

Subarachnoid Hemorrhage from Ruptured Aneurysm in Sawanpracharak Hospital

Nrongpong Lowprukmanee MD*

* Department of Surgery, Sawanpracharak Hospital, Nakhonsawan, Thailand

Objective: To study the relationship of factors associated with clinical outcome in surgical groups of aneurysmal subarachnoid hemorrhage patients (SAH).

Material and Method: The data were retrospectively collected from surgically treated SAH patients who had surgery at Sawanpracharak Hospital between January 2007 and March 2016. The following risk factors were studied: hypertension, diabetes mellitus, hyperlipidemia, heart disease, previous stroke, family history of stroke, hematologic disease, history of medication, smoking, alcoholic consumption, obesity, Glasgow Coma Scale, doll's eye reflex, pupillary examination, size of aneurysm, midline shift, intraventricular bleeding, subdural hematoma, hydrocephalus, convulsion, pneumonia, rebleeding, vasospasm, postoperative brain edema, postoperative brain infarction, intraoperative aneurysm rupture, bacterial meningitis, location of aneurysm, ventriculoperitoneal (VP) shunt, ventriculostomy, tracheostomy, time to surgery, hospital stay, operating time, and intraoperative blood loss.

Results: One hundred sixty five patients with SAH underwent surgical treatment. Factors that were statistically significant related to outcome of SAH were postoperative brain edema [OR 3.79 (1.35 to 10.69), $p = 0.01$], intraoperative rupture [OR 2.59 (1.16 to 5.80), $p = 0.02$], MCA aneurysm [OR 0.23 (0.08 to 0.69), $p = 0.05$], ventriculostomy [OR 3.04 (1.32 to 6.99), $p = 0.009$], VP shunt [OR 0.08 (0.01 to 0.74), $p = 0.026$], and doll's eye reflex [OR 0.14 (0.03 to 0.47), $p = 0.002$].

Conclusion: Outcome of surgical treatment of SAH were related to postoperative brain edema, intraoperative rupture, MCA aneurysm, ventriculostomy, VP shunt, and doll's eye reflex.

Keywords: Aneurysmal subarachnoid hemorrhage, Glasgow Coma Scale, Intraventricular bleeding, Hydrocephalus, Glasgow Outcome Scale

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Stroke remains a major cause of mortality and disability worldwide. Subarachnoid hemorrhage (SAH, most from aneurysm) accounts for only 3% of all strokes⁽¹⁾. The incidence of SAH has been stable over past 30 years. In a meta-analysis of relevant studies, the pooled incidence rate was 10.5 per 100,000 patient year⁽²⁾. SAH from rupture of an aneurysm arising from the circle of Willis kills or disables approximately 18,000 individuals in North America each years^(3,4). Despite improved medical and operative treatment, SAH is still a severe disease with an overall mortality of 40 to 45%, about 30% of long-term survivors are severely handicapped and only another 30% are considered as having a good neurological outcome⁽⁵⁾. The most fearful complications in the acute state after aneurysmal SAH are hydrocephalus and rebleeding as well as vasospasm with subsequent delayed ischemic neurological deficit (DIND).

Aneurysmal SAH is associated with a high risk of morbidity and mortality. Early aneurysm repair treatment is required to prevent catastrophic rebleeding and intensive medical care to manage associated problems including hydrocephalus, cerebral vasospasm, electrolyte disorders, infection, and seizures. Rosengart et al⁽⁶⁾ analyzed a large series of patients with ruptured aneurysms and determined that the most important factors leading to unfavorable outcome were cerebral infarction, worse clinical grade on admission, advanced age, fever, and symptomatic vasospasm. Others significant variables were greater clot thickness on admission CT scan, posterior circulation aneurysms, large aneurysms, intraventricular or intracerebral hemorrhage, anticonvulsant use and not using hypertensive hypervolemic therapy. Patient outcome is often evaluated by the Glasgow Outcome Scale (GOS), which was developed for grading neurological outcome following head injury and is easy to perform with a good correlation to quality-of-life and general assessment of health. The GOS is commonly used for the evaluation of neurological outcome after SAH.

Correspondence to:

Lowprukmanee N; Department of Surgery, Sawanpracharak Hospital, Nakhonsawan 60000, Thailand.
Phone: +66-95-6346872
E-mail: doctor-bair@hotmail.com

Material and Method

This is a retrospective analysis of patients who were admitted to the Department of Surgery, Sawanpracharak Hospital, Nakhornsawan province between January 2007 and March 2016. The research proposal had been reviewed and approved by the Sawanpracharak Hospital Ethic Committee. In all patients suspected of aneurysmal SAH, computerized tomography (CT) scan and computerized tomographic angiography (CTA) of the brain were done at the hospital. All CT and CTA scan findings were evaluated by neurosurgeon and a radiologist.

The inclusion criterion for SAH in the present study were cases of SAH from artery of anterior circle of Willis (anterior cerebral artery, anterior communicating artery, middle cerebral artery (MCA), posterior communicating artery, and internal carotid artery). Patients who had SAH from artery of posterior circle of Willis (vertebral artery, posterior inferior cerebellar artery, anterior inferior cerebellar artery, superior cerebellar artery, basilar artery) were excluded from the study.

