The Causal Relationship of the Hydrocephalus in Patients with Aneurysmal Subarachnoid Hemorrhage

저자	Shin, Tae-Sob ; Jung, Chul-Ku ; Kim, Hyun-Woo ; Park, Keung-Suk ; Kim, Jae-Myung
저널명	Journal of Korean neurosurgical society = 대한신경외과학회지
발행기관	대한신경외과학회
NDSL URL	http://www.ndsl.kr/ndsl/search/detail/article/articleSearchResultDetail.do?cn=JAKO200717960233532
IP/ID	183.98.1.217
이용시간	2018/04/24 06:13:50

Clinical Article

Tae-Sob Shin, M.D.
Chul-Ku Jung, M.D.
Hyun-Woo Kim, M.D.
Keung-Suk Park, M.D.
Jae-Myung Kim, M.D.

Department of Neurosurgery
Konyang University Hospital
Daejeon, Korea

J Korean Neurosurg Soc 42 : 173-178, 2007

The Causal Relationship of the Hydrocephalus in Patients with Aneurysmal Subarachnoid Hemorrhage

Objective : Hydrocephalus is one of the major complications following spontaneous subarachnoid hemorrhage (SAH). However, the risk factors of the hydrocephalus after SAH are not still well known. This study was focused on verification of the causal relationships between the development of hydrocephalus and SAH.
Methods : The patients who developed hydrocephalus after rupture of aneurysms were studied. To obtain prognostic factors regarding to hydrocephalus, several parameters such as age, sex, hypertension, location of aneurysm, existence of intraventricular hemorrhage (IVH) and intracerebral hemorrhage (ICH), Glasgow coma scale (GCS), Hunt-Hess SAH classification & Fisher Grade on admission and the ratio of frontal horn of lateral ventricle diameter to skull inner table diameter at this level (FH/ID) were studied retrospectively.
Results : The development of hydrocephalus following SAH is multifactorial. The age, IVH, FH/ID ratio were related to hydrocephalus in analysis. There is a low clinical correlation between sex, hypertension, location of aneurysm, existence of ICH, GCS, Hunt-Hess SAH classification, Fisher Grade on admission and hydrocephalus.
Conclusion : Knowledge on risk factors related to the occurrence of hydrocephalus may help guide neurosurgeons in the long-term care of patients who have experienced aneurysmal SAH.

KEY WORDS : Hydrocephalus · Subarachnoid hemorrhage · Risk factors.

INTRODUCTION

Subarachnoid hemorrhage (SAH) by aneurysmal rupture could cause several complications and the frequency of developing hydrocephalus varies from 6 to 67%[1, 12]. Various treatments may be required including shunt operation for the changed intracranial pressure and worsened neurological symptoms due to the hydrocephalus after the SAH. Therefore, the understanding of the causes of hydrocephalus in SAH patients has a clinically important meaning.

The location of aneurysm has been known to be highly correlated to the development of hydrocephalus due to the higher frequency of intraventricular hemorrhage (IVH) at the time of anterior communicating artery aneurysmal rupture, but it has been known to have no relationship to other location of aneurysms[4,8]. The quantitative correlationship of higher incidence of SAH indicated to show higher incidence of development of hydrocephalus, and the state accompanied with IVH has been reported to cause stronger possibility of hydrocephalus due to the cerebrospinal fluid circulation disorder. It has been reported that the intracerebral hemorrhage (ICH) has no relationship in the development of hydrocephalus[3]. The consciousness state of patients has been reported to have high correlation to the development of hydrocephalus.

The aim of this study was to analyse the prognostic factors and causes of hydrocephalus following subarachnoid hemorrhage.

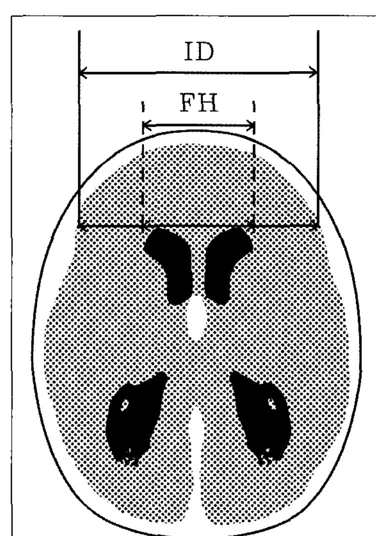

Fig. 1. The estimation of frontal horn/internal diameter of inner table (FH/ID). FH : frontal horn, ID : internal diameter of inner table.

