www.jkns.or.kr

J Korean Neurosurg Soc 43 : 90-96, 2008

10.3340/jkns.2008.43.2.90

Clinical Article

Jong-Yun Chong, M.D.[1]
Dong-Won Kim, M.D.[2]
Cheol-Su Jwa, M.D.[3]
Hyeong-Joong Yi, M.D.[1]
Yong Ko, M.D.[1]
Kwang-Myung Kim, M.D.[1]

Department of Neurosurgery[1] Hanyang University Medical Center, Seoul, Korea

Department of Pain and Anesthesiology,[2] Hanyang University Medical Center Seoul, Korea

Department of Neurosurgery[3] National Medical Center Seoul, Korea

Impact of Cardio-Pulmonary and Intraoperative Factors on Occurrence of Cerebral Infarction After Early Surgical Repair of the Ruptured Cerebral Aneurysms

Objective : Delayed ischemic deficit or cerebral infarction is the leading cause of morbidity and mortality after aneurysmal subarachnoid hemorrhage (SAH). The purpose of this study is to reassess the prognostic impact of intraoperative elements, including factors related to surgery and anesthesia, on the development of cerebral infarction in patients with ruptured cerebral aneurysms.

Methods : Variables related to surgery and anesthesia as well as predetermined factors were all evaluated via a retrospective study on 398 consecutive patients who underwent early microsurgery for ruptured cerebral aneurysms in the last 7 years. Patients were dichotomized as following; good clinical grade (Hunt-Hess grade I to III) and poor clinical grade (IV and V). The end-point events were cerebral infarctions and the clinical outcomes were measured at postoperative 6 months.

Results : The occurrence of cerebral infarction was eminent when there was an intraoperative rupture, prolonged temporary clipping and retraction time, intraoperative hypotension, or decreased O_2 saturation, but there was no statistical significance between the two different clinical groups. Besides the Fisher Grade, multiple logistic regression analyses showed that temporary clipping time, hypotension, and low O_2 saturation had odds ratios of 1.574, 3.016, and 1.528, respectively. Cerebral infarction and outcome had a meaningful correlation (γ=0.147, p=0.038).

Conclusion : This study results indicate that early surgery for poor grade SAH patients carries a significant risk of ongoing ischemic complication due to the brain's vulnerability or accompanying cardio-pulmonary dysfunction. Thus, these patients should be approached very cautiously to overcome any anticipated intraoperative threat by concerted efforts with neuro-anesthesiologist in point to point manner.

KEY WORDS : Cerebral aneurysm · Cerebral infarction · Hypotension · Oxygen saturation · Subarachnoid hemorrhage · Temporary clipping.

INTRODUCTION

In patients with aneurysmal subarachnoid hemorrhage (SAH), the preoperative neurological grade and amount of the subarachnoid blood clot on computed tomogram (CT) are well-known prognostic factors[1]. However, the surgical outcome can vary even when the same surgeon operates upon similar patients. It is still unclear whether these differences in surgical outcome are due to predetermined factors, to intraoperative alterations or to postoperative management. Several reports have shown that many individual factors during surgery can increase the mortality and morbidity of such patients[14,16]. Of the known complications of SAH, vasospasm or cerebral infarction account for most cases with poor outcomes. Patients who sustain cerebral infarction after aneurysmal SAH are less likely to make a good recovery and more likely to die than those without infarction, but relatively little information is available on the factors predicting the occurrence of infarction[4,12].

The most crucial goal of early aneurysm surgery is to eliminate the chance of rebleeding and to manage postoperative vasospasm extensively as soon as possible. However, severe brain swelling, intraoperative rupture of the aneurysm, and cardio-pulmonary complications frequently hamper the safe obliteration of the ruptured aneurysm during this vulnerable period. Even in the most optimal setting (neurovascular surgeon, anterior circulation aneurysm, Hunt and Hess grade I to III, narrow neck, small lesion, and surgery performed during regular working

• Received : December 11, 2007
• Accepted : February 11, 2008
• Address for reprints :
Hyeong-Joong Yi, M.D.
Department of Neurosurgery
Hanyang University Medical Center
Haengdang-dong, Seongdong-gu
Seoul 133-792, Korea
Tel : +82-2-2290-8499
Fax : +82-2-2281-0954
E-mail : hjyi8499@hanyang.ac.kr

hours), intraoperative problems still occur and contribute to the poor outcome in 6% of patients[6].

