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Clinical features and prognostic factors in patients with intraventricular hemorrhage caused by ruptured arteriovenous malformations

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Abstract

Intraventricular hemorrhage (IVH) was associated with poor outcomes in patients with intracerebral hemorrhage. IVH had a high incidence in patients with ruptured arteriovenous malformations (AVMs). In this study, we aimed to discuss the clinical features and prognostic factors of outcomes in the patients with AVM-related IVH.

From January 2010 to January 2016, we collected the data of the patients with AVM-related IVH retrospectively. The data, including clinical and radiological parameters, were collected to evaluate the clinical features. Univariate and multivariate logistic regression analyses were used to identify the prognostic factors for clinical outcomes (hydrocephalus, 6-month outcomes measured by the modified Rankin scale) in our cohort.

A total of 67 eligible patients were included and 19 patients (28%) only presented with IVH. Thirty-three patients (49%) presented hydrocephalus, and 12 patients (18%) presented brain ischemia. Nineteen patients (28%) had a poor outcome after 6 months. In multivariate logistic regression, subarachnoid hemorrhage (SAH) ($P = .028$) was associated with hydrocephalus and higher Graeb score ($P = .080$) tended to increase the risk of hydrocephalus. The high Glasgow coma scale ($P = .010$), large hematoma volume of parenchyma ($P = .006$), and high supplemented Spetzler–Martin (sup-SM) score ($P = .041$) were independent factors of the poor outcome.

IVH was common in ruptured AVMs and increased the poor outcomes in patients with the ruptured AVMs. The AVM-related IVH patients had a high incidence of hydrocephalus, which was associated with brain ischemia and SAH. Patients with lower Glasgow coma scale, lower sup-SM score, and smaller parenchymal hematoma had better long-term outcomes.

Abbreviations: AVMs = arteriovenous malformations, CSF = cerebrospinal fluid, CT = computed tomography, CTA = computed tomography angiography, DSA = digital subtraction angiography, EVD = external ventricular drains, GCS = Glascow coma scale, ICP = intracranial pressure, IVH = intraventricular hemorrhage, MAP = mean arterial pressure, mRS = modified Rankin scale, NCCT = noncontrast CT, PIVH = primary intraventricular hemorrhage, SAH = subarachnoid hemorrhage, sICH = spontaneous intracerebral hemorrhage, sup-SM = supplemented Spetzler–Martin.

Keywords: brain ischemia, cerebral hemorrhage, hydrocephalus, intracranial arteriovenous malformations

1. Introduction

Intraventricular hemorrhage (IVH) is defined as extension of hemorrhage into the ventricular systems.[1] IVH could lead to high mortality of 30% to 80% in spontaneous intracerebral hemorrhage (sICH),[2,3] and many previous studies[3–8] suggested that IVH was related to the poor outcomes, including impaired consciousness, mortality, and functional outcomes. With the extension of hemorrhage into ventricles, the balance of ventricle systems will be broken, which contributes to pathophysiologic mechanisms of ICH.[3,6] Compared to the sICH patients, ICH patients combined with IVH showed significantly increased of mortality rates and higher risk of developing complications.[3] The most common complications of IVH include brain ischemia and hydrocephalus, which are associated with poor outcomes.[9–11]

The possible etiological causes of IVH include sICH and secondary intracerebral hemorrhage. Some studies suggested that ruptured arteriovenous malformations (AVMs) were the major cause of primary intraventricular hemorrhage (PIVH)[9,12,13] and approximately 17% to 47% of ruptured AVMs have been combined with IVH regardless of parenchymal hemorrhage.[14,15] Previous studies[9,13] have well discussed the IVH caused by sICH; however, the available studies[8,14,15] which

focused on the IVH caused by secondary ICH are rare, especially on ruptured AVMs. In addition, the clinical features and prognostic factors of IVH caused by ruptured AVMs remain unclear. Therefore, we conducted a retrospective study and aimed to evaluate the clinical features and prognostic factors for outcomes, including complications and functional outcome in the patients with AVM-related IVH.

2. Materials and methods

2.1. Study population

A retrospective search was conducted in patients with ruptured AVMs, in the Department of Neurosurgery at West China Hospital, Sichuan University from January 2010 to January 2016. The protocol was approved by the Institutional Review Board at the West China Hospital of Sichuan University. All patients signed the consent form after being fully informed.

