



Reinflation Pulmonary Edema Following Decompression of Massive Spontaneous Pneumothorax/Pleural Effusion: A Rare but Avoidable Catastrophe

Ghulam Nabi Lone*, Farooq Ah Ganie, Syed Wahid, Nadeem ul Nazar, Mehmood Ahmad Wani, Mohd Akbar Bhat and Haroon Naqshi

Department of Cardiovascular and Thoracic Surgery, Sher-i-Kashmir Institute of Medical Sciences, India

Abstract

Background: Reinflation pulmonary edema is a rare but fatal complication with a high mortality following rapid reinflation of a chronically collapsed lung for massive pneumothorax or pleural effusion. Pathophysiology is still unclear but hypoxia induced oxidative stress, ischemic reperfusion injury, inflammatory response and negative intrathoracic pressure are possible mechanisms.

Methods: A prospective analysis of 842 patients with spontaneous pneumothorax/pleural effusion is reported. All the patients have reported to the hospital during the period from August, 1997 to August, 2018. Risk factors were evaluated and clinical profile of patients with re-expansion pulmonary edema was studied.

Results: Six of 842 (71%) patients had developed reinflation pulmonary edema after decompression of spontaneous pneumothorax and pleural effusion. Out of 6 patients 5 had massive spontaneous pneumothorax and 1 had massive pleural effusion. Two of 6 (33.3%) patients died instead of all possible manures and mechanical ventilation.

Conclusion: Reinflation pulmonary edema should always be considered when in presence of risk factors patient develops hypoxemia after drainage/re-expansion for spontaneous pneumothorax and deteriorates dramatically. Treating surgeons need to be quite aware of this rare entity and take immediate steps to correct it.

Introduction

Reinflation pulmonary edema or Re-expansion Pulmonary Edema (RPE) is a rare but fatal complication which occurs after rapid re-expansion of collapsed lung after the insertion of a chest tube for massive pneumothorax or pleural effusion. Foucart first described the development of pulmonary edema following large volume thoracocentesis in 1875. However, re-expansion pulmonary edema has been first reported to occur after evacuation of pneumothorax by Carlson in 1958 [1]. Several case reports and case series have been published so far. Mayfood et al. [2] analyzed 47 patients who developed RPE after re-expansion from pneumothorax in between 1958 to 1987. Eighty three percent cases occur as a result of drainage of chronically collapsed lung from pneumothorax. RPE can occur on ipsilateral side (83%) or bilaterally (6.7%) or on contralateral side (10.3%) and can be asymptomatic even [2]. It starts manifesting within first hour of drainage in almost 64% cases but uniformly results in 24 h and can persist for 4 to 5 days. Although, it is likely that rapid re-expansion by application of negative pressure contributes to RPE, it can still result in more than 33% cases when re-expansion is allowed without pleural suction. The incidence ranges between 1% to 14%, as described in the literature available [3,4]. The low incidence of RPE in spontaneous pneumothorax can be explained by the fact that most of the cases report to the hospital due to sudden breathlessness and chest pain, and are treated well in time. The mortality remains very high (20%) and is determined by several risk factors like younger age (<40 years), longer duration of lung collapse (>4 days), large pneumothorax (>30% of a single lung), and timing/technique of lung re-expansion [2,5,4].

Pathophysiology

Human data on the pathophysiology of re-expansion pulmonary edema derive from small series of patients, case reports and reviews of the literature. On the other hand, much reliance is laid on experimental data in cats, monkeys, rabbits, sheep, and goats by creating re-expansion pulmonary

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*Correspondence:

Ghulam Nabi Lone, Department of Cardiovascular and Thoracic Surgery, Sher-i-Kashmir Institute of Medical Sciences, Room No- 1317, Soura, Srinagar, Kashmir, 190011, India, Tel: 9419053025, + 91-0194-2401013-2260; Fax: 0091-0194-2403470;

E-mail: gn_lone@yahoo.co.in

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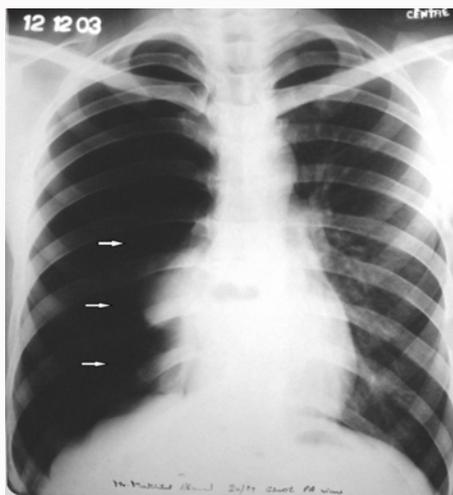


