

ORIGINAL ARTICLE

REEXPANSION PULMONARY EDEMA AND ITS TREATMENT APPROACHES

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Background – Reexpansion pulmonary edema (RPE), a rare complication of the reexpansion of a collapsed lung, typically follows the evacuation of a massive pleural effusion or complete pneumothorax. The aim of this study was to determine the clinical features, outcome, and effective treatment approach of RPE and also to evaluate the correlation of this phenomenon and its outcome with the underlying cause of lung collapse.

Methods – This retrospective descriptive study was carried on 389 cases of chest intubation in 4 subsets of patients with the following underlying conditions: pneumothorax (200 patients), pleural effusion (100 patients), chylothorax (9 patients), and atelectasis (80 patients). Demography and data of variables were extracted from the hospital case records of the patients admitted within a 67-month period (from January 1993 to March 1998) in Thoracic Surgery Department of Razi Hospital and Cardiac Surgery Department of Heshmat Cardiovascular Center, Rasht, Iran. The statistical significance of the association between RPE and its underlying condition or treatment was assessed using χ^2 analysis and Fisher's exact test; $p < 0.05$ was considered significant.

Results – Among 389 cases of lung collapse including 200 pneumothorax, 100 pleural effusion, 9 chylothorax, and 80 atelectasis patients, 20 cases (5.1%) were complicated with RPE. Fifteen patients (75%) had severe and 5 patients (25%) showed mild tachypnea. All pleural effusion cases, 70% of pneumothorax, and 50% of atelectasis cases severe tachypnea. In eighty percent of the RPE cases the complication appeared within the first hour of chest tube insertion while in the remaining 20%, RPE occurred within the first 24 hours. One of the patients with RPE who was a case of pneumothorax died (5% mortality). All patients were given oxygen and 14 patients (70%) received corticosteroids. Indomethacin suppositories were administered to 11 (55%) patients. Twenty-five percent of RPE cases required bronchodilators while only one case (5%) had received a bronchodilator drug.

Conclusion – We concluded that RPE is not a rare complication. Early diagnosis and appropriate approach can reduce mortality. For patients with progressive hypoxemia and respiratory distress, the main aim of the treatment should be slowing the blood flow towards the involved lung; the simplest and quickest way to achieve this is lung recollapse. Meanwhile, appropriate hemodynamic monitoring, adequate fluid replacement, use of diuretics, and mechanical ventilation would be helpful.

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Keywords • pleural effusion • pneumothorax • reexpansion pulmonary edema (RPE)

Introduction

Reexpansion pulmonary edema (RPE), a rare complication of the reexpansion of a collapsed lung, typically follows the evacuation of a massive pleural effusion or

complete pneumothorax.¹⁻³ RPE is more likely to occur when the lung has been collapsed for a prolonged period, but it can also occur when the collapse is of short duration.¹⁻⁴ RPE develops within 1 hour of reexpansion in two-thirds of cases and within 24 hours of reexpansion in the remainder.³ In the majority of reported cases, RPE is distributed diffusely throughout the reexpanded

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Table 1. Age and sex distribution of subgroups in RPE.

Description	Pneumothorax	Pleural effusion	Chylothorax	Atelectasis (n = 4)	Sum
Male [No. (%)]	160 (80%)	70 (70%)	5 (55%)	48% (60%)	283 (73%)
Female [No. (%)]	40 (20%)	30 (30%)	4 (4%)	32(40%)	106 (27%)
Mean age	35	40	46	30	36

lung.¹⁻⁵ Death has been reported to occur in up to 20% of cases.^{3,4} Due to rarity of this complication of a common surgical procedure, the etiology, exact prevalence, and routine management protocol of RPE have not yet been described in the medical text.

We carried out this study to define the clinical features, outcome, and effective ways of management; and also to determine the correlation of RPE and its outcome with the underlying cause of lung collapse.

Patients and Methods

This retrospective study was carried on 389 cases of chest intubation in 4 subsets of patients with the following underlying conditions: pneumothorax (200 patients), pleural effusion (100 patients), chylothorax (9 patients), and atelectasis (80 patients). Demography and data of variables were extracted from the hospital case records of patients admitted within a 67-month period (from January 1993 to March 1998) in Thoracic Surgery Department of Razi Hospital and Cardiac Surgery Department of Heshmat Cardiovascular Center, Rasht, Iran. Variables included patients' symptoms, physical signs, time of onset, treatment approach, and the drugs used for treatment.

