Neurogenic pulmonary edema after rupture of intracranial aneurysm during endovascular coiling

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ABSTRACT

Neurogenic pulmonary edema (NPE) is a well-known entity, occurs after acute severe insult to the central nervous system. It has been described in relation to different clinical scenario. However, NPE has rarely been mentioned after endovascular coiling of intracranial aneurysms. Here, we report the clinical course of a patient who developed NPE after aneurysmal rupture during endovascular surgery. There was significant cardiovascular instability possibly from stimulation of hypothalamus adjacent to the site of aneurysm. This case highlights the predisposition of minimally invasive procedures like endovascular coiling to life-threatening complications such as NPE.

Key words: *Aneurysmal rupture, endovascular coiling, intracranial aneurysm, neurogenic pulmonary edema, subarachnoid hemorrhage*

INTRODUCTION

Neurogenic pulmonary edema (NPE) is a well-known entity since the first clinical report published in 1908 by Shanahan.[1] It occurs after acute severe insult to the central nervous system. Rapidity of its onset complicates the overall clinical outcome. Among the several pathophysiological mechanisms proposed, the release of vasoactive substances and severe sympathetic discharge are thought to be the major culprits.[2] NPE has been described in relation to different clinical scenario. However, its occurrence after endovascular surgery has rarely been mentioned.[3] Endovascular coiling of intracranial aneurysms is undergoing major developments. The procedure has an acceptable morbidity and mortality rates, as well as an efficacy comparable with that of surgical clipping.[4] However, the intra-procedural rupture of aneurysm is one of the most feared complications in the interventional neuroradiology suite, and needs early recognition and management. Here, we report the clinical course of a patient who developed pulmonary edema after aneurysmal rupture during endovascular coiling.

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CASE REPORT

A 35-year-old female was admitted with complaints of left-sided headache and progressive loss of vision over a period of 18 months. She was conscious with a heart rate of 70 beats/min and blood pressure of 120/70 mmHg. Neurological examination revealed no other sensory or motor deficits. Other systemic evaluation was unremarkable. Computed tomographic of head showed a partially thrombosed aneurysm and digital subtraction cerebral angiography showed partially thrombosed anterior communicating artery aneurysm. Hence, endovascular coiling of the aneurysm under general anesthesia was planned. In the interventional neuroradiology suite, routine monitors such as SpO_2, ECG, and non-invasive BP were connected to the patient. Anesthesia was induced with intravenous (IV) propofol 3 mg/kg and fentanyl 2 μg/kg. Tracheal intubation was facilitated with rocuronium 1 mg/kg. Maintenance of anesthesia was done with O_2/N_2O mixture (50:50), isoflurane along with intermittent boluses of fentanyl and rocuronium. The patient was subjected to controlled ventilation with an end tidal CO_2 targeted at 35 ± 2 mmHg. Right femoral artery was cannulated using 8 Fr-macrocatheter and heparinization was done using 1 U/kg of heparin. Subsequently, the microcatheter was introduced into cerebral vasculature and the lumen of aneurysm was coiled with Guglielmi detachable coils (GDC, total 3 in number). During placement of the last coil, there occurred sudden bradycardia. The heart rate dropped