Personal data including sex, age, major risk factors (e.g., hypertension, diabetes mellitus, hyperlipidemia, heart disease, previous stroke), minor risk factors (e.g., family history of stroke, hematologic disease, history of medication, smoking, alcoholic consumption, obesity), Glasgow Coma Scale (GCS), doll's eye reflex, pupillary examination, size of aneurysm, midline shift (MS), postoperative brain edema, intraventricular bleeding, subdural hematoma, hydrocephalus, location of aneurysm, pneumonia, rebleeding, vasospasm, convulsion, postoperative brain infarction, bacterial meningitis, intraoperative aneurysm rupture (IAR), ventriculoperitoneal (VP) shunt, ventriculostomy, tracheostomy, time to surgery, hospital stay, operating time, and intraoperative blood loss were collected.

Severity of neurological status was classified by the GCS into 3 groups (severe GCS ≤ 8 , moderate 9 to 12, and mild GCS 13 to 15). MS was classified into 4 groups (MS 0, MS 1 to 5 mm, MS 6 to 10 mm, and MS >10 mm). Outcome of the patients was classified by the GOS, in the present study GOS was grouped into two groups: 1) GOS 2 to 5 the patients alive, 2) GOS 1 the patients dead.

All SAH patients were surgically treated. Furthermore, those with postoperative hydrocephalus and rebleeding were appropriately treated surgically.

The study was approved by the Ethics Committee, Sawanpracharak Hospital, Nakhornsawan.

Statistical analysis

Statistical analyzes were performed using the STATA 10 SE. The characteristics of the subjects were described in terms of frequency and percentage. Student's t-test was used for comparison of continuous quantitative variables and Chi-square test were used for discrete data. The association between the groups was measured using the odds ratio with 95% confidence intervals for every prognostic factor. Only variables with a p -value <0.05 in the separate analyses were selected and studied in the logistic regression analyses. For all statistical tests used, $p < 0.005$ was considered statistically significant.

Results

There were 165 consecutive SAH patients admitted to the Department of Surgery at Sawanpracharak Hospital. Comparing outcome of the patients regarding demographic data and risk factors, the mean age \pm SD was 56.1 ± 11.74 years in alive group, 62.8 ± 12.45 years in dead group. There was statistically significant association between outcome and family history of stroke ($p = 0.044$) (Table 1).

On the analysis of clinical finding and CT characteristics, there was statistically significant association between outcome and GCS ($p = 0.001$), doll's eye reflex ($p < 0.001$), pupillary examination ($p = 0.001$), MS ($p < 0.001$), intraventricular bleeding ($p = 0.005$), hydrocephalus ($p = 0.05$), MCA aneurysm ($p = 0.05$), postoperative brain edema ($p = 0.004$), postoperative brain infection ($p = 0.05$), IAR ($p = 0.001$), VP shunt ($p = 0.05$), ventriculostomy ($p = 0.05$), time to operation ($p = 0.028$), hospital stay ($p = 0.05$), and intraoperative blood loss ($p = 0.05$) (Table 2, 3).

After univariate logistic regression analysis was done (Table 4) and multivariate logistic regression analysis was done, the present study found that factors affecting clinical outcome of the patients were post-operative brain swelling [OR 3.82 (1.35 to 10.69), $p = 0.011$], IAR [OR 2.62 (1.17 to 5.87), $p = 0.019$], MCA aneurysm [OR 0.24 (0.08 to 0.70), $p = 0.009$], VP shunt [OR 0.08 (0.01 to 0.74), $p = 0.026$], ventriculostomy [OR 3.08 (1.34 to 7.08), $p = 0.008$], and doll's eye reflex [OR 0.14 (0.03 to 0.47), $p = 0.002$] (Table 5).

Discussion

SAH is a significant health care problem and associate with a high risk of morbidity and mortality. SAH has an annual incidence of 10 per 100,000 inhabitants in Europe and 20 to 30 per 100,000 inhabitants in Finland and Japan⁽⁷⁾. Treatment

Table 1. Demographic feature, clinical characteristics, risk factors, and clinical outcome of SAH patients

Risk factor	Alive (n = 110) n (%)	Dead (n = 55) n (%)	p-value
Age (year)			0.055
<40	11 (10.00)	1 (1.82)	
41 to 50	22 (20.00)	9 (16.36)	
51 to 60	40 (36.36)	16 (29.09)	
61 to 70	21 (19.09)	12 (21.82)	
>70	16 (14.55)	17 (30.91)	
Mean ± SD	56.1±11.74	62.8±12.45	
Sex			0.228
Male	36 (32.72)	13 (23.63)	
Female	74 (67.28)	42 (76.37)	
Hypertension			0.693
No	10 (9.09)	4 (7.27)	
Yes	100 (90.91)	51 (92.73)	
Diabetes mellitus			0.159
No	78 (70.90)	33 (60.00)	
Yes	32 (29.09)	22 (40.00)	
Blood lipids (mg/dl)			0.098
≤200	87 (79.09)	37 (67.27)	
>200	23 (20.91)	18 (32.73)	
Heart disease			0.307
No	103 (93.64)	49 (89.09)	
Yes	7 (6.36)	6 (10.91)	
Previous stroke			0.146
No	106 (96.36)	50 (90.91)	
Yes	4 (3.64)	5 (9.09)	
Hereditary			0.044
No	110 (100)	53 (96.36)	
Yes	0 (0)	2 (3.64)	
Hematologic disease			0.378
No	107 (97.27)	52 (94.55)	
Yes	3 (2.73)	3 (5.45)	
Medication			1.000
No	108 (98.18)	54 (98.18)	
Yes	2 (1.82)	1 (1.82)	
Smoking			0.227
No	90 (81.82)	49 (89.09)	
Yes	20 (18.18)	6 (10.91)	
Alcohol consumption			0.424
No	84 (76.36)	45 (81.82)	
Yes	26 (23.64)	10 (18.18)	
Obesity			0.077
No	83 (75.45)	48 (87.27)	
Yes	27 (24.55)	7 (12.73)	

SAH = subarachnoid hemorrhage

requires early aneurysm repair to prevent catastrophic rebleeding.