The purpose of this study is to identify the individual prognostic factors that may affect the development of post-operative cerebral infarction with special emphases on surgery-related and anesthesia-related factors. This retrospective study was conducted during the "pre-embolization" era at our institute, during which microsurgical clipping was the only surgical procedure performed because of unavailability of the endovascular embolization.

MATERIALS AND METHODS

During the period between July 1999 and June 2005, we performed 428 microsurgical operations for aneurysms. Of these, 30 procedures were excluded due to surgery for unruptured cases (n=13), incomplete data (n=11), or surgery performed beyond 72 hours from the ictus (n=6). In total, we enrolled 398 patients who underwent early microsurgical clipping of the ruptured cerebral aneurysms within 3 days from the initial hemorrhage in the present study.

Our management protocol for ruptured aneurysm is as follows. Ruptured aneurysm was obliterated under a normotensive, normothermic general anesthesia, except during the period of temporary clipping by slight blood pressure elevation of 110-120% to the baseline value. Clipping completeness and vascular competence were confirmed with microsurgical mirror, portable transcranial Doppler microprobe (Multidop X4/TCD8; DWL Electronische System GmbH, Sipplingen, Germany), or recently with an invasive perfusion monitoring probe (Qflow-500; Hemedex Inc., Cambridge, MA). Postoperatively, we obtained an immediate CT scan and catheter angiography to identify a securing aneurysm or inadvertent occlusion of the major vessel. Bedside monitoring of blood flow was conducted with a transcranial Doppler sonography (Companion II; Viasys EME, Dublin, OH).

Oro-tracheal intubation was maintained for one night postoperatively, and an anti-spastic regimen and hyperdynamic therapy were instituted. These included systemic nicardipine (1-2 mg/hr), systemic dopamine and/or dobutamin, unless the systolic blood pressure rose above 150 mmHg. Also, systemic fluid resuscitation up to 4 L/day was provided to maintain a pulmonary capillary wedge pressure of 15 mmHg and a hematocrit of 33%.

Medical charts, radiographic findings, operative records including anesthetic records, operative video-tapes, and outpatient charts were reviewed by using HYMC aneurysm registry sheet. Data regarding predetermined factors (age, sex, neurological status at admission, amount of blood on CT, and location, size, multiplicity and cerebral dominance of aneurysms), surgery-related variables (intraoperative rupture, temporary clipping, time for temporary clipping and brain retraction) and anesthesia-related variables (blood pressure, heart rate, oxygen saturation, hematocrit) were all retrieved. We divided patients into 2 groups (good and poor clinical grade) according to Hunt and Hess grades, in order to discriminate and clarify the genuine impact of the initial neurological grade. Throughout this manuscript, the term "good clinical grade" has been used to describe patients with Hunt and Hess grades I to III, whereas "poor clinical grade" describes those with Hunt and Hess grades IV and V.

"Cerebral infarction" is defined as a newly developed low-density lesion as observed by a head CT scan in the immediate postoperative period. This finding should be compatible with patients' neurological condition and angiographic or transcranial Doppler sonography findings of vasospasm or delayed ischemic neurologic deficit (DIND). Moreover, it should last for at least one month to rule out other causes of "low-density lesions" including cerebral edema, transient cortical low-perfusion area either by retraction on the brain parenchyme or superficial veins or resolving intracerebral hemorrhage. The basis for end-point outcome assessment was twofold; the persistence of cerebral infarction for 6 months postoperatively and Glasgow outcome scale (GOS). The GOS ranged from a favorable (IV, V) to unfavorable (I-III) outcome.

"Intraoperative rupture" is defined as a rupture of the aneurysm that occurs before securing of the parent artery or dissecting the aneurysm neck, which results in massive bleeding that is hard to control by small suction and prevents the progression of a normal operative procedure. In other words, intraoperative rupture is a major rupture from a fundus or neck of the thrombus-sealed aneurysm or avulsed aneurysm or atherosclerotic artery that requires two or more large suctions for bleeding control. Minor bleedings from a small tear on the fundus of the aneurysm, or from partially torn surrounding vessels during clip application were sufficiently controlled with one small suction and therefore, were excluded from this study[3]. "Temporary clipping" is defined as mechanical hemodynamic cessation of parent arteries for dissection of the aneurysm and/or control of intraoperative rupture. "Clipping time" is determined as the total amount of time required in temporary clipping the parent artery or arteries. When two or more clipping were attempted, the clipping time is a total sum. "Retraction time" is defined as the total consecutive time of brain retraction, from the placement of self-retained retractors after dural incision to the withdrawal of retractors after aneurysmal neck clipping.