MATERIALS AND METHODS

We conducted a study for 66 patients from January 1, 2004 to December 31, 2005 who underwent

• Received : April 6, 2007
• Accepted : August 10, 2007
• Address for reprints :
Chul-Ku Jung, M.D.
Department of Neurosurgery
Konyang University Hospital
685 Gasuwon-dong, Seo-gu
Daejeon 320-718, Korea
Tel : +82-42-600-8958
Fax : +82-42-544-9500
E-mail : ckjung@kyuh.co.kr

aneurysmal neck clipping among SAH patients. In these patients, parameter such as patients' sex, age, neurological status (consciousness level), degree of SAH amount, location of aneurysm, IVH, ICH, the ratio of frontal horn of lateral ventricle diameter to skull inner table diameter at this level were evaluated for the possible relationship in the development of hydrocephalus.

For the analysis, patients were divided into two age groups; 20-40 of age and 50-70 of age. Analysis of aneurysm was conducted by classifying the regions into the following 4 groups; anterior communicating artery aneurysm, middle cerebral artery aneurysm, posterior communicating artery and the others. For the patients' neurological state, the Glasgow Coma Scale (GCS) and Hunt-Hess SAH Classification were used to classify the consciousness state of patients and SAH was classified by using the Fisher Grade. The ratio of frontal horn of lateral ventricle diameter to skull inner table diameter at this level (FH/ID) was collected based on the maximum ratio of both sides of frontal horn of lateral ventricle and the skull inner table in the same line obtained from the brain computed tomography (CT) (Fig. 1). Patients were divided into two groups, having the ratio of frontal horn of lateral ventricle to skull inner table at this level higher than 40%, and less than 40%. Statistical analysis was conducted by using SPSS 12.0. Between the comparison groups, statistical significance was given to those hydrocephalus causes through the cross analysis when p value is less than 0.05.

RESULTS

Comparison of characteristics by the existence of hydrocephalus development

Age of patients was divided into age groups with 20-40 years old and 50-70 years old. There was higher incidence of developing hydrocephalus in patients older than 50 years compared to patients in younger than 40 years (p=0.031). Although female patients tend to have higher incidence of developing hydrocephalus, it was not statistically significant and the presence of hypertension did not affect statistically significant relationship in the development of hydrocephalus. There was no statistical difference with regards to the location of aneurysm. In case with IVH, there was a statistically significant correlationship in the development of hydrocephalus (p=0.049). The ICH was found to show no statistical significance in the development of hydrocephalus. GCS score at admission of 13-15 was classified as group 1, GCS score of 9-12 was classified as group 2, and GCS score of 3-8 was classified as group 3. However, GCS group did not have

Table 1. Comparison of characteristics by the existence of hydrocephalus development

	Hydrocephalus		Total	χ^2	p-value
	Yes (%)	No (%)	(Person(%))		
Age					
20-40'	3 (4.6)	28 (42.4)	31 (46.9)	4.654	0.031
50-70'	11 (16.7)	24 (36.4)	35 (53.1)		
Sex					
Male	4 (6.1)	21 (31.8)	25 (37.9)	0.654	0.419
Female	10 (15.2)	31 (46.9)	41 (62.1)		
Hypertension					
Existence	8 (12.1)	25 (37.9)	33 (50.0)	0.363	0.547
Not existence	6 (9.1)	27 (40.9)	33 (50.0)		
Location of aneurysm					
Anterior communicating artery	6 (9.1)	24 (36.4)	30 (45.5)	1.880	0.598
Middle cerebral artery	2 (3.0)	13 (19.7)	15 (22.7)		
Posterior communicating artery	3 (4.6)	10 (15.2)	13 (19.7)		
The others	3 (4.6)	5 (7.6)	8 (12.1)		
Intraventricular hemorrhage					
Existence	8 (12.1)	15 (22.7)	23 (34.9)	3.890	0.049
Not existence	6 (9.1)	37 (56.1)	43 (65.1)		
Intracerebral hemorrhage					
Existence	2 (3.0)	9 (13.6)	11 (16.7)	0.073	0.788
Not existence	12 (18.2)	43 (65.2)	55 (83.3)		
Glasgow coma scale group					
group 1	4 (6.1)	12 (18.2)	16 (24.2)	4.105	0.128
group 2	4 (6.1)	5 (7.6)	9 (13.6)		
group 3	6 (9.1)	35 (53.1)	41 (62.1)		
Hunt-Hess SAH Classification group					
group 1	1 (1.5)	16 (24.2)	17 (25.8)	3.346	0.188
group 2	10 (15.2)	26 (39.4)	36 (54.6)		
group 3	3 (4.6)	10 (15.2)	13 (19.7)		
Fisher Grade group					
group 1	7 (10.6)	31 (46.9)	38 (57.6)	0.418	0.518
group 2	7 (10.6)	21 (31.8)	28 (42.4)		
Frontal horn/internal diameter of inner table ratio					
<40%	5 (7.6)	42 (63.6)	47 (71.2)	10.922	0.002
>40%	9 (13.6)	10 (15.2)	19 (28.8)		

