

# Effect on Shunt-dependent Hydrocephalus after Coil Embolization for Aneurysmal Subarachnoid Hemorrhage

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## 뇌동맥류성 지주막하출혈에 대한 코일 색전술 후 발현되는 단락술 의존 수두증에 대한 효과

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**목 적 :** 코일 색전술로 파열성 뇌동맥류를 치료한 후 발현되는 단락술 의존 수두증에 대한 효과를 평가하고자 한다.

**방 법 :** 2004년 8월부터 2009년 2월까지 코일 색전술로 파열성 뇌동맥류를 치료한 연속적인 112명의 환자를 대상으로 후향적으로 분석하였다. 저자들은 주로 파열성 뇌동맥류를 코일 색전술로 치료한 후 발현되는 단락술 의존 수두증의 관련 인자를 분석하였다.

**결 과 :** 109명(97.3%)의 환자에서 단락술 의존 수두증은 발현되지 않았다. 이 중 4명의 환자에게서 일시적인 뇌실배액술로 급성기의 수두증을 치료하기는 하였지만, 영구적인 단락술은 필요하지 않았다. 전체적으로 3명(2.6%)의 환자에게서 영구적인 단락술이 필요하였고, 이 중 2명(1.7%)의 환자는 일시적인 뇌실배액술이 설치하였다.

**결 론 :** 코일 색전술로 파열성 뇌동맥류를 치료한 경우 낮은 빈도의 단락술 의존 수두증이 발현되는데, 이는 지주막하수조에 보다 작은 손상과 연관이 있는 듯하다. 이 사실은 파열성 뇌동맥류의 치료방침과 장기 결과에 영향을 미칠 수 있을 것으로 사료된다.

**중심 단어 :** 코일 색전술 · 수두증 · 단락술.

## Introduction

The incidence of hydrocephalus, a known complica-

tion of aneurysmal subarachnoid hemorrhage (SAH), has been reported to range from 6 to 67%, either early in the course (acute, during the first 3 days, or subacute, days 4-13) or after the first 2 weeks (chronic)<sup>2)3)5)8)10)24)</sup>.

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Poorer neurological outcomes and cognitive deficits have been cited by many authors as some of the adverse outcomes of hydrocephalus. Several factors have been associated with the development of hydrocephalus requiring permanent cerebrospinal fluid (CSF) diversion. Factors such as increasing age, female sex, poor admission Hunt & Hess grade, diffuse or thick subarachnoid blood on the computed tomography (CT) reflected on Fisher's grade, rebleeding, intraventricular hemorrhage, anterior communicating artery aneurysms, posterior circulation aneurysms, acute hydrocephalus and symptomatic vasospasm have been connected with the development of hydrocephalus among patients with SAH<sup>(3)(5)(6)(10)(15)(18)(19)(22)(23)(25)(26)</sup>.

The Guglielmi detachable coils in early 1990s allowed physician to treat directly intracranial aneurysms. This treatment has been accepted by more physicians, especially after a large study that compared clipping to coiling showed favorable results<sup>(4)(7)(17)(20)</sup>. Recently, few studies have examined the effect of aneurysmal subarachnoid hemorrhage after coil embolization on permanent ventricular shunt requirement. Because only surgery is probable to remove blood clot directly, two available treatments, clipping or coiling, may lead to differences in the need for CSF diversion. We report that coil embolization may influence shunt-dependent hydrocephalus after SAH in our experience.

## Materials and Methods

One hundred twelve consecutive patients who underwent endovascular coil embolization treatment of ruptured, nontraumatic, intracranial aneurysms between August 2004 and February 2009, at our hospital, were retrospectively studied. The diagnosis of SAH was made by clinical presentation, CT of head, or lumbar puncture. The diagnosis of a cerebral aneurysm as a cause of SAH was made either by conventional cerebral vessel angiography or cerebral CT angiography. Patients with history of head trauma preceding the development of headache, negative angiograms, primary SAH, or aneurysms measuring more than 20 mm in diameter were excluded from the study. All patients were admitted to the neurosurgical intensive care unit (NICU) before and after aneurysm

obliteration and the ruptured aneurysm was treated within 48 hours after admission. The type of aneurysm treatment was not randomized and selected by neurosurgeons or neuroradiologist on the base of radiological features and patient's condition. Standard SAH management practices were followed in the NICU, which included prevention of rebleeding in the preoperative period, as well as treatment of vasospasm and hydrocephalus in the postoperative period. Perioperatively, all patients underwent lumbar drainage throughout the vasospasm period. For all permanent shunt placement, clinical deterioration should be associated with dilatation of the ventricles in radiological images because of exclusion of vasospasm or other medical problems. To determine the effects of various factors and treatment modality on the development of hydrocephalus, we investigated the factors such as 1) age, 2) sex, 3) admission Hunt and Hess grade, 4) Fisher's grade, 5) intraventricular hemorrhage, 6) intracerebral hematoma 7) hydrocephalus on admission CT, 8) ruptured aneurysmal location,