SAH is a complex pathophysiological event and outcome is determined by many variables. There have been numerous attempts to identify outcome predictors for SAH. Several prognostic models had been proposed and validated to help clinicians in

Table 2. Clinical profile, CT characteristics, and clinical outcome of SAH patients

Risk factor	Alive (n = 110) n (%)	Dead (n = 55) n (%)	p-value
Glasgow Coma Scale			0.001
3 to 8	10 (9.09)	22 (40.00)	
9 to 12	26 (23.64)	18 (32.73)	
13 to 15	74 (67.27)	15 (27.27)	
Doll's eye			<0.001
No	5 (4.55)	14 (25.45)	
Yes	105 (95.45)	41 (74.55)	
Pupil			0.001
No	6 (5.45)	15 (27.27)	
Yes	104 (94.55)	40 (72.73)	
Size of aneurysm (mm)			0.955
1-5	47 (42.73)	24 (43.64)	
6-10	58 (52.73)	28 (50.91)	
>10	5 (4.54)	3 (5.45)	
Midline shift (mm)			0.001
No	73 (66.36)	31 (56.37)	
1-5	25 (22.73)	5 (9.09)	
6-10	8 (7.27)	9 (16.36)	
>10	4 (3.64)	10 (18.18)	
Brain swelling			0.004
No	99 (90.00)	40 (72.73)	
Yes	11 (10.00)	15 (27.27)	
Intraventricular bleeding			0.005
No	40 (36.36)	8 (14.55)	
Yes	70 (63.64)	47 (85.45)	
Subdural hematoma			0.525
No	101 (91.82)	52 (94.55)	
Yes	9 (8.18)	3 (5.45)	
Hydrocephalus			0.050
No	38 (34.55)	11 (20.00)	
Yes	72 (65.45)	44 (80.00)	
ACA			0.352
No	101 (91.82)	48 (87.27)	
Yes	9 (8.18)	7 (12.72)	
ACOM			0.731
No	71 (64.55)	34 (61.82)	
Yes	39 (35.45)	21 (38.18)	
MCA			0.050
No	74 (67.27)	46 (83.64)	
Yes	36 (32.73)	9 (16.36)	
PCOM			0.360
No	87 (79.09)	40 (72.73)	
Yes	23 (20.91)	15 (27.27)	
ICA			0.378
No	107 (97.27)	52 (94.55)	
Yes	3 (2.73)	3 (5.45)	
Complication			0.003
No	16 (14.55)	0 (0)	
Yes	94 (85.45)	55 (100)	

CT = computerized tomography; SAH = subarachnoid hemorrhage; ACA = anterior cerebral artery; ACOM = anterior communicating artery; MCA = middle cerebral artery; PCOM = posterior communicating artery; ICA = internal carotid artery; VP = ventriculoperitoneal

Table 2. (cont.)

Risk factor	Alive (n = 110) n (%)	Dead (n = 55) n (%)	p-value
Pneumonia			0.069
No	74 (67.27)	29 (52.73)	
Yes	36 (32.73)	26 (47.27)	
Rebleeding			0.377
No	104 (94.55)	50 (90.91)	
Yes	6 (5.45)	5 (9.09)	
Vasospasm			0.785
No	105 (95.45)	53 (96.36)	
Yes	5 (4.55)	2 (3.64)	
Convulsion			0.448
No	84 (76.36)	39 (70.91)	
Yes	26 (23.64)	16 (29.09)	
Infarction			0.050
No	102 (92.73)	45 (81.82)	
Yes	8 (7.27)	10 (18.18)	
Meningitis			1.000
No	106 (96.36)	53 (96.36)	
Yes	4 (3.64)	2 (3.64)	
Intraoperative ruptured			0.001
No	83 (75.45)	25 (45.45)	
Yes	27 (24.55)	30 (54.55)	
VP shunt			0.050
No	94 (85.45)	54 (98.18)	
Yes	16 (14.55)	1 (1.82)	
Ventriculostomy			0.050
No	81 (73.64)	31 (56.36)	
Yes	29 (26.36)	24 (43.64)	
Tracheostomy			0.874
No	95 (86.36)	47 (85.45)	
Yes	15 (13.64)	8 (14.55)	
Time to surgery			0.028
≤3 day	48 (43.64)	34 (61.82)	
>3 day	62 (56.36)	21 (38.18)	

CT = computerized tomography; SAH = subarachnoid hemorrhage; ACA = anterior cerebral artery; ACOM = anterior communicating artery; MCA = middle cerebral artery; PCOM = posterior communicating artery; ICA = internal carotid artery; VP = ventriculoperitoneal

predicting mortality and functional outcome. The following independent factors were significantly associated with outcome: family history of stroke, GCS, MS, doll's eye reflex, pupillary examination, intraventricular bleeding, MCA aneurysm, hydrocephalus, complication during treatment, postoperative brain swelling, postoperative brain infarction, IAR, VP shunt, ventriculostomy, time to operation, hospital stay, and intraoperative blood loss were statistical significant association with outcome. Whereas the following factors were not associated with outcome: age, sex, hypertension, diabetes mellitus, hyperlipidemia, heart disease, previous stroke, hematologic disease,

