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The risk factors of chronic ventricular dilatation after aneurysmal subarachnoid haemorrhage

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Abstract

Objectives: The purpose of this study was to explore the independent risk factors of chronic ventricular dilatation after aneurysmal subarachnoid haemorrhage. *Methods:* A retrospective study was carried out in patients with aneurysmal subarachnoid haemorrhage and admitted to the Second Affiliated Hospital of Chongqing Medical University from July 2017 to February 2021. The patients were grouped according to whether they had chronic ventricular dilatation. The patients' demographic, clinical, and imaging datas including gender, age, hypertension, Hunt and Hess grade, Fisher grade, intraventricular hemorrhage, acute ventricular dilatation, aneurysm location, cerebrospinal fluid drainage, surgical methods, and meningitis were recorded and analyzed. And binary multivariate logistic regression models were used to investigate the independent risks for the chronic ventricular dilatation. *Results:* A total of 70 patients were analyzed and 36 (51.4%) developed chronic ventricular dilatation. Univariate analysis showed that age, Hunt and Hess grade, Fisher grade, intraventricular hemorrhage, acute ventricular dilatation, subtentorial aneurysms, and cerebrospinal fluid drainage were significantly different between the two groups. And there was no significant difference between the two groups in gender, hypertension, surgical method, or meningitis. Multivariate logistic regression analysis showed that acute ventricular dilatation was the only independent risk factor for chronic ventricular dilatation after aneurysmal subarachnoid haemorrhage (OR 92.1, 95% CI: 11.7–999.9, $P < 0.001$).
Conclusions: Acute ventricular dilatation was an independent risk factor of chronic ventricular dilatation after aneurysmal subarachnoid haemorrhage. Future research is needed to assess whether early treatment of acute ventricular dilatation can reduce chronic ventricular dilatation.

Keywords: aneurysm; subarachnoid haemorrhage; ventricular dilatation; hydrocephalus; risk factor

INTRODUCTION

Hydrocephalus is a common complication of aneurysmal subarachnoid haemorrhage (SAH), and it is divided into three stages: acute (0-3 days after SAH), subacute (4-13 days after SAH), and chronic (≥ 14 days after SAH).[1] The incidence of chronic shunt-dependent hydrocephalus ranges from 8.9% to 48%.[2] Previous studies found that several factors, such as advanced age, acute hydrocephalus, posterior circulation aneurysm,intraventricular hemorrhage (IVH), poor clinical status at admission, and cerebrospinal fluid (CSF) drainage were related to the development of chronic hydrocephalus.[3-7] However, it is still very difficult to prevent chronic hydrocephalus in clinical practice, so it should be further studied.

As hydrocephalus is defined as symptomatic ventricular dilatation, its diagnosis needs both the imaging evidence of ventricular dilatation and corresponding clinical manifestations, such as cognitive decline, unsteady gait and incontinence.[8,9] So if there is chronic hydrocephalus, there must be chronic ventricular dilatation. If we can find the way to prevent the chronic ventricular dilatation after aneurysmal SAH, then the chronic hydrocephalus can be prevented. However, the risk factors of chronic hydrocephalus have been widely investigated, but there are few studies on chronic ventricular dilatation. These were the reasons that we chose ventricular dilatation as our outcome variable instead of hydrocephalus. Therefore, the purpose of this study was to explore the risk factors of

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chronic ventricular dilatation after aneurysmal SAH.

METHODS

This study was conducted with the approval of the Institutional Review Board at the Second Affiliated Hospital of Chongqing Medical University. The need to obtain informed consent was waived by the IRB as this was a recording based study with no patient contact and personal details.

Patient selection and data collection

We retrospectively analyzed the data of patients with aneurysmal SAH who were admitted to the Second Affiliated Hospital of Chongqing Medical University from July 2017 to February 2021.

The inclusion criteria for this study were: 1) Patients with SAH; 2) Patients with ruptured aneurysms. Exclusion criteria: 1) Patients without computed tomography (CT) scan within 3 days after onset; 2) Patients without CT scan more than 14 days after onset; 3) Patients without surgical treatment; 4) Patients with traumatic brain injury, intracranial vascular malformation, intracranial tumor; 5) Patients with previous ventricular dilatation; 6) Patients with rebleeding; 7) Patients with intracranial haematoma.

Demographic, clinical, and imaging data of patients were collected. The potential risk factors that we chose in this study were based on the previous studies[7,10,11], including gender, age, hypertension, Hunt and Hess grade, Fisher grade, IVH, acute ventricular dilatation, aneurysm location, CSF drainage, surgical methods, and meningitis. The CSF drainage included external ventricular drainage (EVD) and lumbar cistern drainage (LD), and had been removed when the outcome CT scan was done.

