Aneurysm location and clipping versus coiling for development of secondary normal-pressure hydrocephalus after aneurysmal subarachnoid hemorrhage: Japanese Stroke DataBank

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OBJECT The present study aimed to investigate aneurysm locations and treatments for ruptured cerebral aneurysms associated with secondary normal-pressure hydrocephalus (sNPH) after subarachnoid hemorrhage (SAH) by using comprehensive data from the Japanese Stroke DataBank.

METHODS Among 101,165 patients with acute stroke registered between 2000 and 2013, 4693 patients (1482 men, 3211 women) were registered as having had an SAH caused by a ruptured saccular aneurysm. Of them, 1448 patients (438 men and 1010 women; mean age 61.9 ± 13.4 years) who were confirmed to have or not have coexisting acute hydrocephalus and sNPH were included for statistical analyses. Locations of the ruptured aneurysms were subcategorized into 1 of the following 4 groups: middle cerebral artery (MCA; n = 354), anterior communicating artery and anterior cerebral artery (ACA; n = 496), internal carotid artery (ICA; n = 402), and posterior circulation (n = 130). Locations of 66 of the ruptured aneurysms were unknown/unrecorded. Treatments included craniotomy and clipping alone in 1073 patients, endovascular coil embolization alone in 285 patients, and a combination of coiling and clipping in 17 patients. The age-adjusted and multivariate odds ratios from logistic regression analyses were calculated after stratification using the Fisher CT scale to investigate the effects of the hematoma volume of SAH.

RESULTS Acute hydrocephalus was confirmed in 593 patients, and 521 patients developed sNPH. Patients with a ruptured ACA aneurysm had twice the risk for sNPH over those with a ruptured MCA aneurysm. Those with an ACA aneurysm with Fisher Grade 3 SAH had a 9-fold-higher risk for sNPH than those with an MCA aneurysm with Fisher Grade 1 or 2 SAH. Patients with a ruptured posterior circulation aneurysm did not have any significant risk for sNPH. Clipping of the ruptured aneurysm resulted in twice the risk for sNPH over coil embolization alone.

CONCLUSIONS Patients with low-grade SAH caused by a ruptured MCA aneurysm had a low risk for the development of sNPH. In contrast, patients with high-grade SAH caused by a ruptured ACA aneurysm had a higher risk for sNPH. Endovascular coiling might confer a lower risk of developing sNPH than microsurgical clipping.

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KEY WORDS secondary NPH; subarachnoid hemorrhage; acute hydrocephalus; endovascular coil embolization; vascular disorders

ABBREVIATIONS ACA = anterior cerebral artery; ICA = internal carotid artery; MCA = middle cerebral artery; NPH = normal-pressure hydrocephalus; SAH = subarachnoid hemorrhage; sNPH = secondary NPH; WFNS = World Federation of Neurological Societies.

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* See Appendix for listing of institutions participating in the Japan Standard Stroke Registry Study Group.

THE natural history of secondary normal-pressure hydrocephalus (sNPH) occurring 1 or 2 months after subarachnoid hemorrhage (SAH) is becoming clear. The prevalence of sNPH has been reported to be 8.9%–48% in patients with SAH.^{1–7,11,12,16,18,22,23} Acute hydrocephalus coexisting with SAH is known to be one of the most important predictors for sNPH.^{3–6,11,16} Previous studies have shown that severe symptoms at SAH onset and a large amount of subarachnoid blood seen on admission CT images are associated with the development of sNPH.^{3–11,12,16,18,23} A posterior circulation location of the ruptured aneurysm and endovascular coil embolization have been reported to be significantly associated with sNPH.^{1,4–7,12,15,16,22} However, this relationship is controversial, because SAH caused by a posterior circulation aneurysm frequently occurs with severe initial symptoms and acute hydrocephalus, compared to SAH caused by a ruptured anterior circulation aneurysm, which is known to occur with mild symptoms and/or no acute hydrocephalus.¹⁴ In addition, most ruptured posterior circulation aneurysms have been treated with endovascular coil embolization.^{3,5} This selection bias in previous hospital-based studies might have influenced the risk for development of sNPH after coil embolization among patients with a ruptured posterior circulation aneurysm. In the present study, by taking an advantage of a large nationwide registration study, we investigated the risk of sNPH associated with aneurysm location and treatment (clipping or coil embolization) for the ruptured cerebral aneurysm.