Table 2. The relative risk of causal relationship of hydrocephalus

	Hydrocephalus Yes (%)	Hydrocephalus No (%)	Total (%)	Odd ratio	95% Confident interval	*p*-value
Age						
20-40'	3 (4.6)	28 (42.4)	31 (46.9)	4.278	1.067-17.143	0.031
50-70'	11 (16.7)	24 (36.4)	35 (53.1)			
IVH						
Existence	8 (12.1)	15 (22.7)	23 (34.9)	3.289	0.974-11.103	0.049
Not existence	6 (9.1)	37 (56.1)	43 (65.2)			
FH/ID ratio						
<40%	5 (7.6)	42 (63.6)	47 (71.2)	7.560	2.076-27.534	0.002
>40%	9 (13.6)	10 (15.2)	19 (28.8)			

IVH, Intraventricular hemorrhage; FH/ID, Frontal horn/internal diameter of inner table

Table 3. Logistic regression analysis of risk factor of hydrocephalus

	B	Standard deviation	*p*-value	Exp(B)	95.0% Confident interval Lower limit	Upper limit
Age (Reference:20-40')	1.371	0.932	0.141	3.938	0.634	24.465
Sex (Reference:Male)	-0.961	1.084	0.375	0.382	0.046	3.200
Hypertension (Reference : Not existence)	0.559	0.905	0.537	1.748	0.297	10.302
Location of aneurysm (Reference : A-com)			0.738			
MCA	0.827	1.155	0.474	2.288	0.238	22.003
P-com	1.531	1.428	0.284	4.621	0.281	75.909
The others	0.232	1.371	0.865	1.262	0.086	18.530
IVH (Reference : Not existence)	1.175	1.242	0.344	3.238	0.284	36.908
ICH (Reference : Not existence)	0.326	1.506	0.828	1.386	0.072	26.506
GCS group (Reference : group 3)			0.190			
group 2	0.018	2.056	0.993	1.018	0.018	57.223
group 1	2.223	2.042	0.276	9.237	0.169	505.068
Hunt-Hess SAH Classification group (Reference : group 1)						
group 2	-0.117	2.057	0.955	0.890	0.016	50.193
group 3	-2.617	1.931	0.175	0.073	0.002	3.213
Fisher Grade group (Reference : group 1)	-1.137	1.240	0.359	0.321	0.028	3.648
Frontal horn/internal diameter of inner table ratio (Reference : <40%)	1.559	0.912	0.087	4.754	0.796	28.383

A-com : anterior communicating artery, MCA : middle cerebral artery, P-com : posterior communicating artery, IVH : intraventricular hemorrhage, ICH : intracerebral hemorrhage, GCS : Glasgow coma scale

Table 4. Logistic regression analysis using by forward technique

	Standard deviation	*p* value	Exp(B)	95.0% Confident interval Lower limit	Upper limit
FH/ID ratio (Reference : <40)	0.659	0.002	7.560	2.076	27.534

FH/ID, Frontal horn/internal diameter of inner table ratio

any statistical significance in the development of hydrocephalus. The grouping of Hunt-Hess SAH Classification was made by assigning Hunt-Hess grade 1-2 as group 1, Hunt-Hess grade 3 as group 2, and assigning Hunt-Hess grade 4-5 as group 3. The grouping of Fisher grade was made by assigning Fisher grade 2-3 as group 1 and assigning Fisher grade 4 as group 2. The Hunt-Hess group and Fisher grade group did not have statistical significance in the development of hydrocephalus. In FH/ID ratio, the group having the ratio value higher than 40% revealed to show higher incidence of developing hydrocephalus compared to the group having the ratio value less than 40% (p=0.002)(Table 1).