Figure 1: Chest radiogram (PA) revealing massive pneumothorax with collapse of right whole lung. Arrows mark the collapse of lung. This patient reported after 6 days of manifestation.

edema and studying various biochemical and histopathological changes. The exact pathophysiology for this complication remains unknown. However, mechanical stress on the alveoli with alveolar-capillary membrane disruption and ischemic-reperfusion injury after prolonged collapse are thought to be of vital importance. These processes are likely to be initiated by the stretching or distention and increased pulmonary blood flow that occur during pulmonary re-expansion [6]. Once there is microvascular injury, the pathologic process is believed to propagate and worsen by local cellular imbalances in nitric oxide levels and byproducts. Moreover, the activity of different cytokines such as interleukin 8 and Monocyte Chemoattractant Protein 1 (MCP-1) or the activity of xanthine oxidase has been implicated in the pathogenesis of RPE [7-9]. An inflammatory process with migration of polymorphonuclear leucocytes and degranulation is believed to be set in after microvascular injury which worsens the pathological process [10,9,11]. Oxidative stress characterized by release of cytotoxic oxygen metabolites with consequent unilateral lung injury has been implicated as a main factor by some authors [11,12]. These metabolites or oxygen free radicals are released after re-oxygenation of collapsed lung [3,2,6]. It has been demonstrated in an animal model that reinflation pulmonary edema results in increase in Malondialdehyde (MDA) level but decrease in Catalase, Superoxide Dismutase, and Glutathione Peroxidase activity in pulmonary tissue. Rats treated with Proanthocyanidine resulted in less severe re-expansion pulmonary edema, partially reversed Superoxide Dismutase, Catalase, and Glutathione Peroxidase levels. It significantly decreased levels of Malondialdehyde which is a marker of oxidative stress, in the lung tissue [11]. Hypoxia-reoxygenation injury of one lung can manifest as Acute Respiratory Distress Syndrome (ARDS) in the other lung along with systemic multiorgan injuries [13]. This can explain the development of contralateral or bilateral re-expansion pulmonary edema. Hence, reinflation pulmonary edema in absence of high negative intrathoracic pressure can be explained by a mechanism which does not rely on negative intrathoracic pressure like hypoxic reoxygenation or ischemic reperfusion lung injury.

Effect of negative intrathoracic pressure: Much emphasis has been laid by the authors on high negative intrathoracic pressure by pleural suction before developing reinflation pulmonary edema as a plausible

mechanism. Fluid balance between interstitial and vascular bed in the lung as in other microcirculations is determined by the Starling relationship which predicts the net flow of fluid across a membrane. This can be expressed in the following equation:

$$\begin{aligned} \text{Net filtration} &= LP \times S (\Delta \text{Hydraulic Pressure} - \Delta \text{Oncotic Pressure}) \\ &= (LP \times S) \times \{[P_{cap} - P_{if} - S (n_{cap} - n_{if})]\} \end{aligned}$$

Normal intrapleural pressure is - 3 cm to 10 cm H₂O. When the intrapleural pressure becomes high (-50 cm to -100 cm H₂O), the fluid starts moving out of pulmonary capillaries into interstitial and alveolar spaces due to high pressure gradient. Secondly, marked intrathoracic negative pressure results in sudden increase in venous return of blood to the heart which exposes the left ventricle to an after load stress with consequent increase in both end-diastolic and end-systolic ventricular volumes leading to increase in left ventricular end-diastolic pressure. This increase in pulmonary capillary pressure and drop in intra-alveolar pressure favours disruption of capillary-alveolar membrane and development of pulmonary edema. Lung collapse with resultant hypoxemia enhances both pre-capillary and post-capillary pulmonary vascular resistance precipitating hyperadrenergic state and hence, compliments the adverse effects of high intrathoracic negative pressure [14].