## Results

One hundred twelve patients were included in this

**Table 1.** Characteristics of patients treated with coil embolization for ruptured intracranial aneurysms

	Shunt (n=3)	Non Shunt (n=109)
Mean age (yr)	64.3	55.1
Sex (male %)	66.6 (n=2)	41.2 (n=45)
Hunt & Hess grade	2.6	3.1
Fisher's grade	3	3.2
IVH (%)	33.3	7.3 (n=8)
ICH (%)	–	13.7 (n=15)
Acute HCP (%)	66.6 (n=2)	3.6 (n=4)
Aneurysmal location		
ICA	–	6
ACA	2	6
MCA	–	34
ACOM	–	19
PCOM	1	24
Posterior circulation	–	20

ICA : internal carotid artery, MCA : middle cerebral artery, ACA : anterior cerebral artery, PCOM : posterior communicating artery, ACOM : anterior communicating artery, IVH : intraventricular hemorrhage, ICH : intraventricular hemorrhage, HCP : hydrocephalus

study. Patient ages ranged 22 to 82 years (mean age ; 56.3 years). The study population consisted of 47 male and 65 female patients. One hundred nine patients (97.3%) did not develop shunt dependent hydrocephalus. Even if 4 patients developed the acute hydrocephalus treated with temporary external ventricular drainage, they never needed permanent shunt diversion. Overall three (2.6%) patients required permanent shunt diversion. Among them, temporary external ventricular drainage was plac-

ed in two (1.7%) patients. The overall mortality rate was 4.1%. The mortality rate for patients who underwent shunt procedure was 0%.

Factors associated with shunt dependent hydrocephalus were demonstrated by 1) Hunt & Hess grade, (Table 3) 2) Fisher's grade, 3) intraventricular hemorrhage, 4) acute hydrocephalus, and 5) intracerebral hematoma. Table 1. and 2 presents the clinical characteristics of endovascularly treated patients.

## Discussion

### 1. Pathogenesis (mechnism) of post-SAH hydrocephalus

Diverse theories have been proposed to explain the pathogenesis of post-SAH hydrocephalus. Acute hydrocephalus is thought to result from blockage of the CSF flow. That is, blockade within the ventricular system at alus can but does not necessarily lead to chronic hydrocephalus. the present data show that the probability of

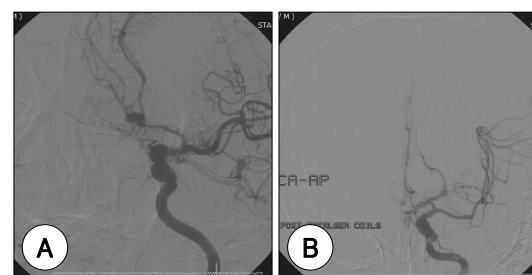
**Table 2.** Outcomes of patients treated with coil embolization for ruptured intracranial aneurysms and permanent shunt operation (n=3)

	case 1	case 2 (Fig. 1, 2)	case 3 (Fig. 3, 4)
Age (yr)	53	71	69
Sex	Male	Male	Female
Hunt&Hess grade	3	3	2
Fisher grade	3	3	4
IVH	-	-	+
Acute HCP	-	-	-
Aneurysmal location	ACOM	ACOM	PCOM
GOS	5	5	5

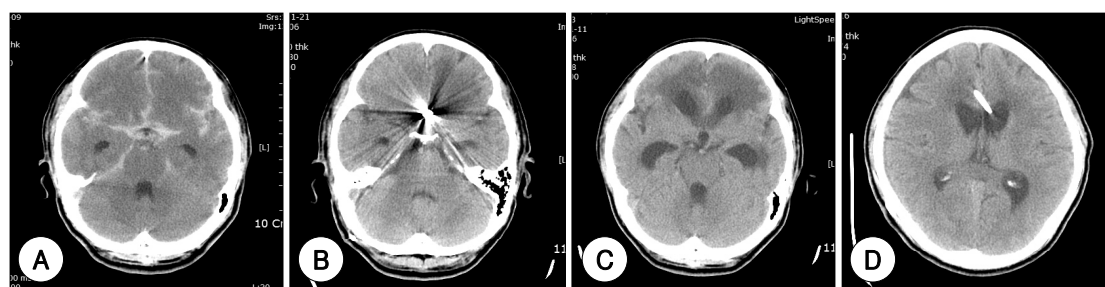
ICA : internal carotid artery, MCA : middle cerebral artery, ACA : anterior cerebral artery, PCOM : posterior communicating artery, ACOM : anterior communicating artery, IVH : intraventricular hemorrhage, ICH : intracerebral hemorrhage, HCP : hydrocephalus