We included the patients following the inclusion criteria: diagnosed with AVMs by computed tomography angiography (CTA), magnetic resonance angiography, or digital subtraction angiography (DSA); initial computed tomography (CT) upon admission showed the extension of hemorrhage into ventricles; and with or without parenchymal hemorrhage. The patients with IVH caused by other factors, such as tumors, trauma, and moyamoya disease, were excluded.

2.2. Data collection

Demographics of eligible patients were collected, including age, gender, first symptoms, medical history, Glasgow coma scale (GCS), subarachnoid hemorrhage (SAH), Graeb score, supplement Spetzler–Martin score (sup-SM), with or without aneurysm, hematoma volume of parenchyma, location of hematoma, treatments, mean arterial pressure (MAP), presence of herniation, and complications (hydrocephalus or brain ischemia). Another researcher who was blinded to the research assessed the end points, including the incidence of complications and modified Rankin scale (mRS) at discharge and 6 months after onset. The 6-month poor outcome was defined as mRS $\geq$ 3 and good outcome was defined as mRS ranging from 0 to 2.

2.3. Imaging procedures

Initial noncontrast CT (NCCT) was conducted using a 64-slice CT scanner (SOMATOM Definition Flash; Siemens Healthcare Sector, Forchheim, Germany). Brain imaging was done by NCCT with 2 or 5 mm slice thickness. CTA was conducted to diagnose the AVMs when the DSA could not be provided. The intraventricular extension was classified according to the score introduced by Graeb.[16] Hydrocephalus was defined as the bicaudate index (the width of the frontal horns at the level of the caudate nuclei/the width of the brain at same level) above 95th percentile of age.[17]

2.4. Statistical analysis

The Student t test and Pearson chi-squared test were conducted to compare continuous variables and categorical variables, respectively. With univariate analysis, the variables were evaluated for the complication and poor outcomes at 6th month (mRS $\geq$ 3). The variables with $P < .10$ from the univariate analysis were subsequently evaluated for the poor outcomes in multivariable

logistic regression model. Statistical significance was defined as $P < .05$. Statistical analyses were performed and conducted by SPSS Statistical Software (version 17.0, SPSS Inc., Chicago, IL).

3. Results

From January 2010 to January 2016, a total of 231 patients were admitted to our hospital due to ruptured AVMs, and 89 patients (38.5%, 89/231) combined with IVH with or without parenchymal hemorrhage were brought into the study. Consequently, only 67 patients were eligible for the study, and 22 patients were excluded, of which 7 patients have no initial NCCT and 15 patients were lost to follow.

3.1. Clinical characteristics of patients and AVMs

Overall, 46 men (69%) and 21 women (31%) were included with a mean age of 26.5 ± 13.3 years. The clinical and radiological data of all included patients were collected. The most common symptoms were headache (76%) and disturbance of consciousness (50%). The mean Graeb score was 6.6 ± 3.1 and mean hematoma volume of parenchyma was 14.2 ± 16.0 mL. Forty-eight IVH patients (72%) were coexisted with the parenchymal hemorrhage and 19 patients (28%) only presented with IVH. In our series, initial treatments for IVH included resection of nidus and hematoma evacuation in 24 patients (24/67, 36%), ventricular puncture and drainage in 11 patients (11/67, 16%), emergency hematoma evacuation in 6 patients (6/67, 9%) and other treatments (26/67, 39%) including embolization (n=12) and gamma knife (n=14). However, most of them underwent more than 1 kind of treatment during hospitalization period. After the treatments, 9 patients were discharged automatically, 37 patients were transferred to rehabilitation hospitals and 21 were discharged normally (Table 1).