The statistical significance of the association between RPE and its underlying condition or treatment was assessed using χ^2 analysis and Fisher's exact test; $p < 0.05$ was considered significant.

Results

Of the 389 patients, 283 (73%) were male while 106 (27%) were female. They were generally young and otherwise fit, with a mean age of 36 years. Thirteen (65%) male and 7 (35%) female cases suffered from RPE complication. The mean age of RPE cases was 34.2 years (Table 1). Description of RPE patient to underlying causes

has been shown in Table 2.

Of 389 cases of lung collapse, including 200 pneumothorax, 100 pleural effusion, 9 chylothorax, and 80 atelectasis patients, 20 cases (5.1%) were complicated with RPE. Fifteen patients (75%) had severe and 5 patients (25%) mild tachypnea.

All of pleural effusion cases, 70% of pneumothorax, and 50% of atelectasis cases showed severe tachypnea. Fifteen patients (75%) had blood-stained or foamy expectoration. Anxiety, apprehension, and fine crepitation on auscultation appeared in all patients (100%) with RPE.

Fourteen patients (70%) had profuse perspiration. Details of clinical features are shown in Table 3. In 80% of RPE cases the complication appeared in the first hour of chest tube insertion. In the remaining 20%, it appeared within the first 24 hours after a chest tube was inserted (Table 4). One patient with RPE died who was a case of pneumothorax (5% mortality of RPE as stated in Table 5).

All patients were given oxygen and 14 patients (70%) received corticosteroids. Indomethacin suppositories were given to 11 (55%). Twenty-five percent of RPE cases required bronchodilators while only one case (5%) had received a bronchodilator drug. Details of treatment approach have been shown in Table 6.

Discussion

RPE was first described in the 19th century as *lalbuminous expectorationi* after the drainage of a pleural effusion.³⁻⁶ RPE after the evacuation of pneumothorax was not recognized until 1958.^{3,4,7} Since then, most reported cases of RPE have occurred after evacuation of pneumothorax.^{3,4} Ravin and Dahmash⁸ have also shown that RPE can occur after reexpansion of an atelectatic lung that collapsed from reversible endobronchial obstruction.

The true incidence of RPE is unknown. In two large series, no cases of RPE were reported after

Table 2. Description of RPE patients according to the underlying cause subgroups.

Description	Pneumothorax	Pleural effusion	Chylothorax	Atelectasis	Sum
Male [No. (%)]	160 (80%)	70 (70%)	5 (55%)	48 (60%)	283
Female [No. (%)]	40 (20%)	30 (30%)	4 (4%)	32 (40%)	106
Mean age	34.2	40	46	30	34.2

RPE and Its Treatment Approaches

Table 3. Frequency of symptoms and signs of RPE patients.

Description		Pneumothorax (n = 10)		Chylothorax (n = 1)	Atelectasis	Total (n = 20)
		Pleural effusion (n = 5)				
Tachypnea	S	7	5	1	2	15
	M	3	0	0	2	5
Foamy or bloody-stained expectoration	Y	7	5	0	3	15
	N	3	0	1	1	5
Humid rales	Y	10	5	1	4	20
	N	0	0	0	0	0
Severe perspiration	Y	7	5	0	2	14
	N	3	0	1	2	6
Anxiety	Y	10	5	1	2	20
	N	0	0	1	4	0

S = severe; M = mild; Y = yes; N = no.

the evacuation of pneumothorax in 776 patients.⁹ However, Mohfood et al⁴ reported RPE in 14% of cases of spontaneous pneumothorax managed in their institution, and Takamura et al¹⁰ found RPE in 27% of their cases. In our study RPE occurred in 20 (5.1%) of 389 patients.

The etiology of RPE has been debated for years. No established mechanism has been described for it yet. Authentic texts include no description of the clinical symptoms, signs, or the treatment approach of RPE.^{1, 2, 4} Previously

study, was only 5.1% in pneumothorax-based lung collapse while it was reported up to 27% in a similar study.⁴ In our study, 16 cases (80%) suffered from RPE after one hour from the onset of expansion. Previous workers have reported the first hour RPE as 64%–66%.^{3, 4, 7} This may be due to the late arrival of patient and prolongation of lung collapse.