Development risk of hydrocephalus for causes related to hydrocephalus

Factors such as age more than 50, existence of IVH and when FH/ID ratio is more than 40% were found to have development risk of hydrocephalus. The development risk of hydrocephalus among patients, the ratio of FH/ID resulted to have 7.560 of risk, and age resulted to have 4.278 of risk. However, the IVH recorded 3.289 of risk, but failed to show statistical significance due to inclusion of 1 in the 95% confident interval. Conclusively, The highest development risk of hydrocephalus was found from the case FH/ID ratio followed by age (Table 2).

Logistic regression analysis on the existence of hydrocephalus development

The logistic regression analysis was conducted to analyze the effect onto the development of hydrocephalus by treating multiple development causes as an independent variable. In the single development cause analysis, the statistical significant result was found from the following variables; age, IVH and FH/ID ratio.

From the logistic regression analysis, no statistically significant risk factors of hydrocephalus were not found, and the p value of FH/ID ratio recorded 0.087 that was the closest to 0.05, and it was followed by age and GCS group with the p values of 0.141 and 0.190, respectively (Table 3). The logistic regression analysis by using the forward technique has shown the statistical significance in FH/ID ratio with p value of 0.002 (Table 4).

DISCUSSION

Bagley[1] made a report on hydrocephalus after SAH first in 1928. And, in 1930, Winkelman and Fay[31] reported that communicating hydrocephalus resulted from closing of arachnoid villi after SAH. In 1948, the clinical aspects of hydrocephalus after SAH were clarified by Krayenbuhl and Luthy[13]. After these, Foltz and Ward[6] reported that operational treatments for hydrocephalus after SAH, would provide good results. Wenig et al.[30] reported that the frequency that the cerebral ventricle to be expanded after SAH on CT was 85.18%, but the incidence of hydrocephalus was 62.9%. Galera and Greitz[7] reported that 34% of the patients showed expansion of the ventricle after SAH, but 19% showed the symptoms of hydrocephalus. Based on the International Cooperative Study on the Timing of Aneurysm Surgery, hydrocephalus was developed in 22.3%[12]. The current study showed the 21.2% incidence of hydrocephalus.

Hydrocephalus after SAH is known to result from closing of the fourth ventricle or the basal cistern caused by blood clot, and absorption disorder and circulation disorder of cerebrospinal fluid caused by fibrosis of meninx[2,16,28]. In case of chronic hydrocephalus, closing and fibrosis of arachnoid granule cause absorption disorder of cerebrospinal fluid, which is associated with development of chronic hydrocephalus[5].

The possible causes of hydrocephalus after SAH are IVH, aging, the quantity of SAH on CT, vasospasm, the location of aneurysm, the level of consciousness at the time of visiting a hospital, hypertension, hyponatremia, and use or non-use of a hemostatic agent[18]. Silver et al.[21] reported IVH as the major cause for hydrocephalus. In the case of SAH, IVH is reported to be 13-37%, but the autopsy of the patients with SAH shows 48-57%[28]. Incidence of IVH in this study was 34.9% which was similar to the result of Silver et al.[21] Hassan and Tanghe[11] reported that over 53% of the patients with acute hydrocephalus had no IVH and they had only the basal cistern hemorrhage. The bloods of the basal cistern increase the resistance of the cerebrospinal fluid, which then increases the pressure of ventricle, but lowers the pressure around the arachnoid villi, so hydrocephalus outbreaks.