3.1.1. Complications after AVM-related IVH.
The most common complication was the hydrocephalus, which was shown in 33 patients (49%), of which 15 patients presented with hydrocephalus within 48 h, and 18 patients after 48 h. The communicating hydrocephalus was found in 14 patients, and 19 patients presented with obstructive hydrocephalus. Four patients presented with recurrent hemorrhage after the emergency hematoma evacuation without resection of nidus. Brain ischemia was identified in 12 patients (18%), epileptic seizure in 5 patients (8%), pneumonia in 12 patients (18%), intracranial infections in 7 patients (10%), renal insufficiency in 1 patient (1%), and gastrointestinal bleeding in 1 patient (1%), respectively. Herniation was identified in 13 patients (19%) during hospitalization. Six patients presented with herniation upon admission with pupil change and unstable vital signs, of which 2 patients underwent resection of nidus and hematoma evacuation and 4 patients underwent emergency hematoma evacuations. Seven patients presented with herniation after 48-h from the onset with aggravating disturbances of consciousness gradually, of which 5 patients underwent ventricular puncture and drainage, 2 patients received emergency hematoma evacuation.

3.1.2. Six-month outcomes after AVM-related IVH.
Twenty-two patients (33%) had poor outcomes at discharging and 19 patients (28%) had poor outcomes after 6 months, including score of 3 in 9 patients, score of 4 in 3 patients, score of 5 in 3 patients, and 6 (death) in 4 patients.

Table 1

Comparison of IVH with or without parenchymal hemorrhage.

Variables	Total	No parenchymal hemorrhage	Parenchymal hemorrhage	P
Total, n (%)	67	19 (28)	48 (72)	
Age, y (mean±SD)	26.5±13.3	30.4±13.1	25.0±13.3	.140
Gender, n (%)				.542
Males	46 (69)	12 (63)	34 (71)	
Females	21 (31)	7 (37)	14 (29)	
Medical history, n (%)				
Hypertension	3 (5)	1 (5)	2 (4)	.845
Stroke	8 (12)	4 (21)	4 (8)	.148
GCS, n (%)				.463
9–15	50 (75)	13 (68)	37 (77)	
3–8	17 (25)	6 (32)	11 (23)	
SAH, n (%)	30 (45)	10 (53)	20 (42)	.416
Graeb score				**.003**
1–8	43 (64)	7 (37)	36 (75)	
9–12	24 (36)	12 (63)	12 (25)	
Combined with aneurysm, n (%)	11 (16)	3 (16)	8 (17)	.930
Location of AVMs, n (%)				**.002**
Brain lobe	35 (52)	6 (33)	29 (59)	
Basal ganglia and thalami	23 (34)	12 (67)	11 (22)	
Infratentorial	9 (13)	0	9 (13)	
Sup-SM score, mean±SD	5.68±1.21	6.44±0.92	5.41±1.19	**<.001**
Treatments				—
Surgery	35	6	29	
Gamma knife	19	4	15	
Embolization	9	7	2	
EVD	9	5	4	
MAP, >100 mm Hg	10 (15)	2 (11)	8 (17)	.525
Hydrocephalus, n (%)	33 (49)	8 (42)	25 (52)	.462
Brain ischemia, n (%)	12 (18)	6 (32)	6 (13)	.066
Occurrence of herniation, n (%)	13 (19)	4 (21)	9 (18)	.830
Hospital stay, d (mean±SD)	15.6±13.3	14.61±5.84	15.96±15.20	.716
ICU stay, d (mean±SD)	4.8±11.6	4.44±6.69	4.90±13.09	.889
Poor outcome, n (%)				
At discharge	22 (33)	4 (21)	18 (38)	.196
After 6 months	19 (28)	2 (11)	17 (35)	**.042**

AVMs = arteriovenous malformations, EVD = external ventricular drains, GCS = Glasgow coma scale, ICU = intensive care unit, IVH = intraventricular hemorrhage, MAP = mean arterial pressure, SAH = subarachnoid hemorrhage, SD = standard deviation, sup-SM score = supplement Spetzler–Martin score.
Bold values signifies $P < .05$.

3.2. Comparison of IVH with or without parenchymal hemorrhage

The AVM of IVH patients without parenchymal hemorrhage often located at basal ganglia and thalami, and they more likely presented with a higher sup-SM score ($P < .001$) and a higher Graeb score ($P = .003$) than parenchymal hemorrhage group (Table 1). In our cohort, AVM-related IVH patients without parenchymal hemorrhage had better outcomes ($P = .042$).

