

Neurogenic pulmonary edema following intracranial coil embolization for subarachnoid hemorrhage

-A case report-

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Neurogenic pulmonary edema (NPE) is a well-known complication of acute central neurologic injury, particularly aneurysmal subarachnoid hemorrhage. Both increased intracranial pressure and severe over-activation of the sympathetic nervous system seem to be pathogenetic for the onset of NPE. Although intracranial endovascular therapy is minimally invasive, it may affect brain stem regions and result in sympathetic activation. We now report the case of a 70-year-old woman who suddenly developed pulmonary edema during coil embolization of a ruptured aneurysm. During the intervention, oxygen saturation declined suddenly and a chest radiograph revealed pulmonary edema. The delayed appearance of NPE in this patient implies a risk for sympathetically mediated NPE during endovascular therapy. (Korean J Anesthesiol 2012; 63: 368-371)

Key Words: Endovascular procedure, Pulmonary edema, Subarachnoid hemorrhage.

Pulmonary edema can be defined as abnormal accumulation of extravascular liquid in the lungs. It leads to impaired gas exchange and may cause respiratory failure. Acute pulmonary edema can also occur in patients with brain injury, and it is called neurogenic pulmonary edema (NPE). NPE is an acute life-threatening complication associated with many forms of central nervous system injury, such as brain or spinal cord hemorrhage, trauma, tumors, epilepsy, or infections [1-4]. NPE usually appears within minutes to hours after injury and has a high mortality rate if not recognized and treated appropriately [3].

Detachable coil embolization of intracranial aneurysms has proved to be a safe and effective treatment. It has been shown to produce superior survival over craniotomy and clipping for selected ruptured intracranial aneurysms [5]. Despite the minimally invasive nature of this procedure, NPE has previously been reported during endovascular therapy complicated by subarachnoid hemorrhage (SAH) [6,7].

We now report a case of a patient undergoing coil embolization who suddenly developed pulmonary edema.

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Case Report

A 70-year-old woman with a history of hypertension for 10 years was found comatose in the bathroom. The patient was about 150 cm tall, and weighed 50 kg. On arrival at the emergency department, her Glasgow Coma Scale was 6 and her vital signs were blood pressure 160/100 mmHg, heart rate 65 beats/min, and oxygen saturation 91%. Her pupils were fixed with sluggish reaction to light. Her trachea was intubated by emergency physicians and the lungs were mechanically ventilated. Respiratory parameters included a tidal volume of 400 ml, a respiratory rate of 15 breaths/min, and an inspired oxygen concentration (FiO_2) of 100%. A chest radiograph showed right bronchial intubation and subsegmental atelectasis in the left lower lung field. The tracheal tube then was withdrawn about 3 cm. Her electrocardiogram (ECG) showed a prolonged QT interval and arterial blood gas analysis yielded a pH value of 7.42, a partial carbon dioxide pressure (PCO_2) of 26.8 mmHg, a partial oxygen pressure (PO_2) of 303 mmHg, a base excess of -5.1, an HCO_3^- concentration of 17.8 mmol/L, and an oxygen saturation of 99.3%.

Computed tomography of her brain showed global cerebral edema and massive SAH with intraventricular hemorrhage, corresponding to Fisher's grade 4 (Fig. 1). Computed tomographic angiography showed a saccular aneurysm involving the distal portion of the basilar artery. Clinically, the patient was presented as Hunt and Hess grade 5. An emergency intracranial detachable coil embolization and external ventricular drainage were planned.

Four hours from the beginning of mechanical ventilation, the patient arrived at the intervention room for intracranial endo-

vascular therapy. Standard monitors were put in place and her endotracheal tube was connected to the anesthetic machine. Blood pressure was 111/78 mmHg, heart rate was 82 beats/min, and oxygen saturation was 100%. Anesthesia was induced with thiopental 200 mg and rocuronium 50 mg, and an arterial catheter was inserted in the right radial artery. Anesthesia was maintained with isoflurane 0.6–1 vol% with air 2.5 L/min and O_2 1.5 L/min ($\text{FiO}_2 = 50\%$). The end tidal CO_2 (ETCO_2) partial pressure was monitored by capnogram and maintained at 30–35 mmHg.

The coil embolization was successfully performed after 2 hours. Immediately following the endovascular procedure, oxygen saturation was suddenly decreased to 93% and peak inspiratory pressure was increased to 22 cmH_2O . Her blood pressure was increased to 153/85 mmHg and heart rate was 85 beats/min. The location of the endotracheal tube was confirmed by auscultation and mild crackle was heard in both lung fields. In addition, a pink frothy secretion was suctioned through the endotracheal tube. Arterial blood gas analysis demonstrated a pH value of 7.39, PCO_2 of 30.2 mmHg, PO_2 of 72.8 mmHg, and saturation of 93.8% while the patient's lungs were ventilated with 100% oxygen. Pulmonary edema was suspected, and chest radiography showed diffuse bilateral infiltrates (Fig. 2). The patient had received 500 ml of normal saline and urine output was 300 ml during the procedure.

