of the left hemithorax were noteworthy. Breathing ambient air, the peripheral oxygen saturation was 88%.

Radiographic assessment revealed a large left pleural effusion, with no evidence of mediastinal or pulmonary parenchymal abnormalities. The patient underwent thoracentesis (diagnostic and therapeutic) and biopsy of the parietal pleura with a Cope needle. One liter of citrine yellow pleural fluid was removed, until thoracentesis had to be interrupted because of chest pain. With persisting pain, a chest computed tomography scan was taken, which showed a persistent mild hydro-pneumothorax on the left hemithorax. No mediastinal abnormalities were noted. The high-resolution scans showed ipsilateral airspace opacities in the previously collapsed lung, which consisted of ill-defined centrilobular micronodules, thickening of interlobular and intralobular septa, and superimposed patchy ground-glass opacities predominantly in the lingula and left inferior lobe with a peripheral and geographic distribution rather than a gravity-dependent distribution. No abnormalities were noted in the right lung (Figure 1).

The patient was maintained on spontaneous respiration with nasal oxygen and, from the second post-puncture day, she displayed spontaneous and progressive improvement in the discomfort and the oxygen saturation level.

The pleural fluid analysis showed a lymphocytic exudate with glucose level of 106 mg/dL, adenosine deaminase of 56 U/L, and negative cultures for pathogens; the oncotic cytology was negative and the pleural biopsy showed chronic non-specific pleuritis. The laboratory examinations and the pleural biopsy established the diagnosis of systemic lupus erythematous: hand arthritis, pleuritis, and positive anti-nuclear (1:1280) and anti-Smith (anti-Sm) antibodies.

On the fourth day, before hospital discharge, a new chest tomography scan showed a decrease in the pneumothorax and ground-glass opacities. The involution of the parenchymal abnormalities allowed us to consider the clinical picture consistent with post-thoracentesis reexpansion edema (Figure 2).

DISCUSSION

Pulmonary reexpansion edema may be considered an iatrogenic complication due to rapid emptying of the pleural cavity. The incidence referred is less than 1%, and mortality can reach up to 20%.[1,2] The greatest risk affects young patients with extensive pneumothoraces or pulmonary collapses of more than 7 days' duration. Included in this category are patients with large pleural effusions in which the volume of fluid removed exceeds 3 L.[1]

The pathophysiological mechanisms are not yet totally clarified. The main hypothesis considers the existence, after pulmonary reexpansion, of an acute inflammatory response that includes damage to the alveolar–capillary membrane and changes in the pulmonary lymphatic vessels and in the surfactant resulting from various factors, including reperfusion of a previously collapsed lung.[2] Experimental study has shown that endothelial vascular damage after hypoxic vasoconstriction induces an increment in the expression of the inflammatory mediators tumor necrosis factor (TNF)α and interleukin (IL)-1β.[3] In this way, the association of local and systemic factors explains the bilateral cases of edema after unilateral manipulation.

Recently, it was investigated whether the ventricular complacency and the pulmonary capillary pressure can influence the redistribution of the extravascular lung fluid.[4] Nevertheless, even with normal cardiac function and occlusion pressures of lung capillaries, reexpansion edema can occur.[5] Posteriorly, Sue et al.[6] concluded that reexpansion edema after thoracostomy or thoracentesis is essentially hydrostatic and not a consequence of increased permeability of the alveolar–capillary barrier. It should be emphasized that these patients presented no dysfunction of left chambers or signs of hypervolemia.