3.3. Univariate and multivariate logistic regression

With univariate logistic regression, there were significant different incidence of hydrocephalus with several variables, such as gender, GCS, presence of SAH, Graeb score, hematoma volume, and MAP. After multivariate logistic regression, the results showed that SAH (OR: 4.0, 95% CI: 1.2–13.8, $P = .028$) was an independent predictor for the hydrocephalus and the higher Graeb score might increase the risk of hydrocephalus (Table 2). With univariate logistic regression, the variables, including gender, GCS, parenchymal hemorrhage, hematoma volume, sup-SM grade, and occurrence of hernia had the significant difference between good outcomes and poor out-

comes. After multivariate logistic regression, the GCS (OR: 1.6, 95% CI: 1.1–2.4, $P = .010$), hematoma volume of parenchyma (OR: 3.4, 95% CI: 1.4–8.1, $P = .006$), and sup-SM score (OR: 1.3, 95% CI: 1.0–1.7, $P = .041$) were independent prognostic factors for the functional outcome at 6 months (Table 3).

4. Discussion

IVH is a common complication of intracerebral hemorrhage and SAH, and it is associated with poor functional outcomes.[6–8,18] It is reported that ruptured AVMs is a major cause of PIVH[19,20] and 23% of survivors will become disabled.[19] Previous studies have well discussed the factors for the prognosis in IVH caused by sICH or ruptured aneurysm[21–24]; however, the understanding regarding to IVH caused by ruptured AVMs was limited. Therefore, a retrospective study was conducted to discuss the clinical features and prognostic factors in a relatively large sample. We found that there is a high incidence of IVH caused in ruptured AVMs and PIVH caused by AVMs had a better outcomes than other factors. The high Graeb score and SAH increased the risk of hydrocephalus; in addition, patients with lower GCS, lower sup-SM score, and smaller parenchymal hematoma had a better long-term outcomes.

Table 2

Univariate analysis and multivariate logistic regression of the presence of hydrocephalus.

Variables	Hydrocephalus	No hydrocephalus	Univariate analysis OR (95% CI)	P	Multivariate regression analysis OR (95% CI)	P
Total	33 (49)	34 (51)				
Age > 20 y, n (%)	21 (46)	25 (54)	1.9 (0.7–5.6)	.217		
Gender, n (%)						
Males	27 (58)	19 (41)	3.5 (1.2–10.8)	.022	2.7 (0.7–9.9)	.132
Females	6 (29)	15 (71)				
Medical history, n (%)						
Hypertension	2 (67)	1 (33)	2.1 (0.2–24.7)	.537		
Stroke	6 (75)	2 (25)	3.6 (0.7–19.1)	.121		
GCS, n (%)						
9–15	21 (42)	29 (58)	3.3 (1.0–10.8)	.042	1.6 (0.4–7.2)	.511
3–8	12 (71)	5 (29)				
SAH, n (%)	21 (70)	9 (30)	4.9 (1.7–13.8)	.002	4.0 (1.2–13.8)	**.028**
Graeb score, n (%)						
1–8	17 (39)	26 (61)	3.1 (1.1–8.7)	.033	3.5 (0.9–14.3)	.080
9–12	16 (67)	8 (33)				
Combined with aneurysm, n (%)	4 (36)	7 (64)	0.5 (0.1–2.0)	.350		
Hematoma volume of parenchyma > 20 mL, n (%)	5 (29)	12 (71)	0.3 (0.1–1.1)	.058	0.5 (0.1–1.9)	.297
Parenchymal hemorrhage, n (%)	23 (48)	25 (52)	1.5 (0.5–4.4)	.462		
Sup-SM score > 6, n (%)	9 (60)	6 (40)	1.8 (0.5–5.6)	.345		
MAP > 100 mm Hg, n (%)	8 (80)	2 (20)	5.1 (1.0–26.3)	.035	2.1 (0.3–14.1)	.458

CI = confidence interval, GCS = Glasgow coma scale, MAP = mean arterial pressure, OR = odds ratio, SAH = subarachnoid hemorrhage, sup-SM score = supplement Spetzler–Martin score.
Bold values signifies $P < .05$.

