COR ET VASA 55 (2013) E533–E535

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journal homepage: www.elsevier.com/locate/crvasa

Case Report

Re-expansion pulmonary oedema—fatal complication of mediastinal tumour removal

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ARTICLE INFO

Article history:
Received 24 April 2013
Accepted 9 May 2013
Available online 17 May 2013

Keywords:
Atelectasis
Mediastinal neoplasms
Re-expansion pulmonary oedema
Teratoma

ABSTRACT

We report a case of re-expansion pulmonary oedema (RPE) occurring after a mediastinal tumour removal procedure. RPE is a rare complication associated with the treatment of a collapsed lung caused by pneumothorax, pleural effusion or tumour. The risk factors are a longer period (more than three days) of collapsed lungs, the volume of intrathoracic lesion, loss of surfactant and the patient's age. Treatment of RPE is difficult due to there being no clear pathophysiology, and is associated with high mortality. Careful management and clinical guidelines are needed to render the therapy more effective.

1. Introduction

Re-expansion pulmonary oedema (RPE) was first described by Carlson in 1959. He reported that RPE occurred after the re-expansion of collapsed lungs caused by the pneumothorax being treated with thoracocentesis. The RPE may be a rare complication of evacuation of pleural effusion, mediastinal tumour resection, removal of large extrathoracic lesions (e.g. giant abdominal mass). Because of the associated high mortality (about 21%), the most effective clinical approach is identification of the risk factors, careful management of the patient and preventative assurance of the equipment needed for treatment of this complication.

2. Case

A 33-year-old man was admitted to our hospital and was scheduled for mediastinal tumour resection. His medical history was unremarkable. He had been having fatigue, malaise and low-degree fever for three months and dyspnoea for the last month. A chest X-ray revealed a giant tumour mass of the left hemithorax. CT examination showed a non-homogeneous tumour mass filling the whole of the left hemithorax (Figs. 1 and 2). The transparietal biopsy specimen could not determine the diagnosis: it showed necrotic tissue, old organised haematoma, the lung tissue was not present; immunohistochemical analysis did not confirm the malignant tumour. Tumour removal was recommended in our hospital, with the cooperation of cardiac and thoracic surgeons.

At the time of admission, the patient's pulmonary function studies revealed a severe restrictive ventilatory defect, but his haemoglobin saturation was 99% (room air). Laboratory tests showed mild normocytic anaemia (RBC 3.72 T/l, Hgb 95 g/l, Hct 0.309, MCV 83.1 fl), kidney function tests were normal, liver tests, except for a higher level of gamma glutamyltransferase, were also in the normal ranges. We found a low serum concentration of iron, total cholesterol and elevated C-reactive protein and ferritin level. INR was elevated (1.33), very high D-dimer levels were detected as the indicator of haemostatic activation and fibrinolysis. The ECG showed sinus tachycardia, without ischaemic changes.

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http://dx.doi.org/10.1016/j.crvasa.2013.05.001

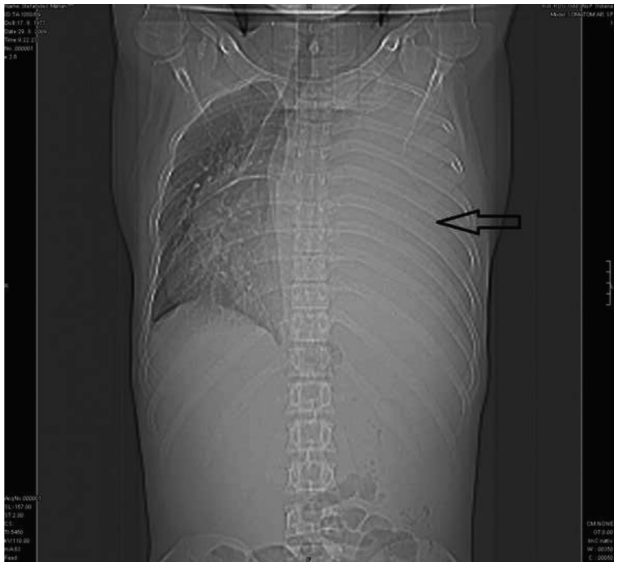

Fig. 1 – CT examination showed a non-homogeneous tumour mass filling the whole of the left hemithorax.