**Table 3.** Incidence of patients : correlated with Hunt & Hess grade

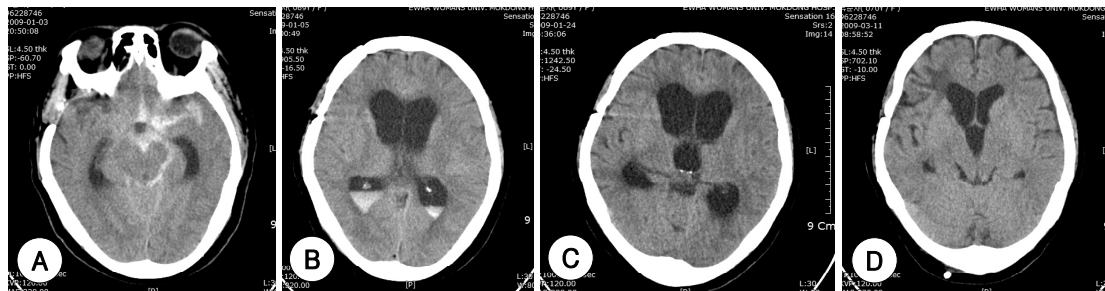
Hunt & hess grade	No.of cases (%)
I	2(2)
II	48(44)
III	50(45.8)
IV	8(7.3)
V	1(1)



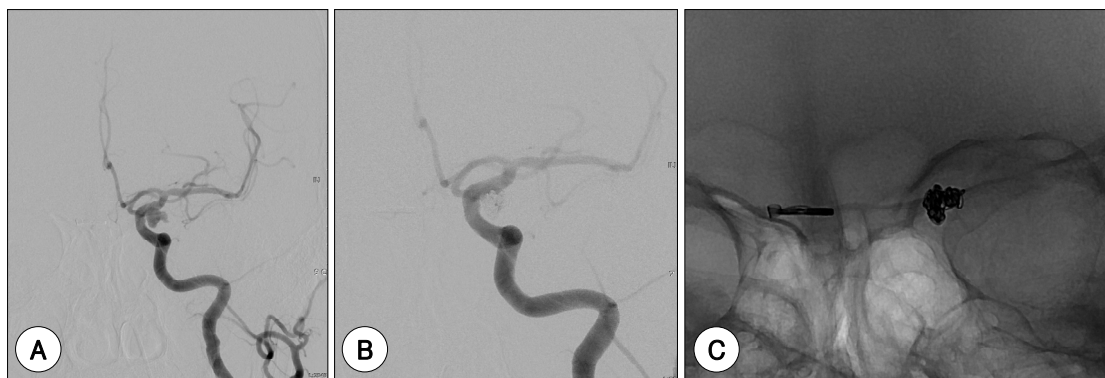
**Fig. 2.** (case 2) : Cerebral angiographic findings A : cerebral angiography shows saccular aneurysm in anterior communicating artery. B : postembolization angiography shows obliteration of aneurysm.



**Fig. 1.** (case 2) : 53-aged male patient admitted with sudden headache A : computed tomography(CT) shows diffuse subarachnoid hemorrhage in basal cistern. B : CT demonstrate the coil artifact in anterior communicating artery aneurysm. C : one month later, CT shows hydrocephalus with periventricular edema. D : CT shows that ventriculo-peritoneal shunt is performed.



**Fig. 3.** (case 3) : 69-aged female patient admitted with loss of consciousness A : computed tomography(CT) shows diffuse subarachnoid hemorrhage in basal cistern. B : CT demonstrate intraventricular hemorrhage in both lateral ventricle. C: 40 days later, CT shows communicating type of hydrocephalus. D : CT shows reduced ventricle size after ventriculoperitoneal shunt.