4.1. Occurrence of IVH in ruptured AVMs

Extension of hemorrhage into ventricles was common in the ruptured AVMs. In our study, we found 38.5% (98/231) of ruptured AVMs patients had IVH, which was similar to the rate of 17% to 47% reported in previous studies.[14,15] The rate of IVH without parenchymal hemorrhage in ruptured AVMs patients was 7.8% (18/231), which was lower than previously reported 17%.[14,15] The inconsistency might attribute to the different scanning thickness of NCCT. The NCCT with scanning slices of 5 mm in thickness were widely used, while 7 of 67 patients with the small hematoma in parenchyma could only be detected by NCCT of 2 mm thickness rather than 5 mm thickness. In addition, several patients without parenchymal hemorrhage might be excluded who did not provide initial NCCT or lost during following-up.

4.1.1. Clinical features and outcomes.
The patients of AVM-related ICH were younger and had less medical comorbidity than ICH due to other causes.[25,26] In addition, the state of consciousness in patients with AVM-related IVH was better.

Table 3

Univariate and multivariate regression analyses of functional outcome at 6 months after onset.

Variables	Good outcome	Poor outcome	Univariate analysis OR (95% CI)	P	Multivariate regression analysis OR (95% CI)	P
Total	48 (72)	19 (28)				
Age > 20 y, n (%)	31 (67)	15 (33)	2.1 (0.6–7.2)	.253		
Gender, n (%)			3.2 (0.8–12.5)	.084	1.0 (0.9–1.1)	.359
Males	30 (65)	16 (35)				
Females	18 (86)	3 (14)				
GCS, n (%)			4.5 (1.4–14.6)	.009	1.6 (1.1–2.4)	**.010**
9–15	40 (80)	10 (20)				
3–8	8 (47)	9 (53)				
SAH, n (%)	22 (73)	8 (27)	0.86 (0.3–2.5)	.782		
Graeb score			0.8 (0.2–2.4)	.649		
1–8	30 (69)	13 (31)				
9–12	18 (75)	6 (25)				
Combined with aneurysm, n (%)	9 (72)	2 (18)	0.5 (0.1–2.6)	.413		
Parenchymal hemorrhage, n (%)	31 (65)	17 (35)	4.7 (0.9–22.6)	.042	2.8 (0.7–11.1)	.142
Hematoma volume of parenchyma > 20 mL, n (%)	6 (35)	11 (65)	9.6 (2.8–33.6)	<.001	3.4 (1.4–8.1)	**.006**
Sup-SM score > 6, n (%)	8 (53)	7 (47)	2.9 (0.9–9.7)	.074	1.3 (1.0–1.7)	**.041**
MAP > 100 mm Hg, n (%)	6 (60)	4 (40)	1.9 (0.5–7.5)	.376		
Hydrocephalus	24 (73)	9 (27)	0.9 (0.3–2.6)	.846		
Brain ischemia	8 (67)	4 (33)	1.3 (0.4–5.1)	.673		
Occurrence of hernia, n (%)	6 (46)	7 (54)	4.1 (1.2–14.5)	.023	2.3 (0.4–13.6)	.959

CI = confidence interval, GCS = Glasgow coma scale, MAP = mean arterial pressure, OR = odds ratio, SAH = subarachnoid hemorrhage, sup-SM score = supplement Spetzler–Martin score.
Bold values signifies $P < .05$.
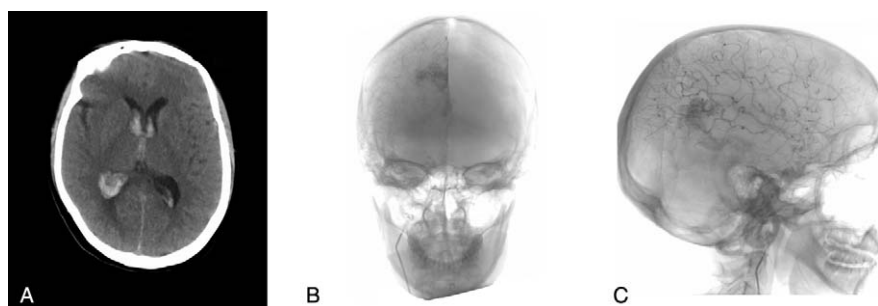
Figure 1. Representative case of primary intraventricular hemorrhage caused by ruptured arteriovenous malformations. A 27-year-old woman had a primary intraventricular hemorrhage diagnosed by computed tomography scan (A), with a Graeb score of 7 points and presence of subarachnoid hemorrhage. Digital subtraction angiography identified the arteriovenous malformation (B and C) located in peri-ventricle with supplemented Spetzler–Martin score of 7, supplying by bilateral posterior cerebral arteries.