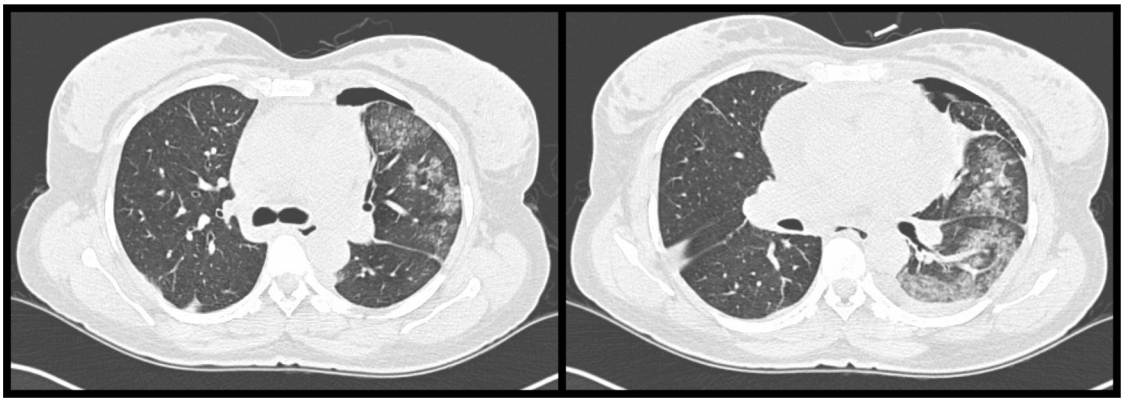

Figure 1 - Computed tomography scans immediately after thoracocentesis showing mild hydro-pneumothorax, ground-glass opacities predominantly in the lingula and left inferior lobe, centrilobular micronodules, and thickening of interlobular septa.