Table 3. Treatment and progression on clinical outcome of SAH patients

Risk factor	Alive (n = 110) mean ± SD	Dead (n = 55) mean ± SD	p-value
Hospital stay (day)	16.01±10.33	8.32±9.25	0.05
Operating time (minute)	211.35±68.37	205.00±83.24	
Blood loss (ml)	624.54±481.00	1,088.18±1,022.20	

SAH = subarachnoid hemorrhage

Table 4. Univariate logistic regression analysis on clinical outcome of SAH patients

Risk factor	Odds ratio	95% CI	p-value
Hereditary	3.07	1.02 to 9.21	0.050
Intraventricular bleeding	2.41	1.39 to 4.36	0.003
Hydrocephalus	1.68	0.98 to 2.88	0.050
Brain swelling	2.00	1.24 to 3.22	0.004
Infraction	1.81	1.04 to 3.15	0.030
Intraoperative ruptured	2.27	1.48 to 3.46	0.001
MCA aneurysm	0.52	0.29 to 0.92	0.026
VP shunt	0.16	0.03 to 0.66	0.011
Ventriculostomy	1.63	1.06 to 2.52	0.025
Doll's eye	0.38	0.23 to 0.61	0.001
Pupil	0.38	0.24 to 0.62	0.001
Time to surgery >3 day	0.61	0.39 to 0.94	0.027

SAH = subarachnoid hemorrhage; VP = ventriculoperitoneal

Table 5. Multivariate logistic regression analysis on clinical outcome of SAH patients

Risk factor	Adjusted odds ratio	95% CI	p-value
Brain swelling	3.82	1.35 to 10.79	0.011
Intraoperative ruptured	2.62	1.17 to 5.87	0.019
MCA aneurysm	0.24	0.08 to 0.70	0.009
Ventriculostomy	3.08	1.34 to 7.08	0.008
VP shunt	0.08	0.01 to 0.74	0.026
Doll's eye reflex	0.14	0.03 to 0.47	0.002

SAH = subarachnoid hemorrhage; MCA = middle cerebral artery; VP = ventriculoperitoneal

history of previous medication, smoking, alcoholic consumption, obesity, size of aneurysm, acute subdural hematoma, location of aneurysm (ACA, ACOM, PCOM, ICA), pneumonia, rebleeding postoperative, vasospasm, convulsion, meningitis, tracheostomy, and operating time.

Lanzio et al study confirmed that age is an important predictor of poor outcome after SAH⁽⁸⁾. Several studies have found that the risk of poor outcome after surgery for intracranial aneurysms increases with age and that the risk of poor outcome is significant increased in patients older than 60 years^(6,9).

However, there was no statistical significant association between age and outcome ($p = 0.055$). Though the mean age of the alive groups was less than in dead groups. Hyperglycemia is a frequent finding in critically ill patients and associated with infarct expansion, worse functional outcome, longer hospital stays, high medical costs, increased risk of death, and poor clinical outcome⁽¹⁰⁻¹²⁾. The hyperglycemia was probably not directly harmful to the brain but reflecting stress relating to stroke severity. In the present study, there was no statistical significant association between diabetes mellitus and outcome ($p = 0.159$). In the present study there was statistical significant association between family history of stroke and outcome [OR 3.07 (1.02 to 9.21), $p = 0.05$]. Smoking increases the risk of developing aneurysmal SAH. The relative risk of spontaneous aneurysmal SAH for smokers was twice that of non-smokers. There was no statistical significant association between smoking and outcome ($p = 0.227$), which was the same as reported by other study⁽¹³⁾. Excessive alcohol intake are the important risk factors for aneurysmal SAH⁽⁷⁾ but in the present study there was not statistical significant association between alcohol intake and outcome ($p = 0.424$). An elevated body mass index (BMI) does not seem to play a role as an independent risk factor for aneurysm rupture. Especially in younger patients, low BMI seems to be more positively correlated with the risk of SAH. However, an elevated BMI seems to be correlated with death or a poor neurological outcome. In a recent study by Juvela et al, overweight SAH patients had an increased risk of cerebral infarction. It was therefore suggested that patients with an increased BMI might recover less well from aneurysm surgery⁽¹²⁾. In the present study, there was no statistical significant association between obesity and outcome ($p = 0.077$), which was similar to other study⁽⁷⁾.

The GCS is the most universally accepted system for grading level of consciousness. The GCS grading system was most strongly associated with all outcome measures and the strongest predictor of morbidity and mortality. There was statistical significant association between GCS and outcome ($p = 0.001$), which was the same as reported by other study^(6,14,15). Doll's eye reflex positive means brain stem function was normal, if the patients have increased intracranial pressure (ICP) which are sign of brain herniation, doll's eye reflect will absent. The present study found that absence doll's eye reflex is the significant predictor of surgical outcome [OR 0.14 (0.03 to 0.47), $p = 0.002$]. We also found that pupillary

non-reactive to light is the significant predictor of surgical outcome [OR 0.38 (0.24 to 0.62), $p = 0.001$].

The previous investigation have demonstrated increasing aneurysm size to be predictive of poor outcome^(6,16). This is contrast to some studies that have been confirmed this relationship⁽¹⁷⁾. In the present study, increasing aneurysm size was not associated with poor outcome ($p = 0.955$). With regard to radiological variables based on CT imaging, a significant association between MS and functional outcome and survival time was observed. The MS was found to be an important outcome predictor. This study found that MS is the significant predictor of functional outcome ($p = 0.001$) similar to other studies⁽¹⁸⁾. Brain edema most often manifests as MS. Postoperative brain swelling is likely to be a very powerful predictor of outcome in the present study [OR 3.82 (1.35 to 10.69), $p = 0.011$], same as other studies⁽⁶⁾. Intraventricular bleeding seems to be a very powerful predictor of outcome, the intraventricular bleeding is the strong risk factor for outcome [OR 2.41 (1.39 to 4.36), $p = 0.003$].