All physiological hemodynamic parameters were continuously monitored through the arterial catheterized lines or noninvasive monitoring devices and controlled by an attending neuro-anesthesiologist. Comparisons were all made with regard to the initial values. "Decreased blood pressure (hypotension)" is defined as at least a 20% reduction of systolic pressure compared with the initial systolic pressure or a 30 mmHg reduction of systolic pressure exceeding 15 minutes, despite pressor agents or fluid therapy. We considered it abnormal if the heart rate dropped below 55/minute or exceeded 105/minute and was sustained for more than 15 minutes. We defined decreased O_2 saturation as less than 90% of SaO_2 or a 5-point decrement relative to the initial value that lasted for more than 10 minutes despite controlled ventilation. A decreased hematocrit was defined as a value less than 30%, lasting more than 60 minutes despite transfusion.

Parameters were expressed as the mean±standard deviations, and statistical analysis was carried out using SPSS software (version 10.0) for personal computers (SPSS Inc., Chicago, IL). Univariate analyses of the correlation between reference events such as GOS and cerebral infarction, and factors regarding demography, surgery, and anesthesia were calculated in 3 groups (whole patient group, good and poor clinical grade group) for independence. Nominal scales and interval scales were compared using the chi-squared test (for frequency) and the independent-Samples t-test (for means). Continuous variables were also compared using the independent-Samples t-test. Multiple logistic regression analyses were performed to calculate the odds ratio (OR) at a 95% confidence interval (CI) for reference end-point event-related variables that had been confirmed by the univariate analyses. A backward stepwise selection method was chosen for analysis to minimize omission of important variables. Finally, the correlation between the occurrence of cerebral infarction and unfavorable outcome was calculated using the partial correlation coefficient adjusted by the Hunt-Hess grade. A probability value of less than 0.05 was considered statistically significant. Any figures or graphs depicting a correlation could not be drawn due to the dichotomized, non-linear relationship of the independent variables.

RESULTS

The demographic data of the patients and characteristic features of the aneurysms are summarized in Table 1. Cerebral infarction was found in 76 patients upon immediate postoperative CT and in 56 patients at 6 months after surgery. The size of the initial infarct decreased or resolved as time passed, and it was more prominent when a compromise of the parent artery or major branch vessels (n=28) was not present. A favorable outcome was noted in 333 patients, while an unfavorable outcome occurred in 65 patients. Surgery-

Table 1. Clinical summary of 398 consecutive SAH patients undergoing early aneurysmal surgery*

Variables	Good grade group (n=289)	Poor grade group (n=109)	Total (n=398)
Age (years)	54.1±12.69	57.4±15.57	55.7±13.28
Sex			
Male	88	31	119
Female	201	78	279
Fisher grade			
I, II	159	8	167
III, IV	130	101	231
Aneurysm location			
ICA (PCoA)	95	24	119
ACA (ACoA)	107	46	153
MCA	75	35	110
VBA	12	4	16
Aneurysm size (mm)	8.0±1.51	8.8±0.27	8.2±0.32
Cerebral dominance (+)	98	38	136
Multiplicity (+)	17	11	28
GOS (6 months)			
Good (IV, V)	261	72	333
Poor (I–III)	31	34	65
Cerebral infarction (+)			
Immediate	43	33	76
6 months	31	25	56

*This group was dichotomized according to the initial neurological grade of Hunt–Hess classification. Some values are expressed as mean±SD. SAH : subarachnoid hemorrhage, ICA : internal carotid artery, PCoA : posterior communicating artery, ACA : anterior cerebral artery, ACoA : anterior communicating artery, MCA : middle cerebral artery, VBA : vertebrobasilar artery, GOS : Glasgow outcome scale

Table 2. Summary of surgery-related and anesthesia-related variables in 398 consecutive SAH patients undergoing early aneurysmal surgery*