Methods

Population

Between 2000 and 2013, the Japanese Standard Stroke Registry Study accumulated data on 101,165 patients with acute stroke treated in 163 institutions across Japan.^{10,20} This nationwide stroke database was established with support from the Japanese Ministry of Health, Labor and Welfare to provide evidence for the standardization of Japanese stroke management. The present study was approved by the ethics committee of the Shimane Medical University. Details of data collection and management have been published elsewhere.^{10,20} A total of 5344 patients (5.3% of the registered population; 1772 men and 3572 women; mean age 62.5 ± 14.4 years) were registered as having had an SAH. Among them, 4693 patients were diagnosed with a ruptured saccular aneurysm, 196 had a dissecting aneurysm, 27 had an infectious or traumatic pseudo-aneurysm, and 428 had an aneurysm for which the origin was unknown or not recorded. In this study, the patients confirmed to have had a ruptured cerebral saccular aneurysm were included because of the different pathological characteristics of dissecting and pseudoaneurysms. In addition, to clarify the relationship between acute hydrocephalus and sNPH, this study included the patients who were confirmed to have or not have coexisting acute hydrocephalus and sNPH, as diagnosed by the attending neurosurgeons at the registered hospitals. We also gathered data on patients diagnosed with acute hydrocephalus who experienced neurological deterioration and underwent immediate CSF diversion by placement of an external ventricular or lumbar drain on admission. The records of patients with

sNPH after SAH were also reviewed to note their symptoms and whether they underwent ventriculoperitoneal or lumboperitoneal shunt placement for permanent CSF diversion. Finally, 1448 patients (438 men and 1010 women; mean age 61.9 ± 13.4 years) were included for statistical analyses. The database recorded age, sex, medical history, medication status, family history of stroke, lifestyle factors, neurological severity grading at SAH onset (NIH Stroke Scale, World Federation of Neurological Societies [WFNS] scale, and Hunt and Kosnik scale), Fisher CT scale grade (from the admission CT scan), and treatment for the ruptured cerebral aneurysm (neurosurgical clipping and/or endovascular coil embolization). Locations of the ruptured cerebral aneurysms were categorized as follows: middle cerebral artery (MCA; $n = 354$), anterior communicating artery ($n = 416$), anterior cerebral artery (ACA; $n = 80$), internal carotid–posterior communicating artery ($n = 395$), other internal carotid artery (ICA; $n = 7$), posterior cerebral artery ($n = 14$), basilar tip ($n = 48$), basilar–superior cerebellar artery ($n = 21$), vertebral artery–posterior inferior cerebellar artery ($n = 47$), and unknown or unrecorded ($n = 66$). The aneurysm locations were subcategorized into 1 of the following 4 groups: MCA, ACA (which included the ACA and the anterior communicating artery), ICA (which included the ICA–posterior communicating artery and other ICAs), and posterior circulation (which included the posterior cerebral artery, basilar tip, basilar–superior cerebellar artery, and vertebral artery–posterior inferior cerebellar artery).

Statistical Analysis

Odds ratios and 95% CIs for the development of sNPH were calculated. Using logistic regression analyses, we investigated the association between sNPH and the following variables: age (< 60 or ≥ 60 years), sex, acute hydrocephalus, craniotomy and microsurgical clipping or endovascular coil embolization, hypertension, diabetes, dyslipidemia, smoking habit, alcohol consumption, WFNS scale grade, Hunt and Kosnik scale grade, and Fisher CT scale grade. All analyses were adjusted by the continuous variable of age at registration. To assess the effects of modification and interaction, we conducted multivariate analyses after adjusting for age, sex, hypertension, smoking habit, acute hydrocephalus, and Fisher CT scale grade. In addition, to investigate the effects of acute hydrocephalus and hematoma volume, age-adjusted and multivariate odds ratios were analyzed after stratification according to acute hydrocephalus (versus no acute hydrocephalus) and Fisher CT scale grade. All missing variables were treated as deficit data that did not change the other variables. Statistical significance was assumed at a Fisher exact test probability value (p) of < 0.05 . Statistical analyses were performed using R software (version 3.1.2, R Foundation for Statistical Computing; <http://www.R-project.org>).

