Endovascular Treatment for Aneurysmal Subarachnoid Hemorrhage with Neurogenic Pulmonary Edema in the Acute Stage

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ABSTRACT

AIM: Severe neurogenic pulmonary edema (NPE) can occur in a variety of brain insults, including subarachnoid hemorrhage (SAH), and severe case of NPE can cause devastating consequences. But the literature on the treatment strategy about aneurysmal SAH with NPE is very scant. We present that SAH patients with severe NPE, who were treated first by embolization of aneurysm followed by insertion of lumbar spinal drainage, had comparatively good outcome.

MATERIAL and METHODS: We present 12 consecutive cases of aneurysmal SAH with NPE in the acute stage, which were treated by endovascular treatment between April 2002 and December 2012. We classified the patients according to the Hunt and Hess grading system as follows: grade-3 (1 patient), grade-4 (4 patients), and grade-5 (7 patients). All patients needed respiratory management, with the assistance of a ventilator, and underwent endovascular treatment for the ruptured aneurysms within 72 hours from onset. For all the patients, immediately after the endovascular treatment, we performed lumbar spinal drainage.

RESULTS: The pulmonary edema disappeared rapidly after respiratory management and endovascular treatment. The outcomes were as follows: good recovery (GR; 3 patients), moderate disability (MD; 4 patients), severe disability (SD; 3 patients), and death (D; 2 patients). Five patients (42%) developed pneumonia, and we postponed extubation until recovery from pneumonia. The cause for severe disability and death was symptomatic vasospasm and primary brain damage. No patients had rebleeding from ruptured aneurysms.

CONCLUSION: Endovascular treatment for ruptured aneurysm and placement of lumbar spinal drainage is an excellent treatment option for severe SAH with NPE.

KEYWORDS: Neurogenic pulmonary edema, Endovascular treatment, Aneurysm, Subarachnoid hemorrhage
by insertion of lumbar spinal drainage, had comparatively
good outcome (10). We now have experience in performing
this procedure on 12 consecutive cases thus far, including
the 4 SAH cases mentioned. To identify the usefulness of
this treatment, in this present study, we retrospectively
investigated the clinical characteristics and outcome in those
12 patients with SAH and NPE.

**MATERIAL and METHODS**

Four hundred and sixty-six patients with aneurysmal SAH
underwent treatment in our hospital between April 2002 and
December 2012. Computed tomography (CT) scan was used
to diagnose SAH. Of the 466 patients, 13 (2.8%) showed
severe NPE. Severe NPE include the presence of all of the
following criteria: (i) immediate onset of respiratory failure after
ictus; (ii) presence of crackles, which suggested fluid in the
lungs by chest auscultation, and frothy pink tracheal fluid;
(iii) requirement of endotracheal intubation and respiratory
management with a mechanical ventilator; (iv) chest X-ray
film on admission revealing interstitial edema in bilateral lung
fields; and (v) no previous history of cardiac and lung disease.

After the diagnosis of SAH with severe NPE, all patients
underwent endovascular treatment for ruptured aneurysms
under general anesthesia, following diagnostic angiography,
as soon as the respiratory condition became stable, as judged
by the anesthesiologist. After the endovascular treatment, we
placed continuous lumbar spinal drainage for early discharge
of SAH and control of intracranial pressure (ICP).

**RESULTS**

Of the 13 patients who presented with SAH and severe NPE,
12 were treated by endovascular treatment within 3 days of
ictus, and 1 patient, with intracerebral hematoma, was treated
by surgical clipping and removal of hematoma. Figure 1 shows
the initial brain CT scan of the 12 patients. All patients had
diffuse SAH and 3 of 12 patients showed enlargement of lateral
ventricles, but every patient did not have so much volume of
SAH and they had no intracerebral hematoma (Figure 1). Four
of 12 patients (cases 2, 3, 6, and 7) were previously reported
(10). Mean age of the 12 patients was 52 (range, 31–74) years,
and 6 patients (50%) were male. The Hunt and Hess grades
on admission were as follows: grade-3 (1 patient), grade-4 (4
patients), and grade-5 (7 patients). The locations of aneurysm
in the 12 patients (in parentheses) were as follows: basilar
top (2), posterior communicating artery (1), middle cerebral
artery (1), anterior cerebral artery (1), anterior communicating
artery (2), basilar-anterior inferior cerebellar artery (1), internal
carotid-posterior communicating (2), and vertebral artery
dissecting aneurysm (2). We treated the 2 patients with
vertebral artery dissecting aneurysm by internal trapping of
vertebral artery, and the remaining 10 patients by aneurysmal
sac embolization. All patients required early intratracheal

![Figure 1: Initial head CT scans of 12 patients shows diffuse subarachnoid hemorrhage.](image-url)
intubation and successfully underwent endovascular treatment following diagnostic angiography within 72 h from onset of ictus. Immediately after the endovascular treatment, we placed continuous lumbar spinal drainage for early discharge of SAH and control of ICP. All patients required respiratory management with a mechanical ventilator. Pulmonary edema disappeared in all patients within 6 days, but 5 patients (42%) developed complicated pneumonia. We postponed extubation until recovery from pneumonia, and 1 patient had tracheotomy for pneumonia and prolonged disturbance in consciousness. We maintained the continuous lumbar spinal drainage for 7 to 14 days (mean 10 days). Three of the patients achieved good recovery, 4 patients suffered mild disability, 3 patients suffered severe disability due to the symptomatic vasospasm, and 2 patients died of primary brain damage after 6 and 9 days, respectively, from onset. Mean follow-up periods were 38 months, and no aneurysms reruptured in this period.