Dense focal hematoma is frequently observed in the Sylvain fissure and temporal lobe after rupture of MCA aneurysm. Sylvain hematoma is associated with mortality rate 21% to 85%, even after successful aneurysm clipping and simultaneous hematoma evacuation. Emergency surgery is particularly indicated in case of Sylvain hematoma to prevent irreversible brain damage and serious cerebral edema associated with progression of hematoma⁽¹⁹⁾. In the present study, there was statistical significant association between MCA aneurysms and outcome [OR 0.24 (0.08 to 0.70), $p = 0.009$], which was the same as reported by other study⁽²⁰⁾.

The major causes of death following SAH were initial bleeding, recurrent hemorrhage, and vasospasm⁽²¹⁻²³⁾. Aneurysmal rebleeding has long been recognized as a devastating serious and complication of SAH associated with high morbidity and mortality⁽²⁴⁾. Unfortunately, the incidence of aneurysmal rebleeding is considerably high within hours after the initial SAH^(25,26). Several investigators have reported that rebleeding occurred more often in patients with advanced age, large aneurysm, poor clinical condition or increased systolic arterial blood pressure on admission, loss of consciousness, and procedure of ventricular drainage⁽²⁷⁾; but in the present study there was no statistical significant association between aneurysmal rebleeding and outcome ($p = 0.377$). Cerebral vasospasm after aneurysmal SAH is a frequent but unpredictable complication associated

with poor outcome. The most powerful prognostic factors for vasospasm are the amount of blood on the CT scan obtained at hospital admission⁽²⁸⁾, poor admission clinical grade⁽²⁹⁾, and loss of consciousness at presentation⁽³⁰⁾. There was statistical significant association between cerebral vasospasm and outcome in previous study⁽³¹⁾, but in the present study there was no statistical significant association between cerebral vasospasm and outcome ($p = 0.785$). DINDs from cerebral vasospasm have become the major cause of morbidity and mortality in patients with SAH. An ischemic lesion on postoperative CT scan is a strong, independent risk factor for a poor prognosis after SAH. Risk factors for postoperative cerebral infarction were the amount of subarachnoid blood, duration of temporary and permanent artery clipping, and admission hyperglycemia. In the present study, there was statistical significant association between postoperative cerebral infarction and outcome ($p = 0.05$), which was the same as reported by other studies^(22,32). Seizures are a well-recognized complication after aneurysmal SAH and have been correlated with higher aneurysm grade, lower GCS Score at presentation, extent of subarachnoid blood on CT scan and rebleeding⁽³³⁾. Early studies reported seizures in over 10% of survivors of SAH; there were more likely in younger patients, those with MCA aneurysm, and those with coexisting intracerebral hemorrhage. The administration of prophylactic antiepileptic drugs (AEDs) has in the past been a standard protocol for patients undergoing many neurological procedures. More recently routine use of AEDs for SAH patients has come under question. Low seizures rates have been reported in patients with SAH. The use of AEDs as a primary prophylactic in SAH patients remains controversial. In the present study, seizure was not associated with outcome ($p = 0.448$). IAR is a severe complication that occurs during surgery for an aneurysm in 7% to 40% of cases^(34,35). Its occurrence has been associated with increased neurological deficits, worsened outcome, and increased mortality⁽³⁴⁾. In the majority of cases, IAR is the result of direct surgical manipulation, whether by retraction, dissection, clipping, or surgical experience^(34,36). In the present study, IAR was the strong risk factor for outcome [OR 2.59 (1.16 to 5.80), $p = 0.02$], which was the same as reported by others study⁽³⁷⁾.

Hydrocephalus is a known common sequel following treatment of aneurysmal SAH. It has been reported to range from 6 to 67%⁽³⁸⁾. The most important factors to predict the development of hydrocephalus

requiring permanent cerebrospinal fluid (CSF) diversion are intraventricular hemorrhage, SAH, increased age, low GCS, vasospasm, and aneurysm site^(38,39). Several mechanisms have been proposed to explain the development of hydrocephalus among patients after SAH, some theories suggest a role for alterations in CSF dynamics⁽⁴⁰⁾. Hydrocephalus may occur via obstructive mechanisms when blood products or adhesions block CSF circulation within the ventricular system⁽⁴¹⁾ or may result from absorption problems attribute to impair CSF absorption at the arachnoid granulations⁽⁴²⁾. The patients who develop hydrocephalus after SAH have a worse prognosis than those who do not. In the present study. There was statistical significant association between hydrocephalus and outcome [OR 1.68 (0.98 to 2.88), $p = 0.05$].

The treatment of choice is external ventricular drainage (EVD), which results in prompt and often dramatic improvement of consciousness following immediate drainage of CSF causing from acute hydrocephalus. EVD is the standard of care for the management of acute hydrocephalus related to aneurysmal SAH. Ventriculostomy accomplishes three objectives: 1) patients with poor clinical grades unfavorable to major surgery, 2) intraventricular hemorrhage, 3) the need for a permanent shunt cannot be accessed. Ventriculostomy is a minor procedure compared to permanent shunt⁽⁴³⁾. In the present study, there was statistical significant association between ventriculostomy and outcome [OR 3.08 (1.34 to 7.08), $p = 0.008$], which was the same as reported by other study⁽⁴³⁾.