down from 86 to 40 beats/min with irregular rhythm. There was associated fall in BP to about 60/46 mmHg for few seconds which was followed by an increase up to 220/126 mmHg. Ventricular ectopics appeared on ECG which was normalized with IV lignocaine. At the same time, extravasation of contrast media, suggestive of aneurysmal rupture, was observed [Figure 1]. The leak was sealed with the same coil. The effects of heparin were reversed with protamine so as to prevent further bleeding. However, the neuromuscular blockade was not reversed and the patient was shifted to intensive care unit (ICU) for elective ventilation in view of aneurysmal rupture. The patient was put on mechanical ventilation with SIMV mode (FiO_2 0.4, tidal volume 500 ml, and respiratory rate 12/min). After 30 min, the patient developed hypotension 70/45 mmHg along with drop in arterial oxygen saturation (SpO_2 85%). Auscultation of chest revealed bilateral basal crepitations. Simultaneously, an increase in airway pressure to 35 mmHg was observed. Endotracheal suction had copious pink, frothy secretions; and hence, pulmonary edema was suspected. Dopamine infusion (10 μg/kg/min) was started for hemodynamic stabilization; however, it was ineffective. Hence, infusion of dobutamine (5 μg/kg/min) and noradrenaline (0.1 μg/kg/min) were started. Central venous catheterization via subclavian vein was done, and a CVP of 15 mmHg was recorded. Arterial blood gas (ABG) analysis was done which showed metabolic acidosis (pH 7.12, PaO_2 68 mmHg, $PaCO_2$ 22.5 mmHg, bicarbonate 12.7 meq/l, and lactate 6.6 mmol/l). Chest X-ray revealed diffuse bilateral pulmonary infiltrates [Figure 2a]. Frusemide 20 mg and morphine 5 mg were given IV, every 8 h. The troponin T-test was negative and ECG documented nonspecific T-inversion in leads I, V_1, V_2, and V_3 along with tachycardia. Two-dimensional echocardiography showed a normal left ventricular function with mild diastolic dysfunction. Thus, a diagnosis of neurogenic pulmonary edema was made. The patient required pressure support ventilation along with positive end-expiratory pressure (PEEP) 10 mmHg and a FiO_2 of 0.9 to maintain adequate oxygenation, for 24 h. Inotropic support was tapered off gradually, and the trachea was extubated on the second day after coiling when the chest X-ray [Figure 2b] and ABG picture was improved. On fifth post-procedural day, the patient was discharged with an advice for follow-up.

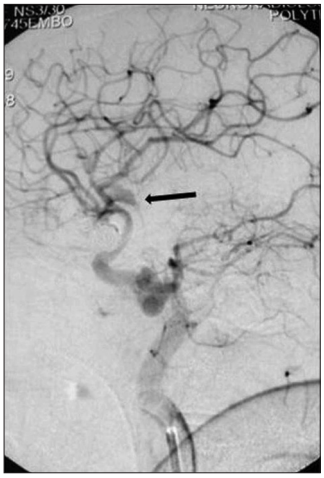

Figure 1: Digital subtraction angiography showing contrast leakage (arrow) suggestive of ruptured anterior communicating artery aneurysm

DISCUSSION

NPE is usually defined as an acute pulmonary edema occurring shortly after a neurologic insult. It can lead to acute cardiopulmonary failure with consequent global hypoperfusion and hypoxia. The etiopathogenesis of NPE is still not clearly understood. Various mechanisms such as neuronal damage directly or indirectly involving the pulmonary vascular bed, over-stimulation of vasomotor centers, activation of trigger zones (neurons in the hypothalamus, brain stem and cervical cord), distortion of brainstem by raised intracranial pressure, have been