**ILLUSTRATIVE CASE**

**Case 9**

A 51-year-old man was admitted to our hospital with a sudden onset of unconsciousness. He presented with coma and respiratory dysfunction with pinkish foamy sputum on admission. CT scan revealed diffuse subarachnoid hemorrhage, and a chest roentgenogram revealed diffuse pulmonary infiltrates in the bilateral lung field indicating the presence of pulmonary edema (Figure 2). He was also intubated immediately after admission and was controlled by continuous positive-pressure ventilation with positive end-expiratory pressure. Angiography was performed under general anesthesia after 4 h of onset. Left vertebral angiogram revealed a saccular aneurysm at the top of the basilar artery (Figure 3A). The patient underwent endovascular embolization with detachable coils following diagnostic angiography. Complete occlusion of the aneurysm was performed (Figure 3B). Immediately after the embolization, continuous lumbar spinal drainage was inserted and placed for 14 days.

**Figure 2:** Chest X-ray reveals diffuse pulmonary infiltrates in the lung indicating the presence of pulmonary edema.

**Figure 3:** A) 3D-digital subtraction angiography of the right vertebral injection reveals top of basilar artery aneurysm. B) The aneurysm was completely embolized by detachable coils.
The pulmonary edema findings disappeared rapidly after endovascular treatment, but aspiration pneumonia developed as a complication. Therefore, extubation was done after 8 days from onset. Serial chest roentgenograms revealed complete resolution on the tenth day. After removal of the spinal drainage, hydrocephalus developed. Ventriculoperitoneal shunt was performed on day 24, but neurological recovery was incomplete, and recent memory disturbance was present due to primary brain damage. CT scan on the 40th day from onset revealed mild brain atrophy (Figure 4).

**DISCUSSION**

NPE is usually defined as an acute pulmonary edema occurring shortly after neurogenic insult (3,9). Frequent causes of NPE are traumatic head injury or aneurysmal SAH. Several mechanisms have been implicated in the pathogenesis of NPE. Sudden increase in ICP induces massive sympathetic discharge (catecholamine surge) that causes systemic arterial hypertension and peripheral and pulmonary microvascular vasoconstriction. Severe generalized vasoconstriction leads to a shift of intravascular volume from high-resistance systemic circulation to low-resistance pulmonary circulation. The resultant hydrostatic force along with an already increased capillary permeability causes formation of pulmonary edema (5,7,12). However, the exact interactions remain unknown.

Muroi, et al. reported that patients with NPE presented significantly more often with clinically severe hemorrhage compared with patients without NPE (11). Nevertheless, there have been few reports regarding the best management for aneurysmal SAH with NPE in the acute stage. There is a therapeutic dilemma between the policy of early aneurysm surgery and the successful management of pulmonary edema during radical intervention. Yabumoto et al reported a case of aneurysmal SAH with NPE (14). They insisted on early and aggressive management of the ruptured aneurysm and NPE, and proposed that the syndrome of NPE should not be an obstacle to radical intervention when cardiorespiratory control can maintain the minimal anesthetic limit. In the present study, the authors also carried out an early and aggressive management of the ruptured aneurysms. Although 11 of 12 patients (92%) were poor grade SAH (Hunt and Hess grade IV or V), 7 of 12 patients (58%) were good recovery or mild disability in Glasgow Outcome Scale at discharge. This is quite a good outcome. The initial head CT scan showed that every patient did not have so much volume of SAH, and NPE might worsen the SAH grading. Even a little volume of SAH might cause severe NPE.

Although the conventional treatment of pulmonary edema are tracheal intubation, controlled ventilation with supplemental oxygen, moderate positive end-expiratory pressure, diuretics, etc., the essential management of NPE is based on the rapid control of the triggering central neurological insult, and therefore, the primary goal therapeutic approach needs to be focused on decreasing ICP (1). Recently, endovascular treatment has been recognized as a less invasive treatment for cerebral aneurysm when compared to direct surgical clipping (4). The most important advantages of endovascular treatment for aneurysmal SAH are that it can immediately follow the diagnostic angiography and that the period required for intervention is shorter than that for direct clipping (4). Lumbar spinal drainage also can be performed in a shorter time than ventricular drainage. Rapidly control of ICP after endovascular treatment could contribute to recovery from NPE. We therefore suggest that early endovascular intervention for ruptured aneurysm followed by lumbar drainage of cerebrospinal fluid for decreasing ICP should be the first choice for poor-grade aneurysmal SAH with NPE.

In the acute stage, unconscious patients are at risk of aspiration of gastric contents, or using of ventilation for treatment of NPE might induce nosocomial infection. In either event, the patients of aneurysmal SAH with NPE might develop pneumonia. Of 12 patients, 5 (42%) developed pneumonia after improvement of NPE, and we postponed extubation until recovery from pneumonia. Fortunately, all 5 patients were successfully treated with antibiotics. However, pneumonia is a severe complication that must not be ignored.

**CONCLUSION**

Early endovascular treatment of ruptured aneurysm and placement of lumbar spinal drainage is an excellent treatment option for severe SAH with NPE.

**REFERENCES**