In the patients who had consistently elevated ICP and had the clinical picture of hydrocephalus from CT scan were underwent placement of a shunt system. Factors that significantly related to the need of VP shunt: increasing age, the presence of hydrocephalus, preoperative CSF diversion, low admission Hunt and Hess grades, amount of cisternal bleeding or ventricular bleeding, and massive bleeding. In the present study, there was statistical significant association between VP shunt and outcome [OR 0.08 (0.01 to 0.74), $p = 0.026$].

The timing of aneurysm surgery has been the subject of a major neurosurgical controversy. Early surgery defined as 1 to 3 days post SAH. There was no uniform agreement on the optimum time of surgical intervention with ruptured intracranial aneurysms. Some surgeons have chosen to operate early to prevent a fatal rebleed⁽⁴⁴⁾. The majority, discouraged by the mortality and morbidity rates ascribed to the early surgical intervention, have elected to delay surgery for

a week or longer after bleed. The late surgical groups' reason was to minimize the risk of postoperative vasospasm, presumably triggered by early surgery, and to allow resolution of the friable edematous brain that would be encountered during early surgery. Yet, Samon et al⁽⁴⁵⁾ observed no difference in the incidence of intraoperative complications or postoperative morbidity when compared both early or late operations, whereas neurologically damaged patients are likely to improve in delayed groups. However, delayed operation is also associated with significant morbidity and mortality from rebleeding and vasospasm occurred during the waiting period.

Conclusion

Outcome of surgical treatment of SAH were related to postoperative brain edema, intraoperative rupture, MCA aneurysm, ventriculostomy, VP shunt, and doll's eye reflex.

What is already known on this topic?

Aneurysmal SAH is associated with a high risk of morbidity and mortality. Clipping ruptured aneurysmal SAH was one of the most difficult operation in neurosurgical surgery.

Most important factors leading to unfavorable outcome were: cerebral infarction, worse clinical grade on admission, advanced age, symptomatic vasospasm, large aneurysms, intraventricular, or intracerebral hemorrhage.

The following independent factors were significantly associated with outcome: GCS, intraventricular bleeding, MCA aneurysm, postoperative brain infarction, IAR, ventriculostomy, early surgery, and intraoperative blood loss, which were the same as reported by the previous study.

What this study adds?

This study found the following independent factors were significantly associated with outcome: family history of stroke, MS, doll's eye reflex, pupillary examination, hydrocephalus, postoperative brain swelling, VP shunt, and hospital stay.

Known the important risk factors associated with the outcome will help neurosurgeon's plan of treatment, care, and predict outcome of the patients.

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Potential conflicts of interest

None.

Reference

1. Sudlow CL, Warlow CP. Comparable studies of the incidence of stroke and its pathological types: results from an international collaboration. *International Stroke Incidence Collaboration. Stroke* 1997; 28: 491-9.
2. Linn FH, Rinkel GJ, Algra A, van Gijn J. Incidence of subarachnoid hemorrhage: role of region, year, and rate of computed tomography: a meta-analysis. *Stroke* 1996; 27: 625-9.
3. Kassell NF, Drake CG. Timing of aneurysm surgery. *Neurosurgery* 1982; 10: 514-9.
4. Tomer JC. Epidemiology of subarachnoid hemorrhage. *Semin Neurol* 1984; 4: 354-69.
5. van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. *Brain* 2001; 124: 249-78.
6. Rosengart AJ, Schultheiss KE, Tolentino J, Macdonald RL. Prognostic factors for outcome in patients with aneurysmal subarachnoid hemorrhage. *Stroke* 2007; 38: 2315-21.
7. Kagerbauer SM, Kemptner DM, Schepp CP, Bele S, Rothorl RD, Brawanski AT, et al. Elevated pre-morbid body mass index is not associated with poor neurological outcome in the subacute state after aneurysmal subarachnoid hemorrhage. *Cent Eur Neurosurg* 2010; 71: 163-6.
8. Lanzino G, Kassell NF, Germanson TP, Kongable GL, Truskowski LL, Torner JC, et al. Age and outcome after aneurysmal subarachnoid hemorrhage: why do older patients fare worse? *J Neurosurg* 1996; 85: 410-8.
9. Chiang VL, Claus EB, Awad IA. Toward more rational prediction of outcome in patients with high-grade subarachnoid hemorrhage. *Neurosurgery* 2000; 46: 28-35.
10. Kruyt ND, Biessels GJ, de Haan RJ, Vermeulen M, Rinkel GJ, Coert B, et al. Hyperglycemia and clinical outcome in aneurysmal subarachnoid hemorrhage: a meta-analysis. *Stroke* 2009; 40: e424-30.
11. Frontera JA, Fernandez A, Claassen J, Schmidt M, Schumacher HC, Wartenberg K, et al. Hyperglycemia after SAH: predictors, associated complications, and impact on outcome. *Stroke* 2006; 37: 199-203.
12. Juvela S, Siironen J, Kuhmonen J. Hyperglycemia, excess weight, and history of hypertension as risk