There were 75% of our patients had an admission GCS >9, while the patients with hypertensive intracerebral hemorrhage (HICH)-related IVH had a lower mean GCS of 6.3.[27] The younger age and better consciousness could explain the lower rate of 6-month poor outcomes compared with patients with hypertension-related IVH.[3] The incidence of hydrocephalus was higher in AVM-related IVH (49%) than HICH-related IVH (28.6%),[28] and 1 study[29] also suggested the high rate of shun-dependent hydrocephalus (44%) in ruptured AVMs. Although the rate of hydrocephalus was relatively higher than the rate of brain ischemia and herniation, these complications attributed to the increased poor outcomes in AVM-related IVH compared with ruptured AVMs without IVH.[30–32]

4.1.2. PIVH caused by AVMs. IVH without parenchymal hemorrhage was regarded as PIVH (Fig. 1). Ruptured AVMs were the major cause of PIVH[33] and 27% of our series presented with PIVH, which was consistent with the previous studies.[14,15] In PIVH, with the less resistance from brain tissue, the volume of blood into ventricles and the Graeb score increased. Although PIVH patients tended to have a higher Graeb score, we found that long-term outcome of PIVH was better than IVH with parenchymal hemorrhage (Table 1). This could be explained by the fact that extension of all the hemorrhage into the ventricles would decrease the damage to the healthy brain parenchyma.[25,26,34] In addition, IVH combined with parenchymal hemorrhage was always caused by the AVMs which located in deeper location or closer to the wall of ventricle,[35] such as basal ganglia and thalami, which played an important role in motor and sense. With the injury to parenchyma brain in these areas, the patients would suffer from poor functional outcomes.

Compared to the poor outcomes of 31% in idiopathic IVH, the PIVH secondary to AVMs resulted in less poor outcome (11%), which was similar to the previous study (9%).[14,33] Theoretically, sICH had a higher trend of hematoma expansion while the AVMs-related hemorrhage were relatively stable, which could lead to this consequence.[22] The AVMs in cerebellum were more likely to present with hemorrhage compared with AVMs in lobes,[36] and we found none of AVMs in cerebellum presented as PIVH in our series.

Here, PIVH caused by AVMs had better outcomes than other hemorrhage patterns and the location of PIVH-related AVM was common at basal ganglia and thalami.