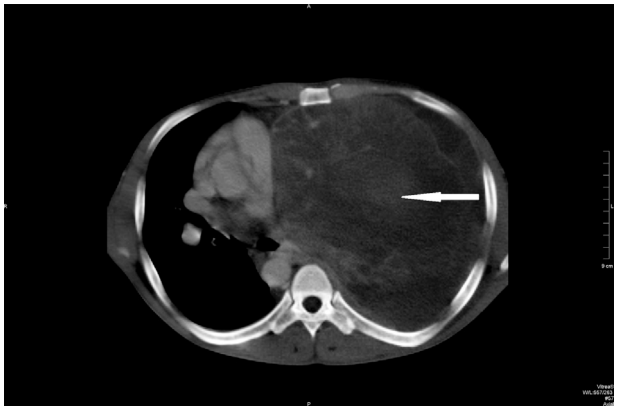

Fig. 2 – Giant tumour mass of the left hemithorax.

The transthoracic echocardiography examination in the usual projection was not possible. The massive tumour caused a mediastinal shift with displacement of the heart to the other side of the chest. There were no abnormalities of the valves, no left ventricular hypertrophy with ejection fraction 60%, the right atrium and ventricle were deformed by tumour pressure.

His pre-operative blood pressure was 125/70 mmHg, and heart rate was 100 bpm. The operation was performed in the supine position. After right subclavian vein cannulation, general anaesthesia was induced with etomidate 20 mg, esmeron 50 mg and midazolam 5 mg IV. The patient was intubated and mechanical ventilation was started in a pressure-controlled mode. The surgery was initiated using extracorporeal circulation (ECC) through cannulation of the femoral vessels; the basic central venous pressure (CVP) was high at 20–23 mmHg. We performed a median sternotomy and found a giant retrosternal tumour filling the whole left hemithorax and deviating the mediastinum and their organs to the right. The tumour mass was polycystic and of a firm consistency. We mobilised the tumour and, due to the big volume (weight was 3,165 kg), we made a left-side thoracotomy in the 4. intercostal space (Clamshell). The tumour infiltrated the completely atelectatic left lung. Partial resection of the tumour was not possible because of infiltration of the surrounding tissues and hypervascularisation. After preparation, the whole tumour was removed 5 h after surgery had started. During this period, we made precise haemostasis of nutrition vessels with double ligation, the amount of blood transfused was 550 ml, haemofiltration by ECC 2450 ml, the CVP was 16–17 mmHg, and the left lung became expanded. When ECC was stopped, the patient's blood pressure was low: 80/40 mmHg; we detected extensive diffuse bleeding. Locally and IV haemostasis was started, blood transfusion (2500 ml). We made a revision of the surgical wound without identification of the source of bleeding. We continued treating for circulatory shock, pharmacological resuscitation with vasopressors (noradrenalin, adrenalin), administration of haemostyptic therapy (antitrombin, protaminsulphat, terlipressin, plasma, acidum 4-aminomethylbenzoicum, acidum tranexamicum, etamsylatum, phytomenadionum) and hydrocortisone. Despite maximal intensive care, three hours later we terminated the resuscitation; the patient died as “mors in tabula”.

Histology confirmed a teratoma with mature and immature components accompanied by residual semi-nomatous tumour cell. The other pathological diagnoses after section were brain oedema, bilaterally fluidothorax and pulmonary oedema, pulmonary parenchymal and subpleural bleeding. Our suspicion was that the direct cause of death was a re-expansion pulmonary oedema following removal of a giant mediastinal tumour and haemostasis activation with fibrinolysis and shock due to operative trauma.

3. Discussion

RPE is a rare complication of the treatment of lung collapse secondary to atelectasis, pleural effusion, pneumothorax or mediastinal tumour and this complication generally manifests early, following re-expansion [1,2]. Re-expansion pulmonary oedema may also be related to: bronchial obstruction, application of excessive suction to the tracheobronchial tree during bronchoscopy and/or suctioning with a tracheal suction catheter, alteration of the pulmonary artery pressure, removal of large extra-thoracic lesions [3]. The incidence of RPE following pneumothorax and effusion is between 0% and 1% in most studies. The British Thoracic Society guidelines suggest –1.5 l pleural fluid should be drained at a time. The condition is associated with a high mortality (higher than 21%) [3].