- factors for poor outcome and cerebral infarction after aneurysmal subarachnoid hemorrhage. *J Neurosurg* 2005; 102: 998-1003.
13. Morris KM, Shaw MD, Foy PM. Smoking and subarachnoid haemorrhage: a case control study. *Br J Neurosurg* 1992; 6: 429-32.
 14. Gotoh O, Tamura A, Yasui N, Suzuki A, Hadeishi H, Sano K. Glasgow Coma Scale in the prediction of outcome after early aneurysm surgery. *Neurosurgery* 1996; 39: 19-24.
 15. Starke RM, Komotar RJ, Otten ML, Schmidt JM, Fernandez LD, Rincon F, et al. Predicting long-term outcome in poor grade aneurysmal subarachnoid haemorrhage patients utilising the Glasgow Coma Scale. *J Clin Neurosci* 2009; 16: 26-31.
 16. Ogilvy CS, Carter BS. A proposed comprehensive grading system to predict outcome for surgical management of intracranial aneurysms. *Neurosurgery* 1998; 42: 959-68.
 17. Chiang VL, Claus EB, Awad IA. Toward more rational prediction of outcome in patients with high-grade subarachnoid hemorrhage. *Neurosurgery* 2000; 46: 28-35.
 18. Kazumata K, Kamiyama H, Yokoyama Y, Asaoka K, Terasaka S, Itamoto K, et al. Poor-grade ruptured middle cerebral artery aneurysm with intracerebral hematoma: bleeding characteristics and management. *Neurol Med Chir (Tokyo)* 2010; 50: 884-92.
 19. Başkaya MK, Menendez JA, Yüceer N, Polin RS, Nanda A. Results of surgical treatment of intrasylvian hematomas due to ruptured intracranial aneurysms. *Clin Neurol Neurosurg* 2001; 103: 23-8.
 20. Ha SK, Lim DJ, Kang SH, Kim SH, Park JY, Chung YG. Analysis of multiple factors affecting surgical outcomes of proximal middle cerebral artery aneurysms. *Clin Neurol Neurosurg* 2011; 113: 362-7.
 21. Proust F, Hannequin D, Langlois O, Freger P, Creissard P. Causes of morbidity and mortality after ruptured aneurysm surgery in a series of 230 patients. The importance of control angiography. *Stroke* 1995; 26: 1553-7.
 22. Schütz H, Krack P, Buchinger B, Bödeker RH, Laun A, Dorndorf W, et al. Outcome of patients with aneurysmal and presumed aneurysmal bleeding. A hospital study based on 100 consecutive cases in a neurological clinic. *Neurosurg Rev* 1993; 16: 15-25.
 23. Sundt TM Jr, Kobayashi S, Fode NC, Whisnant JP. Results and complications of surgical management of 809 intracranial aneurysms in 722 cases. Related and unrelated to grade of patient, type of aneurysm, and timing of surgery. *J Neurosurg* 1982; 56: 753-65.
 24. Bederson JB, Connolly ES Jr, Batjer HH, Dacey RG, Dion JE, Diringer MN, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke* 2009; 40: 994-1025.
 25. Ohkuma H, Tsurutani H, Suzuki S. Incidence and significance of early aneurysmal rebleeding before neurosurgical or neurological management. *Stroke* 2001; 32: 1176-80.
 26. Fujii Y, Takeuchi S, Sasaki O, Minakawa T, Koike T, Tanaka R. Ultra-early rebleeding in spontaneous subarachnoid hemorrhage. *J Neurosurg* 1996; 84: 35-42.
 27. Brilstra EH, Rinkel GJ, Algra A, van Gijn J. Rebleeding, secondary ischemia, and timing of operation in patients with subarachnoid hemorrhage. *Neurology* 2000; 55: 1656-60.
 28. Brouwers PJ, Dippel DW, Vermeulen M, Lindsay KW, Hasan D, van Gijn J. Amount of blood on computed tomography as an independent predictor after aneurysm rupture. *Stroke* 1993; 24: 809-14.
 29. Rabb CH, Tang G, Chin LS, Giannotta SL. A statistical analysis of factors related to symptomatic cerebral vasospasm. *Acta Neurochir (Wien)* 1994; 127: 27-31.
 30. Hop JW, Rinkel GJ, Algra A, van Gijn J. Initial loss of consciousness and risk of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage. *Stroke* 1999; 30: 2268-71.
 31. Shirao S, Yoneda H, Kunitsugu I, Ishihara H, Koizumi H, Suehiro E, et al. Preoperative prediction of outcome in 283 poor-grade patients with subarachnoid hemorrhage: a project of the Chugoku-Shikoku Division of the Japan Neurosurgical Society. *Cerebrovasc Dis* 2010; 30: 105-13.
 32. Siironen J, Porras M, Varis J, Poussa K, Hernesniemi J, Juvela S. Early ischemic lesion on computed tomography: predictor of poor outcome among survivors of aneurysmal subarachnoid hemorrhage. *J Neurosurg* 2007; 107: 1074-9.
 33. Raper DM, Starke RM, Komotar RJ, Allan R, Connolly ES Jr. Seizures after aneurysmal