She was then brought to the operating room for external ventricular drainage for treatment of hydrocephalus. On arriving at the operating room, blood pressure was 143/75 mmHg, heart rate was 83 beats/min, and oxygen saturation was 95%. Anesthesia was maintained with sevoflurane 1 to 1.5 vol% with O_2 5 L/min. A central venous catheter was inserted in the

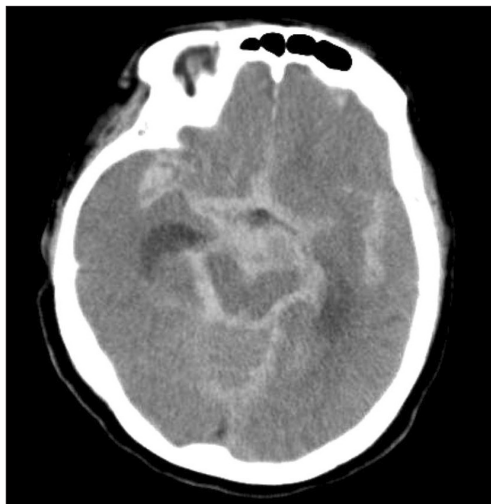


Fig. 1. Computed tomography imaging of the brain reveals a massive subarachnoid hemorrhage and intraventricular hemorrhage.

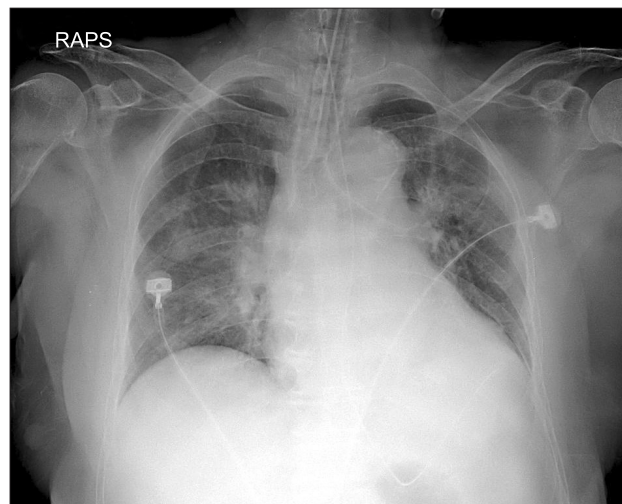


Fig. 2. Chest radiograph shows diffuse bilateral infiltrates in the lung indicating the presence of pulmonary edema.

left subclavian vein by surgeon and the initial central venous pressure was 7 mmHg. The pulmonary edema was treated with intravenous 10 mg of furosemide and a positive end expiratory pressure (PEEP) of 10 cmH₂O.

External ventricular drainage produced bloody cerebrospinal fluid and the initial intracranial pressure (ICP) was about 15 mmHg (normal intracranial pressure = 7 to 15 mmHg). Forty five minutes after application of PEEP with 100% oxygen and external ventricular drainage, her oxygen saturation increased to 100%. The overall anesthetic time was four hours and the patient received 1,200 ml of normal saline and had a urine output of 1,400 ml.

The patient was admitted to the intensive care unit for pharmacologic and ventilatory support. An immediate postoperative arterial blood gas analysis showed a pH value of 7.37, PCO₂ of 32.5 mmHg, PO₂ of 99.3 mmHg, and oxygen saturation of 97%. Postoperative echocardiography showed preserved left ventricle systolic function (left ventricle ejection fraction = 65%) and no significant segmental wall motion abnormalities. Within 72 hours, her respiratory status improved with a resolution of the pulmonary edema (Fig. 3). However, her consciousness level was not improved and she was transferred to another hospital for conservative management after a prolonged hospital stay.

Discussion

NPE occurs in 8–23% of patients with SAH [3,8]. The incidence of NPE was significantly higher in patients with clinically and radiologically severe bleeding and in those with a ruptured aneurysm in the posterior cerebral circulation, leading to an acute rise of ICP in the vicinity of the medulla oblongata [8].