Variables	Good grade group (n=289)	Poor grade group (n=109)	Total (n=398)
Intraoperative rupture (+)	29	27	56
Temporary clipping (+)	59	42	101
Clipping time (minutes)	5.1±2.29 (0–18)	9.3±5.61 (0–32)	6.8±5.09 (0–32)
Retraction time (minutes)	55.8±16.03 (25–135)	72.1±18.04 (28–212)	63.6±17.68 (25–212)
Decreased blood pressure† (>20% or 30 mmHg)	19	32	51
Abnormal heart rate† (<55/min or >105/min)	48	35	83
O_2saturation† (SaO_2<90% or >5point drop)	15	24	39
Hematocrit‡ (<30%)	33	16	49
GOS (poor)	31	34	65
Cerebral infarction (+)	31	25	56

*This group was dichotomized according to the initial neurological grade of Hunt–Hess classification. Some values are expressed as mean±SD. SAH : subarachnoid hemorrhage, GOS : Glasgow outcome scale. †Lasting more than 15 minutes during the operation. ‡Lasting more than 60 minutes during the operation

Table 3. The results of univariate analyses with regard to the GOS*

Variables	Good grade group (n=289)	Poor grade group (n=109)	Total (n=398)
Age (years)	0.511	0.229	0.388
−40			
41−50			
51−60			
61−70			
71−			
Sex	0.112	0.775	0.259
Fisher grade	0.005†	0.184	0.005†
Aneurysm location	0.385	0.674	0.149
Aneurysm size	0.048†	0.062	0.041†
Cerebral dominance	0.039†	0.102	0.082
Multiplicity	0.080	0.451	0.410
Intraoperative rupture	0.085	0.028†	0.057
Temporary clipping	0.147	0.059	0.094
Clipping time	0.035†	0.540	0.491
Retraction time	0.084	0.272	0.517
Decreased systolic pressure	0.192	0.018†	0.042†
Abnormal heart rate	0.497	0.721	0.237
Decreased O_2saturation	0.736	0.054	0.167
Decreased hematocrit	0.771	0.088	0.335
GOS (poor)	31	34	65

*This group was dichotomized according to the initial neurological grade of Hunt−Hess classification. GOS : Glasgow outcome scale. †Statistically significant, if p value<0.05. p value was calculated by chi−square test (for frequencies) or t−test (for means)

Table 4. The results of univariate analyses with regard to the cerebral infarction*

Variables	Good grade group (n=289)	Poor grade group (n=109)	Total (n=398)
Age (years)	0.328	0.059	0.199
−40			
41−50			
51−60			
61−70			
71−			
Sex	0.610	0.810	0.461
Fisher grade	0.012†	0.291	0.035†
Aneurysm site	0.061	0.105	0.085
Aneurysm size	0.028†	0.035†	0.029†
Cerebral dominance	0.042†	0.073	0.062
Multiplicity	0.071	0.149	0.096
Intraoperative rupture	0.185	0.013†	0.037†
Temporary clipping	0.069	0.075	0.055
Clipping time	0.081	0.008†	0.024†
Retraction time	0.053	0.002†	0.001†
Decreased systolic pressure	0.019†	0.004†	0.015†
Abnormal heart rate	0.237	0.721	0.493
Decreased O_2saturation	0.062	0.008†	0.028†
Decreased hematocrit	0.271	0.071	0.182
GOS (poor)	31	25	56

*This group was dichotomized according to the initial neurological grade of Hunt−Hess classification. GOS : Glasgow outcome scale. †Statistically significant if p value<0.05. p value was calculated by chi−square test (for frequencies) or t−test (for means)

Table 5. The results of multiple logistic regression analyses with infarction*

Variables	Significance	Exp (B)	95% CI for Exp (B)
Fisher grade (III, IV)	0.005*	2.186	1.724−3.568
Aneurysm size (>8.2±0.32)	0.308	0.851	0.259−1.361
Intraoperative rupture (+)	0.103	1.123	0.714−2.628
Clipping time (>6.8±5.09)	0.036*	1.574	1.084−2.142
Retraction time (>63.9±17.68)	0.295	1.273	0.618−1.954
Hypotension (+)	0.008*	3.016	1.285−7.075
Low SaO_2 (+)	0.041*	1.528	1.125−2.381

*Statistically significant. Exp (B) : Odds ratio, exponential form of square of standardized regression coefficient (B), CI : confidence interval. Some values are expressed as mean±SD

related and anesthetic variables are reviewed in Table 2.