Results

The clinical characteristics of the 1448 patients diagnosed with SAH caused by a ruptured cerebral saccular aneurysm are summarized in Table 1. Of them, 521 were registered as having had sNPH after aneurysmal SAH, and 444 (85%) of these patients underwent shunt surgery.

TABLE 1. Clinical characteristics in each location of ruptured cerebral aneurysm on admission*

Characteristic†	Aneurysm Location					
	All	MCA	ACA	ICA	Posterior Circulation	Unknown/Unrecorded
No.	1448	354	496	402	130	66
Age (yrs)	61.9 ± 13.4	61.6 ± 12.7	60.8 ± 13.4	62.6 ± 14.2	64.1 ± 13.2	62.8 ± 12.2
Female sex	1010 (70)	248 (70)	288 (58)	322 (80)	113 (87)	39 (59)
Acute hydrocephalus	593 (41.0)	120 (33.9)	218 (44.0)	152 (37.8)	79 (60.8)	24 (36.4)
Clipping alone	1073 (74.1)	320 (90.4)	385 (77.6)	296 (73.6)	51 (39.2)	21 (31.8)
Coiling alone	285 (19.7)	20 (5.7)	97 (19.6)	91 (22.6)	70 (53.9)	7 (10.6)
Hypertension	718 (49.6)	189 (53.4)	241 (48.6)	181 (45.0)	74 (56.9)	33 (50.0)
Diabetes mellitus	112 (7.7)	20 (5.7)	34 (6.9)	34 (8.5)	11 (8.5)	13 (19.7)
Dyslipidemia	180 (12.4)	39 (11.0)	65 (13.1)	54 (13.4)	17 (13.1)	5 (7.6)
Smoking habit	444 (30.7)	114 (32.2)	177 (35.7)	102 (25.4)	33 (25.4)	18 (27.3)
Alcohol consumption	499 (34.5)	135 (38.1)	192 (38.7)	117 (29.1)	30 (23.1)	25 (37.9)
NIH stroke scale	13.2 ± 16.1	13.29 ± 15.7	12.4 ± 16.2	12.5 ± 16.1	16.1 ± 16.4	15.1 ± 17.9
WFNS scale						
Grade I	484 (33.4)	116 (32.8)	169 (34.1)	145 (36.1)	27 (20.8)	27 (40.9)
Grade II	360 (24.9)	71 (20.1)	134 (27.0)	100 (24.9)	41 (31.5)	14 (21.2)
Grade III	124 (8.6)	35 (9.9)	45 (9.1)	32 (8.0)	9 (6.9)	3 (4.6)
Grade IV	282 (19.5)	86 (24.3)	79 (15.9)	76 (18.9)	33 (25.4)	8 (12.1)
Grade V	194 (13.4)	44 (12.4)	69 (13.9)	47 (11.7)	20 (15.4)	14 (21.2)
Hunt & Kosnik scale						
Grade I	160 (11.0)	36 (10.2)	61 (12.3)	40 (10.0)	9 (6.9)	14 (21.2)
Grade II	568 (39.2)	132 (37.3)	198 (39.9)	168 (41.8)	46 (35.4)	24 (36.4)
Grade III	326 (22.5)	76 (21.5)	110 (22.2)	93 (23.1)	38 (29.2)	9 (13.6)
Grade IV	248 (17.1)	78 (22.0)	76 (15.3)	69 (17.2)	19 (14.6)	6 (9.1)
Grade V	144 (9.9)	32 (9.0)	51 (10.3)	30 (7.5)	18 (13.8)	13 (19.7)
Fisher CT scale						
Grade 1	41 (2.8)	7 (2.0)	10 (2.0)	14 (3.5)	5 (3.9)	5 (7.6)
Grade 2	220 (15.2)	49 (13.8)	66 (13.3)	77 (19.2)	16 (12.3)	12 (18.2)
Grade 3	957 (66.1)	217 (61.3)	345 (69.6)	263 (65.4)	95 (73.1)	37 (56.1)
Grade 4	227 (15.7)	81 (22.9)	73 (14.7)	47 (11.7)	14 (10.8)	12 (18.2)

* Values are mean ± SD or number (%).