Definition and classification of ventricular dilatation

Ventricular dilatation was defined as Evan's ratio > 0.3 or the bicaudate index (BCI) ≥ 0.2 on CT scans. Evan's ratio was defined as the ratio of the maximum width of the bilateral frontal horns to the maximum biparietal diameter.[7] The BCI was defined as the ratio of the width of the frontal horns to the diameter of the brain at the level of the caudate nucleus.[12]

We classified ventricular dilatation into acute (0-3 days after SAH), subacute (4-13 days after SAH) and chronic (≥ 14 days after SAH) to correspond to the classification of hydrocephalus. As acute hydrocephalus was defined as the development of ventricular dilatation within 3 days of the aneurysmal rupture, so it was equal to acute ventricular dilatation.

Classification of the aneurysm location

We used two classification methods for the aneurysm location in this study. One was the traditional classification, which divided aneurysms into anterior circulation aneurysms and posterior circulation aneurysms.[13] We defined this classification as "aneurysm location 1".

Because posterior circulation aneurysms are located under the tentorium, another new classification, which divided aneurysms into supratentorial aneurysms, tentorium notch space aneurysms and subtentorial aneurysms, was presented by us according to their relationship with the tentorium in the vertical direction. We defined this classification as "aneurysm location 2" (Table 1).

Statistical analysis

The patients were divided into two groups

Table 1: Classification of the aneurysm location

	Aneurysm location 1	Aneurysm location 2
AcoA & ACA	anterior circulation	supratentorial
MCA	anterior circulation	supratentorial
ICA	anterior circulation	tentorial notch space
BA	posterior circulation	subtentorial
VA	posterior circulation	subtentorial
PICA & SCA & AICA	posterior circulation	subtentorial
PCA	posterior circulation	supratentorial

Abbreviations: AcoA: anterior communicating artery, ACA: anterior cerebral artery, MCA: middle cerebral artery, ICA: internal carotid artery, BA: basilar artery, VA: vertebral artery, PICA: posterior inferior cerebellar artery, AICA: anterior inferior cerebellar artery, SCA: superior cerebellar artery, PCA: posterior cerebral artery.

according to the presence or absence of chronic ventricular dilatation. Data were summarized as mean ± standard deviation (SD) for continuous variables and as number (%) for categorical variables. Age was expressed as categorical variable (taking the average age as the cut point). The Mann-Whitney U test was used for continuous variables and the chi-squared test or Fisher's exact test was used for categorical variable. Univariate analysis was performed to compare the difference of factors between two groups. Subgroup analysis could be performed if necessary. The variables with a P value of < 0.1 in univariate analysis were included in the multivariate logistic analysis, and the odds ratio (OR) and 95% confidence interval (CI) were calculated. Data analysis was performed with SAS software (version 9.4. SAS Institute, Inc.). The significance was set at $P < 0.05$.

RESULTS

Baseline data

Table 2 showed the baseline data. A total of 70 patients with aneurysmal SAH were included, and the incidence of chronic ventricular dilatation was 51.4%. Univariate analysis sowed that there were significant differences in age, Hunt and Hess grade, Fisher grade, IVH, acute ventricular dilatation, and CSF drainage between 2 groups ($P < 0.05$). There was no significant difference between the two groups in gender, hypertension, surgical method, and meningitis.

Subgroup analysis of aneurysm location 2

Because the *P* value of "aneurysm location 2" was 0.055, which was close to the significance level,

Table 2: Baseline data

Variables	Total (n=70)	Chronic ventricular dilatation		*P* value
		Yes (n=36)	No (n=34)	
Gender (female), n (%)	46 (65.7)	25 (69.4)	21 (61.7)	0.499
Age (≥55 years), n (%)	34 (48.5)	24 (66.6)	10 (29.4)	0.002
Hypertension, n (%)	39 (55.7)	20 (55.5)	19 (55.8)	0.978
Hunt and Hess grade, n (%)				< 0.001
I	4 (5.7)	2 (5.6)	2 (5.9)	
II	39 (55.7)	12 (33.3)	27 (79.4)	
III	24 (34.3)	19 (52.8)	5 (14.7)	
IV	3 (4.3)	3 (8.3)	0	
V	0	0	0	
Fisher grade, n (%)				< 0.001
I	0	0	0	
II	5 (7.1)	0	5 (14.7)	
III	9 (12.9)	0	9 (26.5)	
IV	56 (80.0)	36 (100.0)	20 (58.8)	
Intraventricular haemorrhage, n (%)	56 (80.0)	36 (100.0)	20 (58.8)	< 0.001
Acute ventricular dilatation, n (%)	39 (55.7)	36 (100.0)	3 (8.8)	< 0.001
Aneurysm location 1, n(%)				0.064
Anterior circulation	57 (81.4)	26 (72.2)	31 (91.2)	
Posterior circulation	13 (18.6)	10 (27.8)	3 (8.8)	
Aneurysm location 2, n (%)				0.055
Supratentorial	24 (34.3)	9 (25.0)	15 (44.1)	
Tentorial notch space	35 (50.0)	18 (50.0)	17 (50.0)	
Subtentorial	11 (15.7)	9 (25.0)	2 (5.9)	
Cerebrospinal fluid drainage, n (%)	37 (52.8)	26 (72.2)	11 (32.3)	0.001
Surgical method, n (%)				0.917
Coiling	49 (70.0)	25 (69.4)	24 (70.6)	
Clipping	21 (30.0)	11 (30.6)	10 (29.4)	
Meningitis, n (%)	7 (10.0)	5 (13.8)	2 (5.8)	0.430