The univariate analysis was carried out to identify independent correlations between each demographic or tested variable and the corresponding outcomes for aneurysmal SAH patients. In analyzing the demographic variables for the whole patient group, we found that Fisher grade (p=0.005) and aneurysm size (p=0.041) affected the patients' outcome (GOS). However, the result was not statistically significant when the analyses were performed for the poor grade group (Table 3). Table 3 also shows the results of univariate analyses for the correlation between surgery-related or anesthesia-related variables and the patients' outcomes (GOS). Of the surgery-related factors, we were able to identify only intraoperative hypotension as a significant predictor of outcome for the whole patient group (p=0.042). Intraoperative rupture and hypotension were prognostic factors in the poor grade group (p=0.028 and 0.018, respectively), while prolonged temporary clipping had a statistically significant association with a poor outcome in the good grade group (p=0.035).

The Fisher grade (p=0.035) and size of the ruptured aneurysm (p=0.029) were related to cerebral infarction in the whole patient group. When compared with the poor grade group, the Fisher grade (p=0.012) and cerebral dominance (p=0.042) were 2 factors responsible for the development of cerebral infarction in the good-grade group, in addition to the size of the aneurysm (p=0.028, Table 4). Table 4 also shows that the possibility of postoperative cerebral infarction increased in the whole patient group when there was an intraoperative rupture (p=0.037), prolonged clipping time (p=0.024), longer retraction time (p=0.001), decreased systolic pressure (p=0.015) or decreased O_2 saturation (p=0.028). In subgroups with a poor clinical grade, the same factors identified in the total patient group were statistically significant. However, in the good-grade group, decreased systolic pressure (p=0.019) was the only determinant for impending infarction.

Multiple logistic regression analysis was performed to calculate the OR with a 95% CI. Because intraoperative factors may be closely related to each other, maximizing

the discrimination among them is mandatory for identifying variables with the most probable correlation. Continuous variables with prognostic significance in the development of infarction by univariate analyses (aneurysm size, clipping time, and retraction time) were set according to their mean values ($>8.2\pm0.32$ mm, $>6.8\pm5.09$ min, and $>63.9\pm17.68$ min, respectively) and entered into multiple logistic regression. Categorical variables having a significant correlation with infarction by univariate analysis (Fisher grade III to IV, presence of intraoperative rupture, decreased systolic pressure and decreased O_2 saturation) were also entered into multiple logistic regression analysis. Overall, a Fisher grade of III to IV (95% CI 1.724-3.568), temporary clipping time $>6.8\pm5.09$ min (95% CI 1.084-2.142), intraoperative hypotension (95% CI 1.285-7.075) and low intraoperative O_2 saturation (95% CI 1.125-2.381) were independent risk factors for the development of postoperative cerebral infarction (Table 5).

The partial correlation coefficient between cerebral infarction and the 6-month postoperative outcome, which was adjusted by the Hunt-Hess grade, showed statistical significance ($\gamma=0.147$, $p=0.038$).

DISCUSSION

This study highlights the inherent risk of ongoing infarction with early aneurysm surgery, and the resultant infarct clearly precludes an unremarkable patient recovery. However, it is not still clear whether the occurrence of infarction is due to a single or multiple factors. In the current study, predetermined factors such as the initial amount of SAH, as well as intraoperative factors, including the time required for temporary clipping, low blood pressure, and low oxygen saturation, independently leads to the development of an unfavorable end-point outcome. Moreover, the anticipated threat of a specific aneurysm features (size, location, geometry) and the presence of an intraoperative rupture are overestimated, irrespective of pertinent clinical status. From the results of this study, we emphasize the importance of managing extracerebral and cardio-pulmonary dysfunction with great caution during general anesthesia.

As stated previously, age, clinical grade, aneurysm location, intraventricular hemorrhage, preoperative rebleeding, temporary clipping, ligation or occlusion of a major artery, postoperative CT hypodensity, postoperative intracerebral hemorrhage, and postoperative myocardial infarction or pulmonary embolism are well-known prognostic factors contributing to unfavorable outcomes, regardless of the end-point[10]. Intraoperative complications or technical mishaps having clinical significance still occur in contemporary surgical series, and, overall, between 5 and 25% of morbidity and mortality after SAH can be attributed to these surgical complications[7,9,12,14,16].