† Data for some patients were not recorded: treatment methods (clipping or coiling), 9 patients (3 with MCA aneurysm, 3 with ACA aneurysm, 2 with ICA aneurysm, 1 with no aneurysm location information); history of hypertension, 41 patients (9 with MCA aneurysm, 16 with ACA aneurysm, 13 with ICA aneurysm, 3 with no aneurysm location information); history of diabetes mellitus, 29 patients (11 with MCA aneurysm, 6 with ACA aneurysm, 11 with ICA aneurysm, 1 with posterior circulation aneurysm); history of dyslipidemia, 108 patients (24 with MCA aneurysm, 34 with ACA aneurysm, 38 with ICA aneurysm, 8 with posterior circulation aneurysm, 4 with no aneurysm location information); smoking habits, 249 patients (66 with MCA aneurysm, 90 with ACA aneurysm, 63 with ICA aneurysm, 22 with posterior circulation aneurysm, 8 with no aneurysm location information); alcohol consumption, 248 patients (64 with MCA aneurysm, 91 with ACA aneurysm, 62 with ICA aneurysm, 24 with posterior circulation aneurysm, 7 with no aneurysm location information); WFNS scale, 4 patients (2 with MCA aneurysm, 2 with ICA aneurysm); Hunt & Kosnik scale, 2 patients (with ICA aneurysm); and Fisher CT scale, 3 patients (2 with ACA aneurysm, 1 with ICA aneurysm).

SAH caused by ACA aneurysm rupture tended to have lower grades on the WFNS and Hunt and Kosnik scales and was seen in younger males, and 78% of the aneurysms were treated with clipping alone. SAH caused by posterior circulation aneurysms occurred more often in older females, had a higher concurrence of acute hydrocephalus, had higher grades on the WFNS, Hunt and Kosnik, and Fisher scales, and was treated more often with endovascular coiling rather than with microsurgical clipping. Table 2 lists the age-adjusted and multivariate odds ratios for the development of sNPH. Significant independent predictors for the development of sNPH were age of 60 years or older, concurrence of acute hydrocephalus, and high grades on the WFNS, Hunt and Kosnik, and Fisher scales.

Patients with a ruptured ACA aneurysm had twice the risk for sNPH than those with a ruptured MCA aneurysm; ruptured posterior circulation aneurysms did not confer any significant risk for sNPH (Table 2). The sNPH risk in patients with an ACA aneurysm did not change even after stratification for the coexistence of acute hydrocephalus (data not shown). For patients with Fisher CT Group 1 or 2 SAH, which indicates a small hematoma volume, the ACA and ICA aneurysms conferred a 5.5- and 2.5-times-higher risk for sNPH than MCA aneurysms, respectively (Table 3). In the subgroup of patients with Fisher Group 3 SAH, the sNPH risk conferred by ACA aneurysms was 1.8 times higher than that by the MCA aneurysms. In the subgroup of patients with Fisher Group 4 SAH, however,

TABLE 2. Risk of coexisting sNPH after aneurysmal SAH

Variable	sNPH	No sNPH	aOR* (95% CI)	p Value†	mOR‡ (95% CI)	p Value†
Age (≥60 yrs)	366	456	2.44 (1.94–3.10)	<0.001	1.96 (1.47–2.62)	<0.001
Acute hydrocephalus	321	272	3.62 (2.87–4.57)	<0.001	3.60 (2.77–4.69)	<0.001
Aneurysm location						
MCA	104	250	Reference		Reference	
ACA	211	285	1.93 (1.42–2.61)	<0.001	2.02 (1.39–2.92)	<0.001
ICA	143	259	1.12 (0.96–1.32)	0.160	1.14 (0.94–1.39)	0.180
Posterior circulation	47	83	1.06 (0.91–1.23)	0.450	1.06 (0.88–1.28)	0.530
Treatment						
Coiling alone	95	190	Reference		Reference	
Clipping alone	395	678	1.59 (1.18–2.14)	0.002	1.97 (1.36–2.85)	<0.001
Hunt & Kosnik scale						
Grade I or II	159	569	Reference		Reference	
Grade III	150	176	2.92 (2.17–3.91)	<0.001	2.32 (1.63–3.29)	<0.001
Grade IV or V	212	180	3.78 (2.88–4.96)	<0.001	2.62 (1.85–3.73)	<0.001
Fisher CT rating scale						
Grade 1 or 2	52	209	Reference		Reference	
Grade 3	362	595	2.15 (1.53–3.01)	<0.001	1.95 (1.30–2.91)	0.001
Grade 4	106	121	3.34 (2.21–5.05)	<0.001	2.99 (1.75–5.10)	<0.001

* aOR indicates the age-adjusted OR from logistic regression analysis.