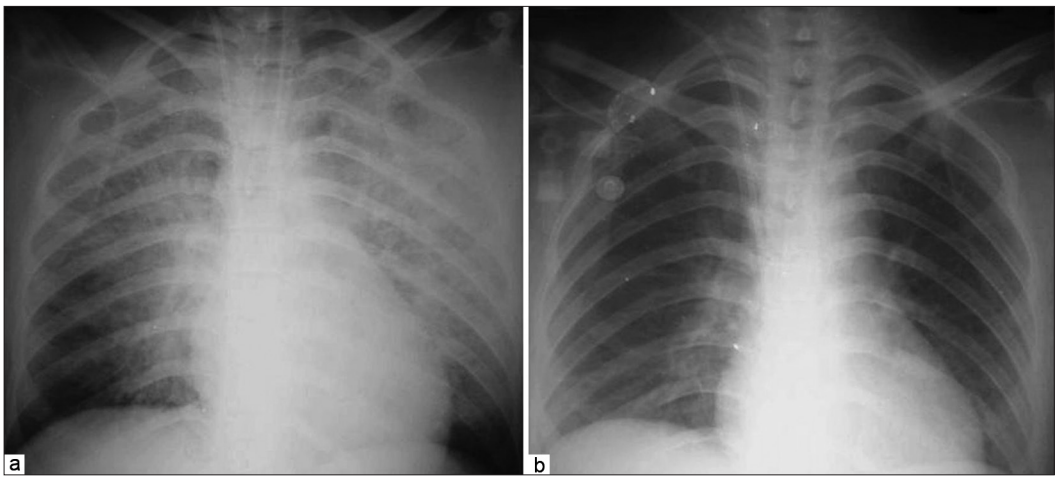

Figure 2: Chest radiography showing diffuse bilateral diffuse pulmonary infiltrates after aneurysmal rupture (a), which resolved 30 h later (b)

proposed.[5] An increase in pulmonary microvascular permeability may play an important role in the development of NPE. Acute cerebral insult causes a local inflammatory mechanism releasing brain cytokines which gain access to the systemic circulation after disruption of blood brain barrier.[5] The resultant systemic inflammatory response affects the lungs to cause leaky pulmonary capillaries. Massive sympathetic discharge (catecholamine surge) causes systemic arterial hypertension, peripheral and pulmonary microvascular vasoconstriction, and stunned myocardium. Severe generalized vasoconstriction leads to shift of intravascular volume from high-resistance systemic circulation to low-resistance pulmonary circulation.[5] The resultant hydrostatic force along with an already increased capillary permeability cause formation of pulmonary edema.

Lighter planes of anesthesia have been implicated for the development of pulmonary edema in animal models.[6] It is hypothesized that an insufficient anesthesia level is not able to inhibit the sympathetic nervous system unlike deeper planes of anesthesia, during an injury of the central nervous system, thus, NPE develops.[7] However, in this patient an adequate depth of anesthesia was maintained throughout the procedure. Other causes of pulmonary edema such as fluid overload were ruled out as there was judicious intra-procedural fluid transfusion. Aspiration pneumonitis which can cause pulmonary edema was unlikely to occur as there was a cuffed endotracheal tube *in-situ*, and the tracheal cultures were sterile. It seems that rupture of aneurysm during the placement of the coil leads to massive sympathetic discharge. Hence, acute hemodynamic alterations occurred, which led to pulmonary edema.

During endovascular procedure, manifestations of aneurysmal rupture can range from visualization of contrast leak into subarachnoid space to massive subarachnoid hemorrhage (SAH) leading to severe intracranial hypertension. Rupture can occur due to high blood pressure at the time of contrast injection, during passage of a guide-wire or a microcatheter, or during placement of an endovascular coil.[8,9] Depending upon clinical severity, coil detachment and subsequent placement of additional coils might be useful, if rupture occurs during the placement of coils. Continuation of the definitive procedure helps most of the patients with treatment-related SAH to survive without serious sequelae.[10] In this case, aneurysmal rupture occurred during placement of the last coil and was managed by continuing with the endovascular procedure. The amount of subarachnoid blood released was minimal, but it resulted in significant cardiovascular instability possibly from stimulation of hypothalamus adjacent to the site of lesion. As the aneurysm lumen was completely occluded, the effects of heparin were reversed with protamine.

The treatment of NPE is usually supportive based on classical pulmonary edema therapies. The goal is to maintain adequate oxygenation, avoid high inflation pressure, use of PEEP to prevent compression atelectasis and, nonetheless, to maintain cerebral perfusion pressure. Unlike other forms of pulmonary edema, NPE typically resolves early, within 48-72 h, with appropriate treatment. A similar course of illness was witnessed, in this patient, as the pulmonary edema was resolved within 30 h. To conclude, this case highlights yet another case of NPE developed after a minimally invasive procedure like endovascular coiling. Intra-procedural vigilance and careful postoperative management helped in diagnosis and successful management of this potentially life-threatening condition.

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