Table 3: Subgroup analysis of aneurysm location 2

Variables	Total	Chronic ventricular dilatation		P value
		Yes	No	
Aneurysm location 2				0.027
Supratentorial, n (%)	24 (68.6)	9 (50.0)	15 (88.2)	
Subtentorial, n (%)	11 (31.4)	9 (50.0)	2 (11.8)	
Aneurysm location 2				0.425
Supratentorial, n (%)	24 (40.7)	9 (33.3)	15 (46.9)	
Tentorial notch space, n (%)	35 (59.3)	18 (66.7)	17 (53.1)	
Aneurysm location 2				0.092
Tentorial notch space, n (%)	35 (76.1)	18 (66.7)	17 (89.5)	
Subtentorial, n (%)	11 (23.9)	9 (33.3)	2 (10.5)	

we further performed a subgroup analysis. And the results showed that subtentorial aneurysm was significantly associated with chronic ventricular dilatation (Table 3).

Exploration of independent factors of chronic ventricular dilatation in patients with aneurysmal SAH

Multivariate binary logistic regression analysis showed that acute ventricular dilatation ($p < 0.001$) was the only independent risk factor of chronic ventricular dilatation (Table 4).

DISCUSSION

In this study, we explored the risk factors of chronic ventricular dilatation after aneurysmal SAH. And our results showed that there were several factors were associated with chronic ventricular dilatation after aneurysmal SAH, and acute ventricular dilatation was the only independent risk factor. The effect of acute ventricular dilatation on chronic ventricular dilatation was independent of age, Hunt and Hess grade, Fisher grade, IVH, aneurismal location, and CSF drainage. So we discussed each risk factor around the acute ventricular dilatation, and then proposed an alternative explanation for mechanism of chronic hydrocephalus.

Some previous studies showed that acute hydrocephalus is a risk factor for chronic hydrocephalus after aneurysmal SAH.[14,15] Because we defined acute ventricular dilatation as ventricular dilatation within 3 days after onset, which was the same definition for acute hydrocephalus[1], it showed that our results was consistent with previous studies.

In clinical practice, acute ventricular dilatation was often accompanied by IVH, which was a well-known risk factor of chronic hydrocephalus.[14,16] And in our univariate analysis, it was significantly related to chronic ventricular dilatation, too. However, it was not significant in multivariate analysis. Our results showed that all the 36 patients with acute ventricular dilatation had IVH, so IVH and acute ventricular dilatation were correlated with each other closely. Therefore, we thought that IVH could established relationship with

Table 4: Multivariate binary logistic regression

Variables	Odds Ratio	95% confidence interval	P value
Age	1.016	0.900-1.147	0.734
Hunt and Hess	1.683	0.006-397.751	0.794
Fisher grade	1.008	0.004-317.472	0.997
Intraventricular haemorrhage*	Not applicable	Not applicable	Not applicable
Acute ventricular dilatation	92.135	11.710-999.999	< 0.001
Aneurysm location 2	0.959	0.094-8.331	0.967
Cerebrospinal fluid drainage	1.636	0.171-18.349	0.618

*IVH had no value, since it was a linear combination of Fisher grade IV.

the chronic ventricular dilatation through acute ventricular dilatation.

Higher Fisher grade was related to the development of chronic hydrocephalus in previous studies[4,17], which was consistent with our univariate analysis results. However, there was also no statistical significance in multivariate analysis for Fisher grade. In this study, Patients with intracranial haematoma were excluded for avoiding affecting the measurement of ventricles in this study, so Fisher grade IV was equal to IVH. It meant that higher Fisher grade could establish relationship with the chronic ventricular dilatation through IVH and further acute ventricular dilatation.