From the surgical viewpoint, intraoperative rupture is the most frequent and devastating incident and its prognostic implication should be weighted by its occurring period; before dissection, during dissection, and during the clip application period[3]. When intraoperative rupture happens before exposure of the sac or rupture at the basilar artery or middle cerebral artery, and hypotension induced during surgery, poor outcome is generally expected[2]. Temporary clipping is performed to control bleeding from an intraoperative rupture or to dissect the aneurysmal sac more safely. The general tolerance limit for temporary clipping is about 15-20 minutes because it is associated with a decrease in brain PO_2 and an increase in brain PCO_2, and this seems to be the critical threshold for the development of postoperative cerebral ischemic events[6,11,18]. In the current study, repeated temporary clipping was carried out if necessary, to permit sufficient interim reperfusion of cerebral blood flow for at least 5-10 minutes to prevent postoperative neurological deficits. Satisfactory clip application to the aneurysmal neck could be done even without temporary clipping, when the intraoperative rupture of the aneurysm occurred during clip reposition, or sufficient exposure of the entire aneurysmal dimension was achieved. In addition to this, temporary clipping to the parent artery or permanent clipping to the neck of the aneurysm could be sufficiently performed only after identification of the parent artery or aneurysmal sac using two suctions. Focal circulatory arrest caused by temporary clipping does not seem to be associated with stroke, but individual susceptibility for survival after circulatory arrest appears to exist and other predisposing factors may be present[8,11,13,18].

A reduction in systemic arterial pressure also decreases the transmural pressure gradient of the aneurysm and thereby the wall stress of the aneurysm. This can facilitate preparation and clipping of the aneurysm as well as helping control of bleeding, but induced systemic hypotension is no longer used routinely in the clipping of aneurysms. Because a reduction in systemic arterial pressure can critically impair overall cerebral perfusion, especially in the presence of hypovolemia, it is associated with an adverse outcome and a higher incidence of severe cerebral vasospasm[5,13]. During a temporary clipping, the blood pressure should be maintained at or even slightly above baseline values to ensure adequate collateral blood flow. Close communication between the neurosurgeon and the anesthesiologist is thus of utmost importance, because with removal of the temporary clip, the unsecured aneurysm is exposed to a much higher shear stress. An elevation of systolic blood pressure up to 150 mmHg in normotensive patient or a 10% increment in patient with hypertension

during temporary clipping, along with the administration of cerebral protective agents such as mannitol (0.5-1 g/kg) and thiopental (15-30 mg/kg) is proved to be effective to overcome hypotensive sequelae[3,8,11]. This was valid unless the total time for temporary clipping exceeded 15 minutes in our institute.

In medical perspective, the proportion of deaths directly attributable to medical complications (23%) is comparable to that of vasospasm (23%) and rebleeding (22%)[17]. Fever, anemia and hyperglycemia were independent prognosticators to have adverse effects in SAH patients[19]. Some studies have stressed the importance of extracerebral organ dysfunction on the development of delayed ischemic deficits more than the intrinsic cerebral pathophysiologic process, either by a broadly simplified acute physiology score (SAPS) or more specifically the PaO_2/FiO_2 ratio[15,17]. The influence of the PaO_2/FiO_2 ratio corresponds well with commonly encountered neurogenic pulmonary edema or pneumonia in patients with SAH. When cardio-pulmonary resuscitative efforts fail, such as with severe ischemic heart disease, arrhythmia, pulmonary edema, or pneumonia, unaided microsurgical clipping might result in an unfavorable outcome. Poor clinical grade, diffuse brain swelling, unstable cardiac performance resulting in hypoperfusion, and low ventilation are major factors hindering both safe microsurgery and postoperative recovery.