Neither in the textbooks nor in the recently published works is there any description of clinical symptoms and signs of RPE.^{11, 12} The present study

Table 4. Distribution of events after one hour and 24 hours in RPE patients.

Description	Pneumothorax	Pleural effusion	Chylothorax	Atelectasis	Sum
After one hour	7 (70%)	4 (80%)	1 (100%)	4 (100%)	16 (80%)
After 24 hours	3 (30%)	1 (20%)	0 (0%)	0 (0%)	4 (20%)
Total	10	5	1	4	20

proposed mechanisms for the development of RPE include increased hydrostatic pressure from vascular flooding of the reexpanded lung in response to negative intrapleural pressure,^{3, 4} altered pulmonary vascular permeability from mechanical linear stretching of the pulmonary microcirculation, and tension on the alveolar walls due to the application of negative intrapleural pressure.¹¹

Pavlin et al¹¹ found very high measured concentrations of 1,311 albumin in the extravascular extracellular lung water of rabbits with RPE, further indicating that RPE represents increased vascular permeability edema. Previous studies have shown that blood flow to the atelectatic lung is decreased, especially when lung volume is reduced by 50% or more, and that pulmonary blood flow remains low for a prolonged time after complete reexpansion.¹²

The incidence of RPE, found in the present

suggests the following as the expected symptoms and signs of clinical pulmonary edema: severe tachypnea was evident in 100% of pleural effusion cases, 70% of pneumothorax, and 50% of atelectasis cases while blood-stained or foamy expectoration was seen in 15 patients (75%); anxiety, apprehension, and fine crepitation on auscultation appeared in all patients (100%) with RPE while 14 patients (70%) had profuse perspiration. This is a small contribution of the present study to our colleagues.

One RPE patient (5%) with pneumothorax died. Post-RPE mortality has been reported up to 20% in previous reports while this study suggested a mortality rate of as low as 5%. This is because of the effective treatment approach including oxygen therapy, mechanical ventilation, corticosteroids, and NSAIDs beside the conventional medical management of acute pulmonary edema.

Recent experimental evidence suggests that

Table 5. Post-RPE mortality; relative frequency.

Description	Pneumothorax	Pleural effusion	Chylothorax	Atelectasis	Sum
Died	1 (10%)	0 (0%)	0 (0%)	0 (0%)	1 (5%)
Survived	9 (90%)	5 (100%)	1 (100%)	4 (100%)	19 (95%)
Total	10	5	1	4	20

Table 6. Relative frequency of treatment approaches for RPE patients.

Description	Pneumothorax	Pleural effusion	Chylothorax	Atelectasis	Sum
Oxygen	10 (100%)	5 (100%)	1 (100%)	4 (100%)	20 (100%)
Corticosteroids	7 (70%)	2 (40%)	1 (100%)	4 (100%)	14 (70%)
Bronchodilators	1 (10%)	0	0	0	1 (5%)
Opening chest tubes	4 (40%)	4 (80%)	1 (100%)	0	9 (45%)
Furosemide injection	2 (20%)	3 (60%)	0	1 (25%)	6 (30%)
Digoxin	2 (20%)	4 (80%)	0	1 (25%)	7 (35%)
Mechanical ventilation	2 (20%)	2 (40%)	0	1 (25%)	5 (25%)
Indomethacin suppository	5 (50%)	3 (60%)	1 (100%)	2 (50%)	11 (55%)

RPE represents altered vascular permeability edema from hypoxic injury to the atelectatic lung and further indicates that both the risk of developing RPE and the severity of RPE increase as the duration of pulmonary collapse and hypoxia increases.^{1, 2, 4}

We conclude that our treatment approach to RPE successfully decreased the mortality rate (5%) of this serious complication in comparison to other centers (20%). We suggest that oxygen therapy and rehydration are sufficient for mild cases of RPE. For patients with severe or progressive hypoxemia and those with respiratory distress, the main aim of treatment should be slowing the blood flow towards the involved lung; and the simplest and quickest way to achieve this, is lung recollapse though it may be harmful. Meanwhile, appropriate hemodynamic monitoring, adequate fluid replacement, use of diuretics, and mechanical ventilation can be helpful. Although it is difficult to predict which case may suffer from RPE, prudent interventions such as slow lung reexpansion using low pressure (-100cm H₂O) over some hours and avoiding prolongation of collapse may avoid reexpansion pulmonary edema.

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