The development of hydrocephalus depending on the location of the cerebral aneurysm increases in the posterior circulation system, especially the basal aneurysm and the anterior communicating artery aneurysm. Neill et al.[18] reported that 29.7% of hydrocephalus was associated with the posterior circulation aneurysm and 15.9% with the anterior communicating artery aneurysm on CT. The hydrocephalus that was diagnosed in relation to clinical symptoms was 24.1% in the posterior circulation aneurysm and 14.6% in the anterior communicating artery aneurysm. But, current study did not show statistically significance depend on the aneurysmal location.

Until so far, literature reviews revealed that there was no difference of developing hydrocephalus by sex in SAH patients. However, it was reported to have higher pathogenic chances of developing hydrocephalus due to the reduction of cerebrospinal fluid absorption capacity caused by fibrosis of leptomeninges and by the diffused distribution of SAH in aged patients due to wide subarachnoid space[24,29]. Graft-Radford et al.[9] previously reported old age, consciousness state at the time of admission and the amount of hematoma found from brain CT, history of hypertension, IVH, and the location of aneurysm as the development causes of hydrocephalus after the SAH. And they pointed out the easier development of hematoma in diffused pattern due to the subarachnoid space in old aged patients, which promotes the subarachnoid fibrosis to cause more frequent development of cerebrospinal fluid absorption disorder.

The current study included FH/ID ratio at the time of admission as the development cause of hydrocephalus for the analysis, Penn et al.[19] stated the inappropriateness of using such ratio to reexistence the degree of ventricular enlargement. Although Van Gijn et al.[26] reported that the patient's debilitated consciousness state at the time of admission is accompanied with ventricular enlargement, which may lead to poor prognosis, other studies did not find any significant relationship between ventricular enlargement and the debilitation of consciousness, suggesting other factors could be associated to the debilitation of consciousness[17]. This is probably explained by the fact that the ventricular enlargement is highly correlated to the increase of age and to IVH. The IVH is most frequently occurred from anterior communicating artery aneurysm patients and 49.2% of these patients developed IVH. Yamamoto et al.[32] reported 48% of IVH occurrence rate among anterior communicating artery aneurysm patients. This could be explained by the fact that the anatomical position of aneurysm is closely located to the position of ventricle. The current study revealed 45.5% of IVH occurrence rate among anterior communicanting artery aneurysm patients.

Vale et al.[25] reported that the development of hydrocephalus caused by SAH after the rupture of aneurysm was about

20%, and the group of Hunt-Hess grade III or higher showed 90% outbreak when the patients were hospitalized. And, in the Fisher scale 4, 46% outbreak was reported. Various scales of a patient's conditions are important to judge his/her prognosis, but Hunt-Hess system tends to have greater difference according to the measurers compared with GCS score[14]. In this study, the number of patients with Hunt-Hess classification group 1 and 3 were too small, therefore statistically significant difference was not measured. This is similar in GCS group 2.

There is no known particular time for development of hydrocephalus after aneurysmal rupture[9,24]. However, it is known that in about 10-14 days after bleeding caused by the aneurysmal rupture, fibrosis and hyperplasia develop in the pia mater and the arachnoid, and then, hydrocephalus starts to appear[8]. The activity of prolyl 4-hydroxylase, a key enzyme in collagen synthesis resulted in an accumulation of type I collagen in the meningeal tissue, suggesting that the meninges are potential sites for fibrosis[22]. In addition to this, vasospasm, hypertension, hyponatremia, rebleeding, focal infarction, and antifibrinolytics are considered as other possible causes, but they are somewhat more closely related with acute hydrocephalus[9,24]. Therefore, in order to find the patients with chronic hydrocephalus early that outbreaks after bleeding by the rupture of the cerebral aneurysm, close follow-up observation will be necessary from the second month after an operation in patients having high risk factors before the operation. However, for the patients who are not highly risky, follow-up observation will be necessary every 6 months after the operation[5]. Kassell et al.[12], in the prospective study of the time for a surgery of the cerebral aneurysm, reported that the outbreak of hydrocephalus was high where delayed surgery was performed. Ljunggren et al.[15] reported that to cleansing out blood clot, which is collected in the basal cistern after a surgery, can reduce fibrosis of subarachnoid space and improve the distribution of cerebrospinal fluid thus the development of hydrocephalus can be reduced.

In addition, 11 (33.3%) out of 27 patients who had rebleedings before operation developed hydrocephalus, which means that IVH, SAH, and rebleeding of aneurysm before a surgery are considered as risk factors for the development of hydrocephalus[4].