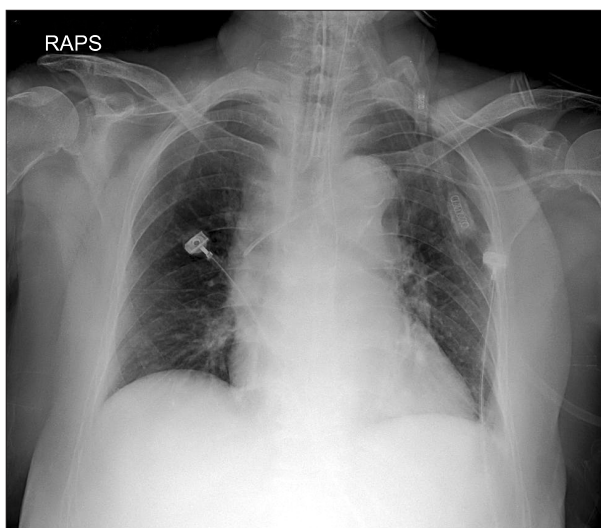


Fig. 3. Chest radiograph shows resolution of the pulmonary edema at postoperative day 3.

Patients with NPE have a high mortality rate, likely due to the severe grade of the bleeding [8].

The clinical symptoms for NPE are nonspecific and often include dyspnea, tachypnea, tachycardia, cyanosis, pink frothy sputum, crackles, and rales on physical examination. The chest radiograph shows bilateral infiltrates and a normal-sized heart [1-4].

The differential diagnoses include aspiration pneumonia, ventilator-associated pneumonia, and ventilator induced pulmonary injury. For this patient, myocardial ischemia or pulmonary embolism was also assumed. However, pulmonary edema was occurring shortly after SAH and there were no signs of a primary cardiovascular event, no ECG changes, and no suspicion of aspiration. Therefore, NPE was diagnosed.

Although several pathophysiologic mechanisms have been implicated in the development of NPE, the exact cascade leading to its development remains unclear. Either a sudden increase of ICP or localized ischemic insult of certain brain trigger zones (hypothalamus and medulla) seem to be pathogenetic for the onset of NPE [3,9,10]. A sudden increase in ICP induces an α -adrenergic catecholaminergic response, which, in turn, causes transient but dramatic increases in both pulmonary and systemic vasoconstriction in the veins or arteries. The resultant striking increase in pulmonary vascular pressure causes an alteration of the Starling forces in the lungs, with a subsequent shift of fluid into the pulmonary alveoli and the interstitial spaces.

It is not clear whether the NPE in this case was caused by changes in ICP or by sympathetic stimulation induced by intracranial endovascular manipulation. Muroi et al. [8] showed that elevated ICP values were found in 67% of the patients with NPE and the ICP values of the other 33% of the patients did not increase. It is suggested that NPE can occur without elevated ICP, even though increased ICP is an important risk factor for developing NPE.

In this case, her ICP checked during external ventricular drainage was high, but within the normal range. In addition, her lungs were hyperventilated during external ventricular drainage (PaCO₂ = 30.2 to 32.5 mmHg). Considering these facts, the possibility of NPE is present, although the ICP did not increase.

During endovascular procedure, cerebral artery may be distended or pulled by catheter or guide wire manipulation. Brewer and Borel [7] demonstrated that endovascular balloon inflation might affect brain stem regions regulating sympathetic outflow and contribute to the development of NPE. The location of the bleeding source and bleeding pattern in correlation with the medulla oblongata is important, because stimulation of the medulla is essential for reactive sympathetic activation [8]. In this case, NPE was developed immediately after coil embolization of aneurysm at the basilar artery which was near

the medulla. And oxygen saturation was suddenly decreased accompanied by an increase in arterial blood pressure (reached 153/85 mmHg from baseline 111/78 mmHg). For these reasons, it must be considered that the endovascular manipulation of coil embolization might result in sympathetic stimulation and eventually develop NPE.

The management of NPE is based on the rapid control of the triggering central neurologic insult and prompt reduction in intracranial pressure. It is also based on conventional treatment of pulmonary edema [3,4]. Tracheal intubation, controlled ventilation with supplemental oxygen, moderate PEEP, and diuresis are essential in optimization of pulmonary status. PEEP values lower than 15 cmH₂O have been shown not to impede the cerebral perfusion pressure [11]. Depending on the severity of the primary insult, NPE may resolve in 48–72 hours with appropriate treatment, and patient prognosis is then dependent on the neurologic injury [12]. In this case, the patient received external ventricular drainage implantation, ventilator support with PEEP, and pharmacologic therapy. As a result, oxygen saturation increased rapidly and the respiratory status was recovered within 72 hours. However, her mental status was not improved.

This is a report of NPE following an endovascular coil embolization for SAH. The combination of a central neurologic insult and the endovascular procedure likely led to the NPE. If not recognized and treated appropriately, NPE can lead to acute cardiopulmonary failure with consequent global hypoperfusion and hypoxia. Therefore, awareness of and knowledge about the occurrence, clinical presentation, and treatment of NPE is essential.

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