IPSILATERAL RE-EXPANSION PULMONARY EDEMA IN A NEUROSURGICAL PATIENT

- A Case Report -

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Abstract

We report a case of a 42-year-old female with right parietal glioma, scheduled for elective craniotomy and tumor excision. The patient developed pneumothorax in the postoperative period. An incidence of unilateral pulmonary edema occurring as a result of rapid re-expansion of collapsed lungs is described. This type of pulmonary edema may delay postoperative recovery and have a catastrophic course especially in neurosurgical patients.

Keywords: neurosurgery; pneumothorax; re-expansion pulmonary edema.

Introduction

Acute ipsilateral re-expansion pulmonary edema (RPE) is a well documented complication of the treatment of lung collapse secondary to pneumothorax, pleural effusion and atelectasis[1]. Earlier reports have mentioned RPE in non-neurosurgical patients. It is important to recognize the significance of RPE in neurosurgical patients because such complications

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may be catastrophic, especially in the immediate post operative period if not diagnosed and managed on time.

Case Report

A 42-year-old female weighing 48 kg, a case of right parietal glioma, was scheduled for elective craniotomy and tumor excision. Her medical history was unremarkable. All routine investigations were within normal limits. Her cardiovascular and respiratory systems were unremarkable.

On the day of surgery she was premedicated with glycopyrrolate 0.2 mg intramuscular, one hour before surgery. General anesthesia was induced with thiopentone 200 mg and fentanyl 100 mcg, followed by rocuronium 50 mg to facilitate tracheal intubation. Anesthesia was maintained with isoflurane in nitrous oxide and oxygen mixture, and fentanyl 1 mcg/kg/hr. Muscle relaxation was achieved with vecuronium intermittent doses.

Intraoperative monitoring included electrocardiography, pulse oximetry, end tidal carbon dioxide, invasive blood pressure, concentration of anesthetic gases, central venous pressure and airway pressure. A double lumen central venous catheter was inserted in the right subclavian vein using infraclavicular approach in single attempt. The intraoperative course of six hours was uneventful.

At the end of surgery, as the patient remained drowsy even after reversal of the neuromuscular blockade, the trachea was not extubated and patient was transported to ICU for supportive care and further management.

Four hours later, blood oxygen saturation dropped to 86%. Decreased breath sounds were noted over the right hemithorax. Arterial blood gas analysis was PaO_2 86 mmHg and $PaCO_2$ 27 mmHg with FiO_2 of 0.8. Chest radiography revealed a right sided pneumothorax which was believed to be due to central line insertion. An immediate evacuation using intercostal tube thoracotomy attached to an underwater seal was done. No dyspnea or hypotension was noted.

Few minutes later vigorous coughing with expectoration of pink frothy

secretion was noted from the tracheal tube. Propped up position, positive pressure ventilation and morphine administration improved the condition. The patient remained hemodynamically stable. Serial chest radiography performed daily, showed progressive expansion and clearing.

Four days later, complete resolution of the edema occurred. Over the next two days, the patient was weaned off ventilator and trachea extubated. Four days later she was discharged neurologically intact with no respiratory complaints.

Discussion

Many factors are associated with development of RPE. The duration and severity of lung collapse and speed of expansion are important factors[2]. In general, it is agreed that edema is more likely to occur as a complication of large pneumothoraces when collapse has been prolonged for a period of at least three days[3]. Duration from onset of symptoms averaged 18 days with a minimum of three day[4].

In the case presented, the duration of pneumothorax was less than 6 hours. There are few case reports mentioning pulmonary edema after a pneumothorax for less than 24 hours whose diagnosis was based on radiographic evidence rather than on symptoms[5]. A more acute form of RPE, only two hours after atelectasis, during thoracic stage of esophagectomy has also been reported[6]. Ravin and Dahmash reported one instance of unilateral pulmonary edema, after re-expansion of an atelactatic lung of a short duration, due to accidental placement of endotracheal tube in the right main bronchus[7].

The possible causes of pulmonary edema, in our case are reviewed. The large volume of intravenous fluids and blood products administered during surgery was to replace the large blood loss. However, since the central venous pressure (CVP) was well maintained during the per-operative period, the replaced fluids and the resulting hemodilution and possible dilutional hypoproteinemia acted as predisposing rather than causative factors.

Head trauma and post craniotomy patients are known to develop 'neurogenic' pulmonary edema either indirectly due to vasoconstriction caused by sympathetic over activity, or directly due to 'missile' emboli from major vessels of the brain. Though not uncommon, there is less likelihood of this mechanism occurring in our patient who underwent supratentorial surgery, since infratentorial structures are more implicated as compared to supratentorial craniotomy. In addition pulmonary edema in these cases is associated with increased intracranial pressure or subarachnoid hemorrhage and is bilateral[8].

Another possibility for pulmonary edema is the effect on capillaries of the sudden and large increase in negative intrapleural pressure which occurs with rapid expansion brought about by the use of intercostal tube drainage rather than needle thoracotomy. Rapid re-expansion of a collapsed lung causes a sudden increase in the negative intrapleural pressure. This draws fluid from pulmonary capillaries into the parenchyma. Experiments have demonstrated that expansion of lung in negative chamber result in rapid increase in pulmonary capillary pressure and blood flow[9]. When lung collapse secondary to any cause is prolonged, the pulmonary capillary endothelial cells undergo anoxic damage and are unlikely to withstand this increased pressure, pulmonary edema develops at a decreased threshold[3].

We believe that in an intracranial procedure, such as in our case, could contribute to accelerate capillary endothelial damage and altered permeability. Large blood loss and fluid shift and relative hypoproteinemia, made it easy for free fluid and plasma to move through the intercellular junctions of already damaged pulmonary capillaries. In addition to this cellular insult, was the rapid evacuation of the air and subsequent rapid expansion of the lung using a wide bore intercostals drain. It is assumed that all these factor acted in conjunction to develop this clinically overt pulmonary edema.

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