† Boldface indicates statistically significant variables.

‡ mOR indicates the multivariate OR for sNPH after adjustment for age, sex, hypertension, smoking habit, acute hydrocephalus, and Fisher grade.

there was no significant difference among the 4 aneurysm locations. ACA aneurysms in patients with Fisher Group 3 SAH conferred a 9-fold-higher risk for sNPH than MCA aneurysms in patients with Fisher Group 1 or 2 SAH.

Among these 1448 patients, 1073 were treated with craniotomy and clipping alone, 285 were treated with coil embolization alone, and 17 underwent a combination of coiling and clipping. Microsurgical clipping conferred a 2-fold-increased risk for sNPH over coil embolization alone (Table 2). This advantage of coil embolization for the reduction of sNPH risk did not change in the subgroup of patients with Fisher Grade 3 SAH (Table 3). In the subgroup of patients with Fisher Grade 1, 2, or 4 SAH, however, there was no statistically significant difference between clipping and coiling in terms of sNPH risk.

Discussion

Our large registration study revealed that ruptured ACA aneurysms conferred a significantly higher risk of sNPH than ruptured MCA aneurysms. Gruber et al.⁷ reported that ruptured aneurysms located in the anterior communicating artery conferred a significant increased risk for the development of sNPH compared with those in other locations ($p < 0.001$). Other previous studies concluded that patients with a ruptured posterior circulation aneurysm most frequently developed sNPH;^{1,5,6,12,16,17,21} 3 of these 7 studies reported that patients with an anterior communicating artery or ACA aneurysm are the second-most frequent to develop sNPH and that developing sNPH after an MCA aneurysm is rare.^{6,17,21} The other 4 studies did not further categorize the anterior circulation aneurysms as being located in the ACA, MCA, or ICA.^{1,5,12,16} The

sNPH risk conferred by posterior circulation aneurysms may be affected by the other modified variables, such as intraventricular hemorrhage and acute hydrocephalus, for which the frequency was reported to be the highest in patients with a ruptured posterior circulation aneurysm.^{9,21} We confirmed that posterior circulation aneurysms co-occurred with acute hydrocephalus more frequently and were treated more often with endovascular coiling than aneurysms in the other locations in this study. Although the patients' characteristics were similar to those in the previous studies, posterior circulation aneurysms did not confer any significantly increased risk for the development of sNPH. SAH in the interhemispheric fissure may contribute to the pathogenesis of a higher prevalence of sNPH with ruptured ACA aneurysms, because it would become an obstacle to the CSF-drainage pathway into the nasal lymphatic system via the perineural subarachnoid space enveloping the olfactory nerve rootlets.^{13,24}

In addition, we also confirmed that increasing age, severe initial symptoms, a large-volume diffuse subarachnoid hematoma, and concurrence of acute hydrocephalus were correlated significantly with the development of sNPH; these findings are supported by previous studies.^{3–7,11,12,16,18,23} On the basis of the evidence of an association between the severity of SAH and the prevalence of subsequent sNPH, many neurosurgeons have believed that several surgical manipulations for facilitating CSF dynamics, such as hematoma evacuation, widening the opening of the cisterns, and fenestration of the lamina terminalis, might help to reduce the subsequent occurrence of sNPH after the surgical treatment of SAH. However, the authors of a recent meta-analysis of 11 nonrandomized studies in which data from 1973 patients were pooled concluded that

TABLE 3. Risk of coexisting sNPH after aneurysmal SAH in the Fisher CT rating scale subgroups