The pathogenesis of re-expansion pulmonary oedema is controversial because the causes are unclear and probably multi-factorial. It can be uni-lobar or multi-lobar, depending on the degree of pre-existing atelectasis. Younger patient age is a risk factor in the development of RPE [1]. A relative lack of surfactant has been suggested as a causative factor [4]. The decrease in alveolar surfactant activity is said to induce pulmonary oedema by lowering the intrapleural pressure and powering the perivascular pressure of pulmonary microvessels [5].

After sudden re-expansion, the lung experiences a simultaneous rapid increase in blood flow and concurrent alveolar distention. This leads to increases in the pulmonary capillary pressure, hydrostatic pressure and pressure-induced mechanical alveolar-capillary disruption. The increased capillary permeability and transudation of fluid into the lung, overflow of fluid and protein contribute to the development of pulmonary oedema, hypoxia and cardiac dysfunction [3]. This enhanced endothelial permeability may then propagate and worsen as a result of the local cellular delivery of free radicals (the main basis of reperfusion injury) and inflammatory mediators. There are reports of the superoxide dismutase and cytochrome oxidase of mitochondria declining in a collapsed lung, but the mechanism of their action is unclear [6]. Sohara points out that the oxygen-derived free radicals and pulmonary microvascular injury induce leucocyte sequestration into the lung which injure the pulmonary microvessels, cause biological injuries and RPE. Nakamura reported increased interleukin 8, leukotriene B4 and polymorphonuclear leucocyte elastasa levels in the sputum in lungs after RPE [7]. Another potential factor in the pathogenesis of RPE is increased hydrostatic pressure from vascular flooding of the re-expanded lung caused by negative intrapleural pressure [8,9]. Hydrostatic pressure elevation is probably associated with the elevation of various selectin proteins (E-selectin, L- selectin, P-selectin) [3].

The clinical presentation of re-expansion pulmonary oedema can vary considerably, ranging from asymptomatic radiological findings to a combination of severe cardiac and respiratory insufficiency and circulatory shock. It is important to detect the initial signs of unilateral pulmonary oedema early in order to commence treatment and prevent the sequence referred to above that can lead to severe respiratory failure and death. The most common symptoms are dyspnoea, thoracic pain and cough. Physical findings are cyanosis, rales on auscultation, nausea, tachycardia, hypotension.

The radiological features are also variable: we can see X-rays with Kerley's B lines, interstitial opacities or consolidation.

The symptoms may appear within the first two hours after RPE [10], but it may be delayed by 24–48 h [3].

The treatment of RPE is difficult due to its unclear pathophysiology. However, there is no definitive treatment for this condition in its severest forms. The most reliable method in these cases is tracheal intubation and non-invasive positive pressure ventilation. In the setting of refractory pulmonary failure, different two-lung ventilatory strategies have been described. Asynchronous differential lung ventilation is a relatively new therapeutic modality for the pre-operative, intra-operative, or post-operative treatment of respiratory failure secondary to a ventilation-perfusion mismatch but is still considered controversial, especially if the patient is haemodynamically unstable [11]. Hung et al. report a case of RPE when pulse contour cardiac output monitoring using a single transpulmonary thermal dilution technique was applied to achieve optimal cardiac preload for organ perfusion and to prevent worsening of the pulmonary oedema from fluid overload. The other choice of effective treatment according to Tung et al. is bilateral, developing re-expansion pulmonary oedema treated with extracorporeal membrane oxygenation [12]. The other symptomatic therapy is the administration of vasopressor and ionotropic agent, body fluid management with diuretics and hyperosmotic colloid solution, glucocorticoids, bronchodilators [13].

4. Conclusion

Pulmonary re-expansion oedema is a rare complication in thoracic surgery. In severe cases the basic symptomatic therapy is not effective. It is important for anaesthesiologists to be aware of the possibility of circulatory collapse, respiratory impairment, and development of RPE during anaesthetic management of a patient with a mediastinal tumour and atelectasis. Guidelines are required for future management of RPE during mediastinal tumour resection procedures.

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