**Fig. 4.** (case 3) : Cerebral angiographic findings A : cerebral angiography shows saccular aneurysm in posterior communicating artery. B : post-embolization angiography shows obliteration of aneurysm. C : right side posterior communicating artery aneurysm is treated with clipping in 10 years ago.

increased intracranial pressure (ICP) during the first 24 to 48 hours after SAH. that is before early surgery in the present series is high. It seems worth considering that normalization of ICP after ventriculostomy before preparation of the aneurysm makes the brain slack for easy surgical access.

present study is that early evacuation of cisternal blood clots and CSF drainage reduce the risk of shunt-dependent hydrocephalus after SAH. In other studies, shunt-dependent hydrocephalus in unselected series of patients is described as a significant problem with an incidence between 18 and 45%<sup>(1)(2)(8)(9)(15)</sup>. However, Acute hydrocephalus was correlated with chronic hydrocephalus statistically significantly. Chronic hydrocephalus occurs when CSF flow is permanently impeded or absorption is permanently reduced. The pathogenesis of chronic hydrocephalus involves arachnoid adhesions that form because of a meningeal reaction to blood products, impairing

CSF absorption at the arachnoid villi and basal cisterns<sup>(2)(3)(6)(21)(25)(26)</sup>.

The level of increased ICP is correlated between the extent of SAH (amount of extravasated intracranial blood volume) and possible explanation is the following : bleeding from a ruptured aneurysm leads to a reduction or even a cessation of cerebral circulation caused by ICP levels approaching or surmounting systolic blood pressure. The duration of extreme intracranial hypertension and brain hypoxia depends on the amount of blood extravasation. During the cessation of circulation the leak in the aneurysm is sealed by a clot : ICP thereafter probably decreases because of displacement of CSF. Cerebral blood volume consecutively rises as a result of reactive hyperemia after the hypoxic episode A trend toward higher ICP levels in patients with a greater amount of SAH is probably the result of the greater likelihood of compromised CSF circulation caused by tamponade of

the subarachnoid space with clotted blood. In addition to reactive hyperemia metabolically induced vasodilation other than the initial hypoxia may play a role<sup>13</sup>). The effect of intraventricular hemorrhage (IVH) on the development of hydrocephalus has been well established. In our series, 40% of patients who underwent definitive shunt placement also had IVH. Some authors assert that the presence of blood clots and high CSF viscosity can lead to an obstructive form of hydrocephalus and early CSF disturbances. Investigators noted no relationship between the development of hydrocephalus and the number of erythrocytes in the CSF, thus indicating that flow disturbances also play an important role in acute hydrocephalus<sup>26</sup>).

The patients with acute hydrocephalus and impaired consciousness after SAH, in contrast to patients with cerebral ischaemia, have decreased cerebral blood flow predominantly in the basal parts of the brain. Apart from showing that impaired consciousness in acute hydrocephalus results from a disturbance predominantly in the basal parts of the brain, this study may also have practical implications. If a patient has deteriorated from acute hydrocephalus and does not respond to treatment, the explanation may be that insufficient cerebrospinal fluid drainage or another complication such as cerebral ischaemia has developed. The latter is most likely if single photon emission computed tomography (SPECT) scanning shows decreased regional cerebral blood flow elsewhere than in the basal parts of the brain, even if CT shows no evidence of infarction<sup>913</sup>). Some study has found a significant association of a history of hypertension, elevated blood pressure on admission, and hypertension postoperatively with both CT and clinical hydrocephalus. Mechanism involved in the ventricular enlargement seemed to be a combination of at least two factors: One was the possible failure of CSF absorption in the face of increased superior sagittal sinus venous pressure, and the other was the increased intraventricular pulse pressure from the choroid plexus<sup>5</sup>).

Previous studies have demonstrated the negative effects of hydrocephalus on cerebral blood flow as well as clinical deterioration. Hydrocephalus can result in long-term memory problems and the development of psycho-organic disorders. Cognitive deficits have been not-

ed after even slight temporary ventricular dilatation. All outcome studies proved increased morbidity among shunt-treated patients<sup>19</sup>).