One of the limitations in this study was that we did not study the metabolites related hydrocephalus. For example, higher cerebrospinal fluid ferritin levels may not reflect the amount of blood in the subarachnoid space that was intracranially metabolized, but rather more intense subarachnoid inflammatory reactions which may cause chronic hydrocephalus after SAH[23]. Further study needs to verify this and find other possible factors in the future.

CONCLUSION

Among the various causes, the analysis on single development cause related to hydrocephalus by the current study was able to verify the development causes as age more than 50, existence of IVH, and FH/ID ratio more than 40%.

References

1. Bagley C Jr : Blood in the cerebrospinal fluid : Resultant functional and organic alterations in the central nervous system : A Experimental data. **Arch Surg 17** : 18-38, 1928
2. Black P McL : Hydrocephalus and Vasospasm after SAH from ruptured intracranial aneurysm. **Neurosurgery 18** : 12-16, 1986
3. Choi CY, Park SW, Kwon JT, Kim YB, Min BK, Hwang SN, et al : Prognostic Factors of the Shunt Operation on the Hyderocephalus Following SAH. **J Korean Neurosurg Soc 27** : 178-185, 1998
4. Choi JJ, Koh HS, Cho JH, Kim SH, Youm JY, Song SH, et al : Clinical Study on Risk Factors of Hydrocephalus after Aneurysmal Subarachnoid Hemorrhage. **J Korean Neurosurg Society 30** : 1375-1380, 2001
5. Fernando LV, Edwin LB, Winfield SF : The relationship of subarachnoid hemorrhage and the need for postoperative shunting. **J Neurosurg 86** : 462-466, 1997
6. Foltz EL, Ward AA : Communicating hydrocephalus from subarachnoid bleeding. **J Neurosurg 13** : 546-566, 1956
7. Galera R, Greitz T : Hydrocephalus in the adult secondary to the rupture of intracranial arterial aneurysm. **J Neurosurg 32** : 634-641, 1970
8. Gjerris F, Borgesen SE, Sorensen PS, Boesen F, Schmidt K, Harmsen A, et al : Resistance to Cerebrospinal fluid outflow and Intracranial Pressure in Patients with Hydrocephalus after SAH. **Acta Neurochir (Wein) 88** : 79-86, 1987
9. Graft-Radford NR, Torner J, Adams HP Jr, Kassel NF : Factors associated with hydrocephalus after subarachnoid hemorrhage : A report of the Cooperative Aneurysm Study. **Arch Neurol 46** : 744-752, 1989
10. Gruber A, Reinprecht A : Chronic shunt-dependent Hydrocephalus after Early Surgical and Early Endovascular Treatment of Ruptured intracranial aneurysm. **Neurosurgery 44** : 503-509, 1999
11. Hassan D, Tanghe H : Distribution of cisternal blood in patients with acute hydrocephalus after subarachnoid hemorrhage. **Annals Neurol 31** : 374-378, 1992
12. Kassell NF, Toner JC, Jane JA, Haley EC Jr, Adams HP : The International Cooperative Study on the Timing of Aneurysm Surgery : Part 1 Overall management results. **J Neurosurg 73** : 37-47, 1990
13. Krayenbuhl H, Luthy F : Hydrocephalus als spatfolge geplatzter basaler Hiraneurysmen. **Schweiz Arch Neurol Psychiat 61** : 7-21, 1948
14. Lindsay KW, Teasdale GM, Knill-Jones RP : Observer variability in assessing the clinical features of subarachnoid hemorrhage. **J Neurosurg 58** : 57-62, 1983
15. Ljunggren B, Brandt L, Kagstorm E : Result of early operation for ruptured aneurysm. **J Neurosurg 54** : 473-479, 1981
16. Mancuso PA, Weinstein PR : Principles of management of subarachnoid hemorrhage : Pathophysiology and management of hydrocephalus after subarachnoid hemorrhage, in Ratcheson RA, Wirth FP(eds) : Ruptured cerebral aneurysms : Perioperative management. Baltimore : Williams and Wilkins, 1994, Vol 6, pp124-132
17. Mayhall CG, Archer NH, Lamb VA, Spadora AC, Baqqett JW, Ward JD, et al : Ventriculostomy related infections. A prospective epidemiologic study. **N Engl J Med 310** : 553-559, 1984
18. Neill RG, James T, Harold PA, Neal FK : Factors associated with hydrocephalus after subarachnoid Hemorrhage. **Arch Neurol 46** : 744-752, 1989
19. Penn RD, Belanger MG, Yasnoff WA : Ventricular volume in man computed from CAT scan. **An Neuro 3** : 216-223, 1978
20. Pietila TA, Heimberger KC, Palleske H, Brock M : Influence of aneurysm location on the development of chronic hydrocephalus following SAH. **Acta Neurochi(Wien) 137** : 70-73, 1995
21. Silver AJ : CT of SAH due to ruptured aneurysm. **AJNR 2** : 13-22, 1981
22. Sajanti J, Bjorkstrand AS, Finnila S, Heikkinen E, Peltonen J, Majamaa K : Increase of collagen synthesis and deposition in the arachnoid and