Material and Methods

842 patients were admitted for management of spontaneous pneumothorax from August, 1997 to August, 2018 in the department Cardiovascular & Thoracic Surgery, Sher-i-Kashmir Institute of Medical Sciences, Srinagar, Kashmir. Patients with pleural effusion were admitted and evaluated under the department of Internal Medicine and shifted to our department in case it was thought mandatory to drain pleural effusion surgically. Many patients with malignant pleural effusion were referred by Medical Oncology for drainage procedure. All such referred patients were returned back to parent department after decompression for further management. Rest of the patients comprising mainly those with spontaneous pneumothorax, were admitted under Emergency ward. They were thoroughly evaluated and actively managed. During the course of their admission, plain chest radiogram was enough to confirm pneumothorax/pleural effusion. Intercostal tube was inserted under local anesthesia (2% Lignocaine) into pleural cavity *via* 5th intercostal space in midaxillary line on the affected side. Negative suction was only applied when lung was seen collapsed totally and expansion was slow. Primary objective of treatment was to decompress pneumothorax and watch for any complication. Our late objective was to elucidate the underlying etiology of pneumothorax etc. Patients were shifted from Emergency ward to Cardiothoracic and Vascular Surgery department after initial treatment. Their detailed history including smoking, past history of pneumothorax and intercostal tube insertion, pulmonary tuberculosis, recent or old chest infection, amenorrhoea, family history of spontaneous pneumothorax etc was noted down. Computed tomogram was done only after stabilisation. Tuberculous profile was examined, as pulmonary tuberculosis is quite rampant in Kashmir due to socio-economic, environmental and cultural factors.

Results

Six of 842 (71%) cases with spontaneous pneumothorax (674) and pleural effusion (168) developed unilateral pulmonary edema. Five were males and one was female. All the five males were smokers but the lonely female patient was non-smoker. As such smoking

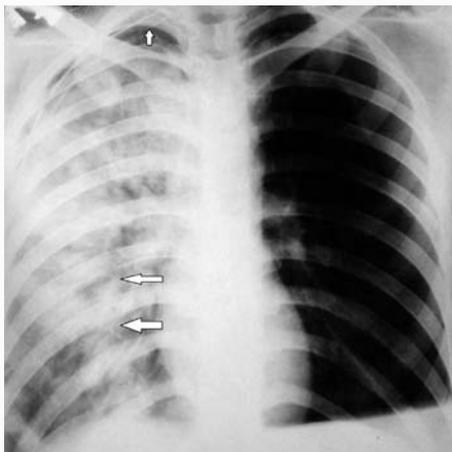


Figure 2: Chest radiogram (PA) taken 2 h after thoracostomy, revealing an intercostal tube inside right pleural cavity and ipsilateral pulmonary edema. Small arrow shows intercostal tube and large arrow marks unilateral re-inflation pulmonary edema.

amongst females in Kashmir is very rare and is seen sometimes in old, tribal rural women and in fact, is thought to be a curse. Strikingly all the patients were between 25 years to 35 years of age. Invariably, all the patients had breathlessness and chest pain on the affected side. Examination of the chest revealed diminished breath sounds on the affected side and tracheal shift. In four, right lung was affected and in other 2 cases left lung was affected. All the five patients with spontaneous pneumothorax had massive collapse of lung and the patient with pleural effusion was drained more than 2.0 liters of fluid by a resident doctor. He had applied negative suction as well. None of the patients had bilateral pulmonary edema. Amongst six, five had prolonged history of pneumothorax for more than 24 h and one presented to us after 144 h with chest pain and breathlessness (Figure 1). He was first taken to a quack and then to an orthopedic surgeon for management of chest pain where he wasted a valuable length of time. Orthopedic surgeon after suspecting pneumothorax referred the patient to our hospital for management. All the five patients with massive pneumothorax were admitted in Emergency ward and they were shifted to operation theatre for urgent intercostal tube insertion. Chest tube was put in midaxillary line on the corresponding side under local anesthesia. In two patients including the patient with pleural effusion negative suction was applied. They improved temporarily but within 60 min to 90 min, they deteriorated. Breathlessness increased paradoxically and blood tinged sputum started pouring out till frank pulmonary edema was evident. Blood gas analysis revealed severe hypoxemia and patients started desaturating. Patients were immediately put on oxygen by facemask and diuretics. Repeat chest radiogram in both of the patients revealed evidence of expanded lung but with unilateral edema on the ipsilateral side which usually is not expected (Figure 2). On failure to improve with diuretics and oxygen inhalation, they were put on mechanical ventilator with positive end-expiratory pressure but could not make it. They developed refractory pulmonary edema with persistent hypoxemia and fulminant hypotension. Inotropic did not work at all and one of them who had presented to us after 6 days (144 h), died 36 h after his hospitalization on ventilator. Just before his death while on ventilator, his chest radiogram revealed homogeneous, opaque right lung (Figure 3). Second patient with re-expansion pulmonary edema after drainage of pleural effusion died after 24 h of ventilation when everything failed.