- subarachnoid hemorrhage: a systematic review of outcomes. *World Neurosurg* 2013; 79: 682-90.
34. Bertalanffy H, Glisbach JM, Mayfrank L, Rohde V, Slansky I. Intraoperative aneurysm rupture: annoyance or serious complication? Paper presented at proceedings of the 46th annual meeting of the Congress of Neurological Surgeons. Montreal, Quebec, Canada; September 28-October 3, 1996.
 35. Le Roux PD, Elliot JP, Newell DW, Grady MS, Winn HR. The incidence of surgical complications is similar in good and poor grade patients undergoing repair of ruptured anterior circulation aneurysms: a retrospective review of 355 patients. *Neurosurgery* 1996; 38: 887-93.
 36. Batjer H, Samson DS. Management of intraoperative aneurysm rupture. *Clin Neurosurg* 1990; 36: 275-88.
 37. Lawton MT, Du R. Effect of the neurosurgeon's surgical experience on outcomes from intraoperative aneurysmal rupture. *Neurosurgery* 2005; 57: 9-15.
 38. Graff-Radford NR, Torner J, Adams HP Jr, Kassell NF. Factors associated with hydrocephalus after subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. *Arch Neurol* 1989; 46: 744-52.
 39. Dorai Z, Hynan LS, Kopitnik TA, Samson D. Factors related to hydrocephalus after aneurysmal subarachnoid hemorrhage. *Neurosurgery* 2003; 52: 763-9.
 40. Auer LM, Mokry M. Disturbed cerebrospinal fluid circulation after subarachnoid hemorrhage and acute aneurysm surgery. *Neurosurgery* 1990; 26: 804-8.
 41. Hasan D, Tanghe HL. Distribution of cisternal blood in patients with acute hydrocephalus after subarachnoid hemorrhage. *Ann Neurol* 1992; 31: 374-8.
 42. Brydon HL, Bayston R, Hayward R, Harkness W. The effect of protein and blood cells on the flow-pressure characteristics of shunts. *Neurosurgery* 1996; 38: 498-504.
 43. Kusske JA, Turner PT, Ojemann GA, Harris AB. Ventriculostomy for the treatment of acute hydrocephalus following subarachnoid hemorrhage. *J Neurosurg* 1973; 38: 591-5.
 44. Hori S, Suzuki J. Early and late results of intracranial direct surgery of anterior communicating artery aneurysms. *J Neurosurg* 1979; 50: 433-40.
 45. Samson DS, Hodosh RM, Reid WR, Beyer CW, Clark WK. Risk of intracranial aneurysm surgery in the good grade patient: early versus late operation. *Neurosurgery* 1979; 5: 422-6.

เลือดออกในสมองใต้เยื่อหุ้มสมองจากการแตกของหลอดเลือดโป่งพองตัวในโรงพยาบาลสวรรค์ประชารักษ์
ณรงค์พงศ์ ใ้วพฤกมณี

วัตถุประสงค์: เพื่อศึกษาความสัมพันธ์ของปัจจัยที่มีผลต่อการรักษาของผู้ป่วยหลอดเลือดสมองโป่งพองตัว

วัสดุและวิธีการ: เก็บข้อมูลย้อนกลับของผู้ป่วยที่ได้รับการผ่าตัด ณ โรงพยาบาลสวรรค์ประชารักษ์ ระหว่างเดือนมกราคม พ.ศ. 2550 ถึง มีนาคม พ.ศ. 2559 ข้อมูลเก็บวิเคราะห์ ได้แก่ อายุ เพศ ความดันโลหิตสูง เบาหวาน ไขมันในเลือดสูง โรคหัวใจ ประวัติโรคหลอดเลือดสมอง ประวัติครอบครัว โรคเลือด ประวัติการใช้ยา สูบบุหรี่ ดื่มสุรา อ้วน ระดับความรู้สึกตัว (GCS) การตรวจ doll's eye reflex การตรวจม่านตา ขนาดของหลอดเลือดสมองโป่งพองตัว ระยะการเคลื่อนที่ของสมองผ่านแนวกลาง เลือดคั่งในช่องน้ำสมอง การมีเลือดออกในสมองชนิด subdural hematoma ภาวะช่องน้ำสมองโต ภาวะลมชัก ปอดอักเสบ การเกิดเลือดออกซ้ำ หลอดเลือดสมองหดรัดตัว ภาวะสมองบวมหลังผ่าตัด ภาวะสมองขาดเลือดตายหลังผ่าตัด การแตกของหลอดเลือดสมองโป่งพองตัวขณะผ่าตัด การติดเชื้อเยื่อหุ้มสมอง ตำแหน่งของหลอดเลือดสมองโป่งพองตัว การผ่าตัด VP shunt การผ่าตัด ventriculostomy การเจาะคอ ระยะเวลาก่อนการผ่าตัด ระยะเวลาการนอนโรงพยาบาล ระยะเวลาการผ่าตัด และการเสียชีวิตระหว่างผ่าตัด

ผลการศึกษา: ผู้ป่วยจำนวน 165 ราย ที่มีหลอดเลือดสมองโป่งพองตัวและได้รับการผ่าตัดรักษา จากการศึกษาพบว่าปัจจัยที่มีความสัมพันธ์กับผลการรักษา ประกอบด้วยภาวะสมองบวมหลังผ่าตัด [OR 3.79 (1.35-10.69), $p = 0.01$] การแตกของหลอดเลือดสมองโป่งพองตัวขณะผ่าตัด [OR 2.59 (1.16-5.80), $p = 0.02$] MCA aneurysm [OR 0.23 (0.08-0.69), $p = 0.05$] การผ่าตัด ventriculostomy [OR 3.04 (1.32-6.99), $p = 0.009$] การผ่าตัด VP shunt [OR 0.08 (0.01-0.74), $p = 0.026$] การตรวจ doll's eye reflex ให้ผลลบ [OR 0.14 (0.03-0.47), $p = 0.002$]

สรุป: การรักษาด้วยวิธีการผ่าตัด จากการศึกษาพบว่าผลการรักษาที่มีความสัมพันธ์กับภาวะสมองบวมหลังผ่าตัด การแตกของหลอดเลือดสมองโป่งพองตัวขณะผ่าตัด หลอดเลือดสมองโป่งพองตัวตำแหน่ง MCA aneurysm การผ่าตัด ventriculostomy การผ่าตัด VP shunt และการตรวจ doll's eye reflex ให้ผลลบ