Previous studies revealed that the incidence of chronic hydrocephalus was higher in posterior circulation aneurysms.[14,16] However, there was no significant relationship between posterior circulation aneurysms and chronic ventricular dilatation in our study. This might be due to the small sample size. Because posterior circulation aneurysms were located under the tentorium, we introduced a new classification defined as the "aneurysm location 2". We divided aneurysms into supratentorial aneurysms, tentorium notch space aneurysms and subtentorial aneurysms according to their relationship with the tentorium in the vertical direction. The results of the subgroup analysis showed that subtentorial aneurysms, compared to supratentorial aneurysms, were significantly associated with chronic ventricular dilatation. We explained it as subtentorial aneurysms could cause higher pressure of posterior fossa than supratentorial aneurysms, which could make it easier for blood to flow back into the ventricle, resulting in IVH. Therefore, subtentorial aneurysms could also be linked to the chronic ventricular dilatation through IVH and acute ventricular dilatation.

As for Hunt and Hess grade, previous studies showed higher Hunt and Hess grades were a risk factor for chronic hydrocephalus.[5,14] Our study also showed that higher Hunt and Hess grade was associated with chronic ventricular dilatation in the univariate analysis. Hunt and Hess grade reflects the severity of clinical symptoms.[5] What was particular in this study was that the difference between two groups was mainly presented at grade II and III, and the key change from grade II to III was consciousness changing from wakefulness to lethargy. For aneurysmal SAH patients, altered consciousness was mainly caused by greater intracranial hypertension, especially extreme high pressure of posterior fossa, which could influence brainstem ascending reticular activating system (ARAS). It is obvious that subtentorial aneurysms are more likely to cause higher pressure of posterior fossa. Therefore, higher Hunt and Hess grade could be linked to chronic ventricular dilatation through subtentorial aneurysms, IVH and acute ventricular dilatation.

CSF drainage, including EVD and LD, was associated with chronic ventricular dilatation in univariate analysis. As a treatment for acute hydrocephalus, EVD had a close correlation with acute ventricular dilatation. And LD is often used to clear bloody CSF in patients with high Hunt-Hess grade or high Fisher grade, and also to drain bloody CSF to facilitate intracranial aneurysm clipping by providing adequatespace for surgery. Therefore, the patient's condition of chronic ventricular dilatation group was more serious at admission. Although the percentage of CSF drainage in patients with chronic ventricular dilatation was higher than those without (72.2% vs 32.3%), we did not think CSF drainage was a risk factor of chronic ventricular dilatation. Further cohort study is needed to evaluate the role of CSF drainage.

In univariate analysis, age ≥ 55 years was significantly associated with chronic ventricular dilatation, which was comparable to previous reports.[14] Although some studies explained it as ageing might accelerate an increase in the CSF compartment due to brain atrophy and that a reduction in CSF turnover contributed to the accumulation of brain metabolites[18,19], it might just because ventricles will gradually become larger with increasing age.

According to the discussion above, the factors which were significant in univariate analysis, were related to the acute ventricular dilatation directly or indirectly. So it was reasonable that acute ventricular dilatation was the only independent risk factor in multivariate analysis. But what does acute ventricular dilatation mean exactly? We thought that acute ventricular dilatation was the direct indicator reflecting the high intraventricular pressure.

Nowadays, most researchers agree that blood clotting and subarachnoid fibrosis influence the circulation and absorption of CSF[6,20,21], but this cannot explain why there is ventricular dilatation instead of subarachnoid space widening and why endoscopic third ventriculostomy is usually ineffective. Therefore, we had a hypothesis as follows: 1) after intracranial aneurysms rupturing, the pressure of the subarachnoid space rises, and bloody CSF flows back into the ventricle through

the median and lateral foramen of fourth ventricle, which causes IVH and intraventricular high pressure, resulting in acute ventricular dilatation; 2) If the duration of high pressure last long enough, the ventricular wall might be damaged, and eventually, enlarged ventricles may become irreversible.

Our findings may have potential clinical management implications. As previous research had shown that early EVD could reduce the incidence of chronic hydrocephalus[22], perhaps solving acute hydrocephalus early could decrease the incidence of chronic hydrocephalus after aneurysmal SAH.

This study has some limitations. First, this was a retrospective analysis and therefore could be subject to bias from factors that was not measured. Second, this was a single-centre study, and the sample size was small.

In summary, we found that acute ventricular dilatation was an independent risk factor for chronic ventricular dilatation after aneurysmal SAH. Future research is needed to assess whether treatment of early acute ventricular dilatation can reduce chronic ventricular dilatation.

DISCLOSURE

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Conflict of interest: None

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