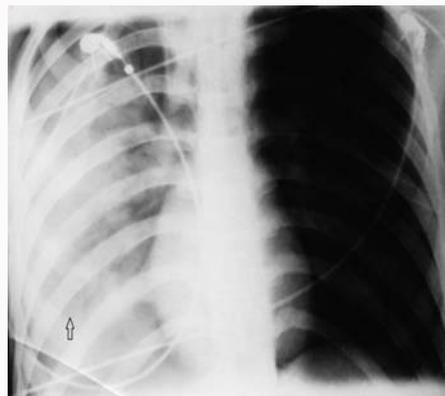


Figure 3: Portable chest radiogram (PA) of same patient taken 36 h after thoracostomy while he was on ventilator. Whole of right lung has turned white and mediastinum has shifted to right side.

Before drainage she was undergoing evaluation and thoracentesis for massive pleural effusion which was most probably secondary to viral pneumonia.

Three of four patients with non-fulminant re-inflation pulmonary edema who were salvaged by prompt management were weaned off from ventilator after 36 h and shifted to ward on oxygen on facemask. One patient did not need ventilation and his pulmonary edema subsided on energetic medical management. In one of four patients who survived, required intercostal tube drainage for 72 h and drainage tubes were removed when air leak stopped and in another 3 of them intercostal tubes were removed after 7 days to 9 days due to air leak. Fourth patient, on evaluation, had an emphysematous bulla as documented by computed tomogram of chest. He was subjected to thoracotomy and excision of the bulla. He had a prolonged air leak for 14 days which subsided of its own. All the four surviving patients were negative for pulmonary tuberculosis which is very common in Kashmir. They were discharged from hospital without any complications.

Discussion

The clinical manifestations of RPE are varied and range from roentgenographic findings alone in asymptomatic patients to severe cardiorespiratory insufficiency. The radiographic evidence of re-expansion pulmonary edema is a unilateral alveolar filling pattern, seen within a few hours of re-expansion of the lung. The condition usually appears unexpectedly, immediately and dramatically or within 1 h of intercostal tube drainage in most of the cases. Usually RPE is self limited and can even be asymptomatic [4]. However, a mortality rate as high as 20% has been described [3,2]. This rare complication and its treatment should be known by clinicians and particularly considered when a patient's condition declines after initial amelioration. Re-expansion pulmonary edema can assume a fulminant course without giving an opportunity to resuscitate and mechanical ventilation [15]. We present even higher mortality (33.3%) i.e., 2 deaths (33.3%) amongst 6 cases of a RPE after the intercostals tube thoracostomy for spontaneous pneumothorax/pleural effusion. All the six were young. Five had massive spontaneous pneumothorax; one had massive non-tuberculous pleural effusion. Initially, they improved after drainage but later on deteriorated dramatically. Two of six cases had a fulminant course and died after initial transient improvement. Four survived who initially were managed by administration of oxygen *via*

face mask but later on 3 of 4 surviving patients required to be put on mechanical ventilator.

Although, clinicians may overlook the risk factors mentioned in the literature but these are of great importance and should never be ignored. Almost all these risk factors were observed in our patients. However, negative suction was not a risk factor always. Mostly these patients report to emergency ward and need urgent hospitalization and treatment. Many times they are being managed by resident doctors not aware of the entity. If the treating doctor will avoid applying negative pressure by suction and perform slow decompression, this catastrophe may be avoided. There is a probability that even if it occurs, the severity shall not be fulminant. In our group of patients, the relevance of these risk factors was also possibly overlooked and all the six patients were managed by resident doctors not aware of this catastrophe. Nevertheless, the presence of the risk factors like young age, massive collapse, rapid decompression and high negative pressure does not always predict an adverse outcome with certainty but should guide the clinician to a more cautious procedure in order to prevent RPE. There is enough evidence in the literature to believe that intercostal tube drainage is sometimes performed on the affected side, RPE manifests on contralateral side [16]. It can occur even bilaterally even if other lung is not touched [17]. It has been also reported to occur after pleurocentesis for decompression of spontaneous pneumothorax [18,19]. It generally affects whole lung uniformly but sometimes only one lobe may be affected. It has also been reported by many authors to occur in patients of recurrent malignant pleural effusion drained by VATS [20]. However, the incidence is very low (0.15%).