Variable	sNPH	No sNPH	mOR* (95% CI)	p Value†
Fisher Grade 1 or 2	52	209		
Age (≥60 yrs)	36	93	2.28 (0.96–5.4)	0.061
Acute hydrocephalus	35	34	11.1 (4.64–26.5)	<0.001
MCA aneurysm	4	52	Reference	
ACA aneurysm	16	60	5.50 (1.20–25.4)	0.028
ICA aneurysm	22	69	2.50 (1.10–5.70)	0.029
Posterior circulation aneurysm	5	16	0.92 (0.39–2.20)	0.848
Coiling alone	8	39	Reference	
Clipping alone	36	158	1.67 (0.47–6.00)	0.430
Hunt & Kosnik scale Grade I or II	33	189	Reference	
Hunt & Kosnik scale Grade III	9	15	1.70 (0.46–6.28)	0.420
Hunt & Kosnik scale Grade IV or V	10	4	7.00 (6.10–790)	<0.001
Fisher Grade 3	362	595		
Age (≥60 yrs)	259	301	1.94 (1.38–2.74)	<0.001
Acute hydrocephalus	217	195	3.04 (2.24–4.14)	<0.001
MCA aneurysm	70	147	Reference	
ACA aneurysm	154	191	1.76 (1.15–2.70)	0.009
ICA aneurysm	92	171	1.03 (0.82–1.29)	0.820
Posterior circulation aneurysm	36	59	1.05 (0.85–1.29)	0.670
Coiling alone	76	140	Reference	
Clipping alone	257	419	1.90 (1.28–2.83)	0.002
Hunt & Kosnik scale Grade I or II	107	343	Reference	
Hunt & Kosnik scale Grade III	129	138	2.41 (1.64–3.56)	<0.001
Hunt & Kosnik scale Grade IV or V	126	114	2.42 (1.61–3.64)	<0.001
Fisher Grade 4	106	121		
Age (≥60 yrs)	70	61	2.40 (1.12–5.05)	0.024
Acute hydrocephalus	68	43	3.29 (1.63–6.66)	<0.001
MCA aneurysm	30	51	Reference	
ACA aneurysm	40	33	2.28 (0.88–5.90)	0.090
ICA aneurysm	29	18	1.49 (0.88–2.52)	0.140
Posterior circulation aneurysm	6	8	0.93 (0.53–1.60)	0.786
Coiling alone	11	11	Reference	
Clipping alone	91	93	5.00 (0.86–29.2)	0.073
Hunt & Kosnik scale Grade I or II	18	36	Reference	
Hunt & Kosnik scale Grade III	12	23	2.09 (0.54–8.02)	0.284
Hunt & Kosnik scale Grade IV or V	76	62	2.56 (1.10–5.96)	0.029

* mOR indicates the multivariate OR for sNPH after adjustment for age, sex, hypertension, smoking habit, acute hydrocephalus, and Fisher CT rating scale grade.

† Boldface indicates statistically significant variables.

there was no significant difference in the prevalence of sNPH between the 975 patients who had undergone fenestration of the lamina terminalis and the 998 who had not ($p = 0.09$).¹¹ On the basis of this evidence, guidelines for the management of chronic hydrocephalus after aneurysmal SAH from the American Heart Association recommend that fenestration of the lamina terminalis not be routinely performed.² Another meta-analysis of 5 nonrandomized studies with data from 1718 patients pooled³ reported that 1336 patients treated with microsurgical clipping had a significantly lower risk of sNPH than 382 patients treated with endovascular coiling (relative risk 0.74; 95% CI

0.58–0.94; $p = 0.01$); however, 3 of the 5 studies found no significant difference between clipping and coiling for the predictive risk of sNPH.^{8,15,19} Our results provide the first evidence that, compared with microsurgical clipping alone, coil embolization alone significantly decreased the risk for the development of sNPH. This finding may support the view that microsurgical manipulation accelerates inflammation of CSF in the subarachnoid space and leads to stagnating CSF that results from thickness and fibrosis of the arachnoid and pia mater.

The advantage of this study includes a much larger sample size than those of previous studies. Despite this

advantage, a register-based study also has some limitations, such as recording bias. In fact, one-third of the 4693 patients registered as having had aneurysmal SAH were included in this study, and 95% of them were treated with coiling or clipping.

Conclusions

Our findings indicate that sNPH after SAH frequently occurs among patients with high-grade SAH caused by a ruptured ACA aneurysm and patients treated with microsurgical clipping. On the contrary, patients with low-grade SAH caused by a ruptured MCA aneurysm and patients treated with endovascular coiling might be less likely to develop sNPH. Studies are needed to elucidate the mechanisms by which ruptured ACA aneurysms and microsurgical treatment can increase the risk for developing sNPH after SAH.