4.2. Brain ischemia after IVH caused by AVMs

The delayed vasospasm after IVH caused by AVMs has been discussed in previous studies, and it is associated with the brain ischemia.[31,32] They suggested that the patients with vasospasm tended to present in relative young females without SAH; however, the mechanism was still unclear. Other studies showed that the vasospasm occurred in 8% to 31% of SAH caused by AVMs.[37,38] We found that 18% of patients presented with brain ischemia (Fig. 2). For the high occurrence of brain ischemia, the treatment of ruptured AVMs should be consider decreasing the risk of brain ischemia caused by vasospasms. However, increasing cerebral blood flow after the treatment with vasospasms may promote the recurrence of hemorrhage.[38,39] Therefore, early resection of nidus and consideration of brain ischemia were important for the IVH caused by AVMs.[40]
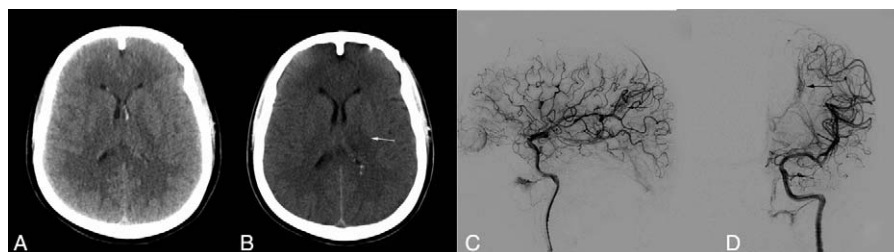
Figure 2. Representative case of brain ischemia after intraventricular hemorrhage caused by ruptured arteriovenous malformations. A 33-year-old woman had intraventricular hemorrhage diagnosed by CT scan (A) with Graeb score of 2. Brain ischemia (hypodense area with white arrow) at the left thalamus at 7 days after onset by CT scan (B). Digital subtraction angiography identified the arteriovenous malformation (C and D) located in peri-ventricle with supplemented Spetzler–Martin score of 4 and combined with an aneurysm in the nidus, supplying by left posterior choroid artery and anterior cerebral artery. CT = computed tomography.
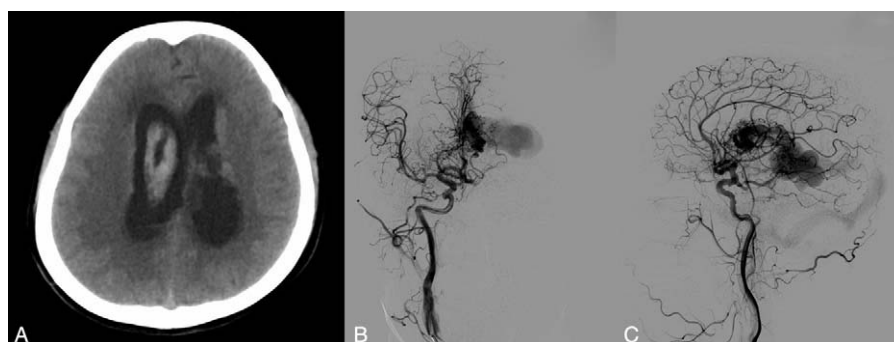
Figure 3. Representative case of hydrocephalus caused by ruptured arteriovenous malformations with intraventricular hemorrhage. A 40-year-old woman had intraventricular hemorrhage diagnosed by computed tomography scan (A), with Graeb score of 4. There was an obvious hydrocephalus with bicaudate index of 0.38 (higher than 0.18). Digital subtraction angiography identified the arteriovenous malformation (B and C) located close to the lateral ventricle with supplemented Spetzler–Martin score of 8, supplying by left anterior cerebral artery, left middle cerebral artery, and left posterior cerebral artery.

4.3. Hydrocephalus caused by AVMs

Hydrocephalus was another major complication of IVH, which could increase the hemorrhage-associated morbidity.[29] In our study, SAH was an independent factor for predicting hydrocephalus (Fig. 3). Same with the hydrocephalus mechanisms in ruptured aneurysm, the cerebrospinal fluid (CSF) circulation within ventricular system was blocked by the obstructive effect of blood products or adhesions[41]; the inflammation after SAH produced the fibrillation can interfere with the absorption of CSF.[42] The amount of blood in ventricular systems could be evaluated by the Graeb score, which is positively correlated to the incidence of hydrocephalus in present study. One previous study[29] reported that the Graeb score was an independent predictor for hydrocephalus for reduce of absorption, or the blockage of channel. Although AVM associated with aneurysm could not predict hydrocephalus in our study, they suggested associated aneurysm were associated with external ventricular drains (EVD) placement.[29] These might be attribute to the placement criteria of EVD while EVD would be placed for patients with aneurysmal SAH.

Although 18.8% of patients with aneurysm SAH presented with rebleeding after EVD,[43] no reported study discussed the rebleeding of ruptured AVMs after EVD. Four cases of our patients presented with rebleeding after hematoma evacuation or EVD; thus, the risk-profit evaluation of EVD for hydrocephalus caused by AVMs should be discussed in future studies.

Here, patients with high Graeb score and SAH had a higher risk of hydrocephalus; however, the treatment for hydrocephalus caused by AVMs warrants more studies.

4.4. Herniation and the necessity of monitoring of vital signs

The presence of herniation also showed a high incidence of 25.7%[30] in secondary ICH and is significantly associated with the mortality and poor outcome in vascular structural abnormality-related intracerebral hemorrhage. Patients with herniation always need urgent treatments to decrease the intracranial pressure (ICP).[30] For the obstruction of CSF, inflammation or vasospasm, patients with IVH had a higher risk of herniation than patients without IVH.[3] In our series, 13 patients (19%) presented a herniation with decreasing of consciousness, and they received emergent surgery to decrease ICP subsequently. The infratentorial AVMs always presented with hemorrhage with a high rate of 72%,[24] which was life threatening for the vital structures in the limited region and always resulted in the herniation. Although the herniation was not significantly associated with the prognostic outcomes at discharge, 4 out of 13 patients who had herniation died in hospital (Table 3). Therefore, closely monitoring of vital signs for the patients with IVH-related AVMs is necessary, especially for infratentorial AVMs, and urgent treatments should be conducted as soon as possible in case of herniation.