Generally, two main contributory factors are involved: Amount and speed with which air/fluid is drained and the chronicity of lung collapse. Other factors do contribute to development of RPE like technique used, pulmonary arterial hypertension, associated hypoxemia, bronchial obstruction. A lung collapse of more than 72 h and rapid evacuation of fluid/air leading to end-expiratory pleural pressure less than -20 cm H₂O, is associated with higher risk of RPE [11]. An increasing number of patients with acquired immunodeficiency syndrome with predilection to spontaneous pneumothorax and RPE have been observed. Such patients can be better managed by a tube thoracostomy or a Heimlich valve [2]. Cardiac disorders like cardiomyopathy and valvular heart defects may also predispose a patient to high negative pressure and consequent pulmonary edema. 50% of patients with high negative pressure induced pulmonary edema do have such disorders in contrast with 1% in general population [14]. Neither high negative intrathoracic pressure nor cardiac disorders did account for higher mortality in our setting. Although, generally we did not practice negative pressure manueers still re-expansion pulmonary edema occurred.

The mainstay of treatment for reinflation pulmonary edema remains oxygenation, a low threshold for mechanical ventilation with positive end-expiratory pressure, diuresis and hemodynamic support. Positioning the patient in the lateral decubitus position with affected side up may help reduce edema and intrapulmonary shunting. Closed drainage with gradual lung expansion should be encouraged and negative suction can be applied only once the lung is almost expanded. Usually it subsides within 72 h. Noninvasive continuous positive pressure ventilation can help some times before putting the patient on mechanical ventilation [3,18,21]. Recently differential ventilation has been utilized for treating re-expansion pulmonary

edema when mechanical ventilation fails [22]. Measurement of intrapleural pressure by pleural manometry can be added while draining large volumes of pleural fluid keeping the pleural pressure above -20 cm H₂O and patients do not develop chest pain during tapping [19]. Prostaglandin analogue misoprostol and ibuprofen or indomethacin suppositories have been used for reasons of anti-inflammatory or cytoprotective actions [2]. After slow drainage, patients with difficult recovery or proved emphysematous bulla can be considered for bullectomy or chemical pleurodesis.

In experimental animals after inducing pneumothorax some authors have used proanthocyanidine which is a combination of biologically active polyphenolic flavonoids [11]. Adult rats were treated with proanthocyanidine for 3 days (100 mg/kg/day). Pneumothorax was induced by injecting about 4 ml of air into thorax by percutaneous route with 22 gauge cannula placed in right hemithorax. Adequacy of pneumothorax was confirmed with control X-rays. Rats were allowed to survive for additional 72 h. Pneumothorax was treated by quickly aspirating air with 22 gauge cannula. Re-expansion was assessed by control X-ray. Two hours after expansion, rats were sacrificed by giving lethal dose of Xylazine and Ketamine. Chest was opened through median sternotomy and both lungs were removed for biochemical and histopathological sampling. Lung tissue was studied both for markers of oxidative stress and histopathological changes which were graded by a pathologist as “normal pulmonary parenchyma”, “minimal changes” (only fluid extravasations), “moderate changes” (fluid in the alveoli as well as fluid extravasations), “advanced changes” (typical histopathological findings of pulmonary edema). These grades have been given pulmonary edema scores like Score-0, Score-1, Score-2, Score-3, respectively. Similarly, control groups were also studied and compared with study groups. Results were subjected to statistical analysis. It was demonstrated that such a treatment exerts a novel spectrum of biological, pharmacological, therapeutic and chemo-protective properties against oxidative stress and oxygen free radicals. By virtue of its potent hydroxyl and other free radical scavenger ability, it has a cardioprotective effect. As compared to Vitamin C, E and β-carotene, it provides better antioxidant efficacy. In addition, it improves cardiac recovery during reperfusion of ischemic conditions [12].

Conclusion

Reinflation pulmonary edema is a rare complication with high mortality rate occurring after thoracic drainage for pneumothorax or pleural effusions. Clinicians should be familiar with this complication and the associated risk factors. Rapid decompression and high negative pressure suction should be always discouraged. A high index of suspicion is required to ensure diagnosis of this catastrophe. Treatment is symptomatic.

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