## 2. Lumbar drainage and post-SAH Hydrocephalus

The rationale we used lumbar CSF drainage to all patient with ruptured intracranial aneurysms is that it promotes CSF circulation from the ventricles through the subarachnoid space, and that it also takes the bloody CSF from the spinal cistern. This means that CSF drainage from the ventricles may actually promote stasis within the subarachnoid space. So, it may decrease the incidence of vasospasm and shunt-dependent hydrocephalus<sup>611141822</sup>). The serial lumbar puncture provided the CSF blockage is in the subarachnoid space and not in the ventricular system. The risk of infection with this procedure is small, and the fall in CSF pressure is probably more gradual. Whether patients with acute hydrocephalus after subarachnoid hemorrhage really benefit from this simple procedure is presently being investigated<sup>10</sup>). During the early stages after SAH, the removal of a large amount of CSF by lumbar drainage could collapse the subarachnoid space and prevent its irrigation, thus retaining spasmogens in the vicinity of the arteries. lumbar drainage makes it impossible to clear the blood-filled subarachnoid channels completely, and thereby allowing some CSF flow. The reduction in CSF absorption and the increase in its formation that occurs with intracranial hypotension may help induce hydrocephalus. Continuous cerebrospinal fluid drainage should not be performed too readily in patients with SAH in comparison with those with acute hydrocephalus as a result of intraventricular hemorrhage or obstructive hydrocephalus, because the removal of a large amount of CSF induces cerebral vasospasm as well as hydrocephalus. We believe that a degree of intracranial hypertension that restores normal CSF circulation, rather than lumbar drainage, is a better way of removing spasmogenic substances from the subarachnoid space after SAH<sup>12</sup>).

## 3. Coiling and post-SAH hydrocephalus

Dorai et al.<sup>3</sup>) and Varelas et al.<sup>24</sup>) reported that a higher percentage of the coiled patients required permanent shunting, compared with the clipped patients. They thought that irrigation and early evacuation of subarachnoid clots

during microsurgery allow the blood to clear more rapidly, decreasing the probability of shunt-dependent hydrocephalus. But, the results of our study is that coiling is not associated with increased risk of shunt procedure. Major cause of our results not in agreement with those articles may be probably our use of lumbar drainage to all patients with ruptured intracranial aneurysms during perioperative and vasospasm period. Because lumbar drainage is able to remove blood clot in subarachnoid space indirectly, the rate of shunt-dependent hydrocephalus in the coiled treated group is not higher. Based on data in the study, the endovascular treatment did not seem to raise the risk of shunt-dependent hydrocephalus. This finding is in agreement with data in the study conducted by Gruber, et al.<sup>6)</sup>, who showed no difference in shunt dependency in a comparison between the surgical treatment and endovascular treatment groups. In contrast, Dorai, et al.<sup>3)</sup>, demonstrated a higher rate of shunt dependency in the endovascular group. Note, however, that these researchers did not match the endovascular and surgical therapy groups with respect to world federation neurological surgery and Fisher grades and that it is unclear whether the two groups were substantially comparable in terms of admission data. An important question is whether patients treated with endovascular coiling are more inclined than patients who are surgically treated with removal of clots within the basal cisterns to develop hydrocephalus. Recent preliminary studies have shown that patients with ruptured aneurysms who are treated exclusively with endovascular methods had no more vasospasm and/or hydrocephalus than those patients who are surgically treated, with the exception of patients with Fisher Grade 3 or 4<sup>3)6)16)19)24)</sup>.

And, because there was an insignificant trend toward a higher rate of extraventricular drainage (EVD) placement among surgical patients. it seemed that the treatment groups were further biased by a higher pretreatment hydrocephalus rate in the surgical treatment group. However, because EVDs were also placed intraoperatively, i.e., to facilitate dissection at the base of the brain rather than to treat extensive acute hydrocephalus, and because we were more reluctant to place EVDs in patients selected for endovascular treatment with postembolization heparinization, it is reasonable to assume that com-

parisons were not substantially confounded by the higher EVD placement rate in the surgical treatment group<sup>6)</sup>.

#### 4. Limitation of our study

A main pitfall of our study is that small number of shunt dependent hydrocephalus group and lumbar drainage was used to all patients with ruptured intracranial aneurysms. Even if lumbar drainage is the method not to evacuate blood clots directly, this method may remove blood clots indirectly. To evaluate the difference in the need for permanent CSF diversion according to treatment modality. Variable such as lumbar drainage should be evaluated in our opinion. More precise evaluation of shunt-dependent hydrocephalus according to treatment modality will be probable only when we exclude from confounding factor such as lumbar drainage.

## Conclusion

Coil embolization of ruptured intracranial aneurysms may be associated with lower risk for developing shunt-dependent hydrocephalus, possibly by lesser damage for cisternal anatomy. This might effect on long-term outcome and decision making of ruptured intracranial aneurysms. However, a randomized prospective study of treatment modality with or without the use of lumbar drainage will be able to complement the pitfall of our study.

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