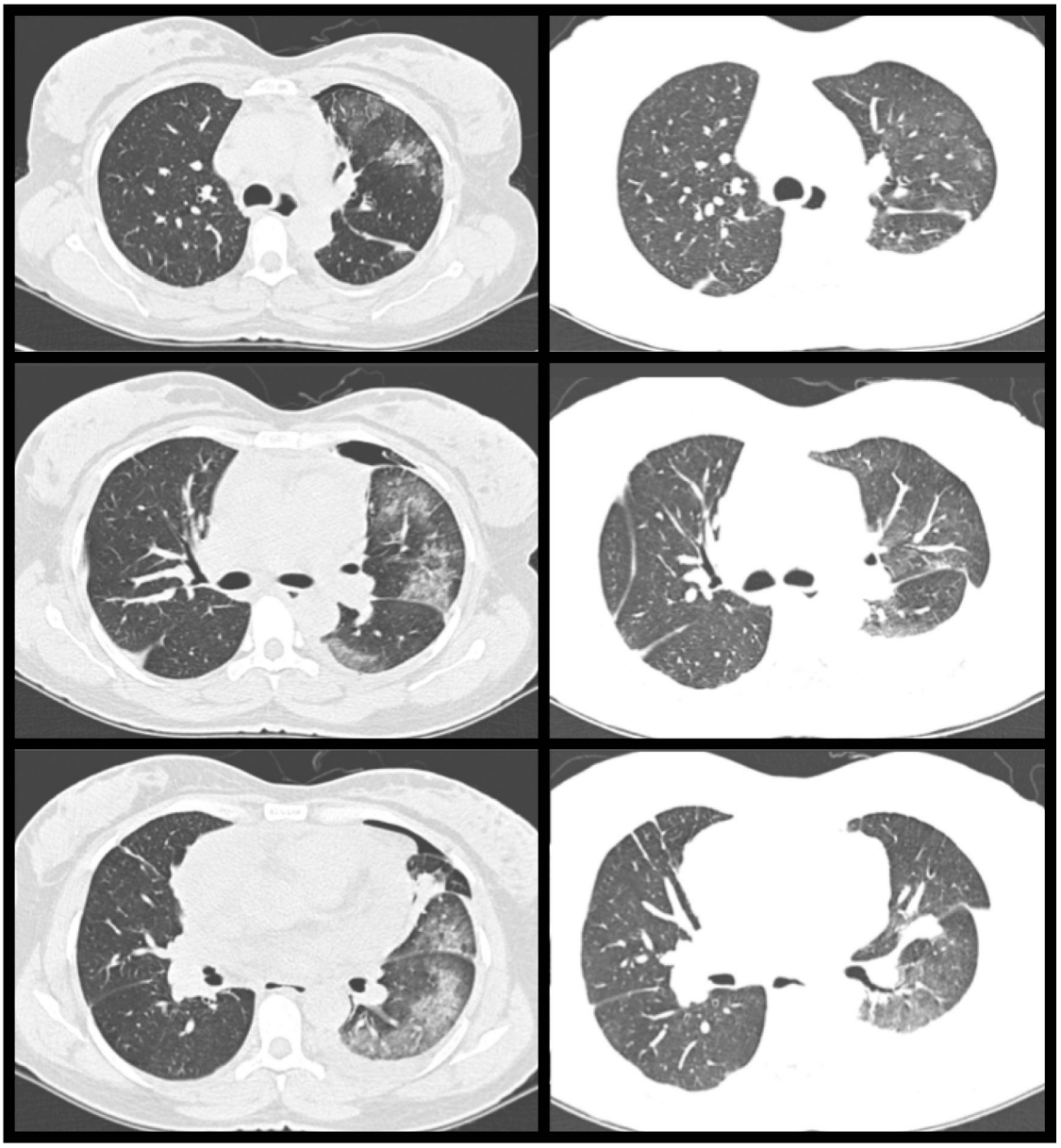

Figure 2 - Comparative analysis from computed tomography scans. On the left, images immediately after thoracocentesis; on the right, 4 days later. Although a discrete pleural effusion remains on the left, the images show a decrease in the pneumothorax and ground-glass opacities, consistent with post-thoracentesis reexpansion edema.

Therefore, current knowledge imputes to the hydrostatic forces the onset of the edema after acute reexpansion of the lungs. However, the concomitance of a variable degree of stress to pulmonary capillaries presupposes damage to the basement membrane with a consequent production of cytokines by the vascular endothelium, especially selectin, generating increased protein permeability.

The clinical picture varies according to the extent of the edema, but about 64% of patients are symptomatic during the first hour post-puncture. Suggestive symptoms are persistent cough (generally for more than 20 min and regardless of the presence of pinkish sputum), tachycardia, tachypnea, hypoxemia, and hemodynamic instability.[7]

The radiographic diagnosis includes the presence of opacities in the previously collapsed lung, which progresses over the 2 days following thoracentesis and then rapidly reverts. Apparently, the tomography pattern of ground-glass is observed in all patients. Characteristically, the lesions tend to be peripheral, seen preferentially in gravity-dependent areas. Additional findings include thickening of interlobular septa, peribronchovascular band-like thickenings, and poorly defined centrilobular micronodules. Pleural effusion is not an usual finding.[8]

Treatment consists of support measures. Lateral decubitus on the affected side is recommended which, in unilateral cases, contributes to reducing the pulmonary shunt and improving oxygenation. Noninvasive ventilation should be considered as good results are obtained, even in serious cases.[9] In patients needing orotracheal intubation and mechanical ventilation, positive pressure improves symptoms after 24–48 h. Asynchronous ventilation is rarely necessary.

The usefulness of manometry in measuring pleural pressures during thoracentesis remains controversial, even when large volumes are drained. Based on animal studies,[10,11] it has been inferred that the procedure should be interrupted if the pleural pressure drops below -20 cmH_20. However, this value is considered conservative, as healthy individuals can spontaneously generate more negative pleural pressures with no clinical repercussion.[12] At our institution, pleural manometry is used in selected cases; it is not adopted routinely.

Independent of pressure control, there is no consensus as to the maximal volume to be drained in a single thoracentesis procedure. Feller-Kopman et al.[12] reported that, of 185 patients submitted to thoracentesis, only one (0.5%) experienced edema with clinical manifestations, and four (2.2%) developed compatible radiographic abnormalities. In this group, the preventive strategy of removing up to 1 L did not prove to be protective.