4.5. Treatments

The IVH-related AVMs always located close to ventricles or in functional areas and had deep vein drainage,[21] which increased the difficulty for neurosurgeons to resect nidus completely. Using gamma knife or embolization might be better than surgery in stable patients. However, when patients had a progressive deterioration, such as herniation, urgent surgery to decrease ICP was necessary. Until now, the treatments for the IVH caused by AVMs were still controversial without guidelines. Some studies[3,14,44] showed that the unfavorable outcome was associated with emergency hematoma evacuation, which may result in rebleeding of nidus for decreasing ICP or without effective hemostasis during the emergency surgery. As for the patients with progressive deterioration and having operation indication, early resection of nidus and EVD may be better in our study.

4.6. Prognosis

The presence of IVH significantly increased the mortality from 12% to 28% and 29% to 59%[3,6,45,46] in patients with sICH. Some animal models[47,48] supported the conclusion that removal of blood clot in ventricles could improve the consciousness and prevent the inflammation, vasospasm, or hydrocephalus. In accordance with IVH caused by sICH, the presence of IVH also increased the poor outcome in our study (28%), which was higher than the rate of 22% in ruptured AVMs.[14]

However, we found that the amount of intraventricular blood was not associated with clinical outcomes, while the characteristics of AVMs itself and the injury by the parenchymal hematoma affected the outcomes of AVM-related IVH patients. This was attributed to the fact that the ICH-related IVH patients were older than AVM-related IVH patients who had some other

medical comorbidities.[25,26] With increasing of intraventricular blood, the aggravation of consciousness would increase the risk of infections and death.[3] Moreover, the hydrocephalus which had a high incidence in IVH patients increased the mortality in sICH patients[5,49]; however, the hydrocephalus had no significant effect on the 6-month outcomes in our study, which was in accordance with Murthy's research.[35] Furthermore, we found that the PIVH caused by AVMs had a lower risk for poor outcomes (11%) than IVH caused by other causes (33%),[50] which also supported the fact that the influence of IVH to AVM patients was weaker than to IVH caused by other causes.

The sup-SM grading system,[22] including age, hemorrhage, compactness, deep vein drainage, size of nidus, and eloquence, is a well-known tool to predict the outcomes for patients underwent surgery. Some studies demonstrated that the sup-SM grade had a higher predictive accuracy for functional outcomes than the Spetzler–Martin grade.[51,52] We found that the patients with lower GCS, lower sup-SM score, and smaller parenchymal hematoma had better outcomes, and the amount of IVH had a relatively weak effect on the AVMs patients.

4.7. Limitation

This was a retrospective observational study in a single institution. The sample size of our study was small but relatively large compared with previous studies, which may result in the statistical bias. The outcome of this study only included functional outcomes, without the obliteration rate, which may influence the assessment of outcomes. In addition, we could not discuss the effects of treatments on the outcomes because some patients received more than 1 type of treatments, including resection of nidus and hematoma evacuation, ventricular puncture and drainage, lumbar drainage, emergency hematoma evacuation, gamma knife, and embolization.

5. Conclusions

The extension of hemorrhage into ventricles was common in ruptured AVMs. Primary IVH caused by AVMs always located in basal ganglia and thalami and had better outcomes than patients with parenchymal hemorrhage. The hydrocephalus had a high incidence rate in AVM-related IVH patients; the patients with SAH had a higher risk of hydrocephalus and CT reexamination was needed. Considering the high incidence of brain ischemia and herniation, early intervention of nidus was necessary for the ruptured AVMs with IVH. The patients with lower GCS or lower sup-SM score and smaller parenchymal hematoma had better long-term outcomes in our series, while the effect of IVH on the outcomes was relative weak than in IVH from other causes.

Acknowledgment

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