Appendix: Japan Standard Stroke Registry Study Group

The institutions that presently contribute to the registration of hospital records for cerebral strokes are as follows:

1. Teine Keijinkai Hospital, Department of Neurosurgery
2. Hakodate Neurosurgical Hospital, Department of Neurosurgery
3. Hokkaido Neurosurgical Memorial Hospital, Department of Neurosurgery
4. Nakamura Memorial Hospital, Stroke Center
5. Hokkaido University Hospital, Department of Neurosurgery
6. Keiwakai Ebetsu Hospital, Department of Neurosurgery
7. Asahikawa Medical University Hospital, Department of Neurology
8. Rumoi Central Clinic, Department of Neurosurgery
9. Iwate Medical University, Department of Neurology and Neurosurgery
10. Iwate Prefectural Kuji Hospital, Department of Neurosurgery
11. Southern Tohoku Research Institute for Neuroscience Hospital, Stroke Center
12. Kohnan Hospital, Stroke Center
13. Research Institute for Brain and Blood Vessels Akita, Stroke Center
14. Saiseikai Central Hospital, Department of Neurology
15. Kanto Medical Center NTT East Corporation, Department of Neurosurgery
16. Keio University School of Medicine, Department of Neurology
17. National Center for Global Health and Medicine, Neurosurgery
18. Hatanodai Neurosurgical Hospital, Department of Neurosurgery
19. Metropolitan Ohkubo Hospital, Department of Neurosurgery
20. Tokyo Women's Medical University Hospital, Department of Neurology
21. Kyorin University Hospital, Department of Neurosurgery
22. Akiru Municipal Medical Center, Department of Neurosurgery
23. Tokyo Medical University Hachioji Medical Center, Department of Neurosurgery
24. Yokohama Rosai Hospital, Department of Neurology
25. Kawasaki Saiwai Hospital, Department of Neurosurgery
26. Sagamiyama Kyoudo Hospital, Department of Neurosurgery
27. Yokohama Stroke & Brain Center
28. Yokohama City University Hospital, Stroke Center
29. Shonankamakura General Hospital, Stroke Center
30. Tokai University, Department of Oiso Hospital, Department of Neurology
31. Tokai University Hospital, Department of Neurology
32. Tokyo Dental College Ichikawa General Hospital, Department of Neurosurgery
33. Jisenkai Yoshida Hospital, Department of Neurosurgery
34. Chiba Rosai Hospital, Department of Neurosurgery
35. Chiba Cardiovascular Center, Stroke Center
36. Dokkyo University School of Medicine, Department of Neurology
37. Ryugasaki Saiseikai Hospital, Department of Neurology
38. Saitama International Medical Research Center Hospital, Stroke Center
39. Hiratsuka Kyosai Hospital, Stroke Center
40. Saitama Medical University Hospital, Department of Neurology
41. Fukui University Hospital, Department of Neurology
42. Toyama University Hospital, Department of Neurology and Neurosurgery
43. Shizuoka General Hospital, Department of Neurology
44. Matsunami General Hospital, Department of Neurosurgery
45. Shiga University of Medical Science Hospital, Department of Neurology and Neurosurgery
46. Osaka University Faculty of Medicine, Department of Neurology
47. National Cardiovascular Center, Department of Cerebrovascular Disease
48. Hoshigaoka Koseinenkin Hospital, Department of Neurology
49. Kyoto Second Red Cross Hospital, Department of Neurology
50. Kyoto Prefectural University of Medicine, Department of Neurology
51. Otowa Hospital, Department of Neurology
52. Itami City Hospital, Department of Neurosurgery
53. Kobe City General Hospital, Stroke Center
54. Hyogo Brain and Heart Center, Department of Neurology
55. Matsue City Hospital, Department of Neurosurgery
56. Shimane Prefectural Central Hospital, Department of Neurology and Neurosurgery
57. Shimane University Hospital, Department of Neurology and Neurosurgery
58. Okayama Rosai Hospital, Department of Neurosurgery
59. Matsue Red Cross Hospital, Department of Neurology
60. Ohda Municipal Hospital, Department of Neurology
61. Masuda Red Cross Hospital, Department of Neurology
62. Kawasaki Medical School Hospital, Stroke Center
63. National Okayama Medical Center, Department of Neurology
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