In this way, it is currently recognized that the amount of fluid drained, the pleural pressure, and the elastance are not predictors of the appearance of edema.[11,12] We point out that there are patients who routinely need drainage of greater volumes for symptomatic relief of dyspnea, and that there is not yet a defined limiting value. This fact is confirmed in the clinical case described, as even the removal of a moderate volume (1000 mL) provoked reexpansion edema.

In conclusion, the strategy suggested and applied at our institution by the Pleura Group is to remove, at the most, 1800 mL (without the use of pleural pressure measurements). The procedure should be interrupted if there is spontaneous cessation of fluid drainage or if the patient experiences chest discomfort or persistent cough. These symptoms have been recognized as correlating with a reduction in pleural pressure and are indicative of interruption of the procedure.[13]

REFERENCES

1. Echevarria C, Twomey D, Dunning J, Chanda B. Does re-expansion pulmonary oedema exist? Interact Cardivoasc Thorac Surg. 2008;7:485–89, doi: 10.1510/icvts.2008.178087.
2. Sherman SC. Reexpansion pulmonary edema: a case report and a review of the current literature. J Emerg Med. 2003;24:23–7, doi: 10.1016/S0736-4679(02)00663-7.
3. Funakoshi T, Ishibe Y, Okazaki N, Miura K, Liu R, Nagai S, et al. Effect of re-expansion after short-period lung collapse on pulmonary capillary permeability and pro-inflammatory cytokine gene expression in isolated rabbit lungs. Br J Anaesth. 2004;92:558–63, doi: 10.1093/bja/aeh101.
4. Chowdhary M, Peng EWR, Sarkar PK. The risk of fatal re-expansion pulmonary oedema in poor left ventricular reserve. Interact Cardiovasc Thorac Surg. 2009;9:350–51, doi: 10.1510/icvts.2008.201640.
5. Jaber S, Perrigault PF, Souche B, Pouzeratte Y. Re-expansion pulmonary edema with normal pulmonary artery occlusion pressure during liver transplantation. Intensive Care Med. 2002;28:376, doi: 10.1007/s00134-001-1195-0.
6. Sue R, Matthay MA, Ware LB. Hydrostatic mechanisms may contribute to the pathogenesis of human re-expansion pulmonary edema. Intens Care Med. 2004;30:1921–6, doi: 10.1007/s00134-004-2379-1.
7. Mingolla GP. Re-expansion pulmonary edema. J Emerg Med. 2009;36:80–2, doi: 10.1016/j.jemermed.2007.11.113.
8. Baik JH, Ahn MI, Park YA, Park SH. High resolution CT findings of re-expansion pulmonary edema. Korean J Radiol. 2010;11:164–8, doi: 10.3348/kjr.2010.11.2.164.
9. Papakonstantinou DK, Gatzioufas ZI, Tzegas GI, Stergiopoulos PI, Tsokantaridis CG, Chalikias GK, et al. Unilateral pulmonary oedema due to lung re-expansion following pleurocentesis for spontaneous pneumothorax. The role of non-invasive continuous positive airway pressure ventilation. Int J Cardiol. 2007;114:398–400, doi: 10.1016/j.ijcard.2005.11.084.
10. Light RW, Jenkinson SG, Minh VD, George RB. Observations on pleural fluid pressures as fluid is withdrawn during thoracentesis. Am Rev Respir Dis. 1980;121:3799–804.
11. Villena V, Lopez-Encuentra A, Pozo F, De Pablo A, Martin Escribano P. Measurement of pleural pressure during therapeutic thoracentesis. Am J Respir Crit Care Med. 2000;Z162:1534–8.
12. Feller-Kopman D, Berkowitz D, Boiselle P, Ernst A. Large volume thoracentesis and the risk of reexpansion pulmonary edema. Ann Thorac Surg. 2007;84:1656–62, doi: 10.1016/j.athoracsur.2007.06.038.
13. Feller-Kopman D, Walkey A, Berkowit D, Ernst A. The relationship of pleural pressure to symptom development during therapeutic thoracentesis. Chest. 2006;129:1556–60, doi: 10.1378/chest.129.6.1556.