the dura following subarachnoid hemorrhage in the rat. **Biochim Biophys Acta 30** : 209-216, 1999
23. Suzuki H, Muramatsu M, Tanaka K, Fujiwara H, Kojima T, Taki W : Cerebrospinal fluid ferritin in chronic hydrocephalus after aneurysmal subarachnoid hemorrhage. **J Neurol 253** : 1170-1176, 2006
24. Tapaninaho A, Hernesneiemi J, Vapalahti M, Niskanen M, Kari A, Luukkonen M, et al : Shunt dependent Hydrocephalus after SAH and Aneurysm Surgery : Timing for Surgery is not a risk factor. **Acta Neurochir(Wein) 123** : 118-123, 1993
25. Vale FL, Bradley EL, Fisher III WS : The relationship of subarachnoid hemorrhage and the need for postoperative shunting. **J Neurosurg 86** : 462-466, 1997
26. Van Gijn J, Hijdra A, Wijdicks EF, Vermeulen M, van Crevel H : Acute hydrocephalus after aneurysmal SAH. **J Neurosurg 63** : 355-362, 1985
27. Vassilouthis J : The syndrome of normal pressure hydrocephalus. **J Neurosurg 61** : 501-509, 1984
28. Vassilouthis J, Richardson AE : Ventricular dilatation and communicating hydrocephalus following spontaneous subarachnoid hemorrhage. **J Neurosurg 51** : 341-351, 1979
29. Von Holst H, Bergstrand G : The amount of blood in CSF and degree of hydrocephalus studies with CT as compared to clinical condition and concentrations of various substances in human CSF after SAH. **Acta Neurochir(Wien) 94** : 38-41, 1988
30. Wenig C, Huber G, Emde H : Hydrocephalus after subarachnoid bleeding : A correlation of clinical findings, the results of radioisotope cisternography and computer-assisted tomography. **Eur Neurol 18** : 1-7, 1979
31. Winkelman NW, Fay T : Pacchionian system, histologic and pathologic changes with particular reference to idiopathic and symptomatic convulsive states. **Arch Neurol Psychiatry 23** : 44-64, 1930
32. Yamamoto Y : Pathology of Intracerebral hematoma produced by ruptured aneurysms of anterior cerebral artery. **No To Shinkei 33** : 38-39, 1981

COMMENTARY

The authors' aim in this study was to investigate the factors causing hydrocephalus in 66 aneurysmal patients with subarachnoid hemorrhage. They concluded that age, IVH, FH/ID ratio were statistically significant factors causing hydrocephalus in this selected patients.

Even though there is some controversy that age, Fisher grade, IVH, acute hydrocephalus were possible factors causing hydrocephalus, but no doubt that large amount of SAH which remaining long in subarachnoid space must be most important factor causing hydrocephalus. We should differentiate between hydrocephalus and simple ventricular dilatation before shunt applied to the aneurysmal patients because about a half of ventricular dilatation patients (30%) progressed to hydrocephalus with symptoms (15%).

Despite this concern, I congratulate the authors for reporting important factors causing hydrocephalus in patients with aneurysmal SAH.

In-Suk Hamm, M.D., Ph.D.
Department of Neurosurgery
Kyungpook National University