Several limitations of this study should be acknowledged. First, because this study was conducted at a single institution mainly by an uncontrolled review of charts and radiographic images, some discrepancies in the data interpretation and unexpected loss or omission of some data might have been possible. To achieve external validity, multicenter investigation is required. Second, too small number of the posterior circulation aneurysms may have created sampling error and outcome assessment. This was largely attributed to referral to the other institutes for endovascular embolization. Third, more deliberate intraoperative monitoring should be employed for hemodynamic and electrophysiologic parameters to detect more delicate abnormalities earlier. Finally, and most importantly, this study did not extend beyond the operative period and the impact of cardio-pulmonary insults on the subsequent development of cerebral infarction during this crucial period was not estimated. But, we have at least tried to provide informations regarding intraoperative perspectives. Our intention was to declare the relationship between intraoperative events or factors and outcome at 6-month period.

CONCLUSION

Early microsurgery for ruptured aneurysm, particularly for poor grade SAH patients, inherently carries a significant risk of ongoing ischemic complication due to vulnerable brain and cardio-pulmonary dysfunction. Cardiovascular and pulmonary dysfunction such as hypotension and low ventilation in the preoperative period can hamper successful postoperative recovery even in unremarkable surgical situations. When encountering such perplexing conditions, careful perioperative preparation including meticulous microsurgical manipulation and hemodynamic correction aided by an anesthesiologist is mandated to avoid poor postoperative recovery.

References

1. Auer LM : Unfavorable outcome following early surgical repair of ruptured cerebral aneurysms. A critical review of 238 patients. **Surg Neurol 35** : 152-158, 1991
2. Batjer H, Samson D : Intraoperative aneurysmal rupture : incidence, outcome, and suggestions for surgical management. **Neurosurgery 18** : 701-707, 1986
3. Chandler JP, Getch CC, Batjer HH : Intraoperative aneurysm rupture and complication avoidance. **Neurosurg Clin N Am 9** : 861-868, 1998
4. Claassen J, Carhuapoma JR, Kreiter KT, Du EY, Connolly ES, Mayer SA : Global cerebral edema after subarachnoid hemorrhage : frequency, predictors, and impact on outcome. **Stroke 33** : 1225-1232, 2002
5. Ferch R, Pasqualin A, Pinna G, Chioffi F, Bricolo A : Temporary arterial occlusion in the repair of ruptured intracranial aneurysms : an analysis of risk factors for stroke. **J Neurosurg 97** : 836-842, 2002
6. Fridriksson S, Saveland H, Jakobsson KE, Edner G, Zygmunt S, Brandt L, et al : Intraoperative complications in aneurysm surgery : a prospective national study. **J Neurosurg 96** : 515-522, 2002
7. Kassell NF, Torner JC, Jane JA, Haley EC Jr, Adams HP : The International Cooperative Study on the Timing of Aneurysm Surgery. Part 2 : Surgical results. **J Neurosurg 73** : 37-47, 1990
8. Lavine SD, Masri LS, Levy ML, Giannotta SL : Temporary occlusion of the middle cerebral artery in intracranial aneurysm surgery : time limitation and advantage of brain protection. **J Neurosurg 87** : 817-824, 1997
9. 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 38** : 887-893, 1996
10. Niskanen MM, Hernesniemi JA, Vapalahti MP, Kari A : One-year outcome in early aneurysm surgery : prediction of outcome. **Acta Neurochir (Wien) 123** : 25-32, 1993
11. Ogilvy CS, Carter BS, Kaplan S, Rich C, Crowell RM : Temporary vessel occlusion for aneurysm surgery : risk factors for stroke in patients protected by induced hypothermia and hypertension and intravenous mannitol administration. **J Neurosurg 84** : 785-791, 1996
12. Ohman J, Servo A, Heiskanen O : Risks factors for cerebral infarction in good-grade patients after aneurysmal subarachnoid hemorrhage and surgery : a prospective study. **J Neurosurg 74** : 14-20, 1991
13. Samson D, Batjer HH, Bowman G, Mootz L, Krippner WJ Jr, Meyer YJ, et al : A clinical study of the parameters and effects of temporary arterial occlusion in the management of intracranial aneurysms. **Neurosurgery 34** : 22-29, 1994
14. Saveland H, Hillman J, Brandt L, Edner G, Jakobsson KE, Algers G : Overall outcome in aneurysmal subarachnoid hemorrhage. A prospective study from neurosurgical units in Sweden during a 1-year period. **J Neurosurg 76** : 729-734, 1992
15. Schuiling WJ, de Weerd AW, Dennesen PJ, Algra A, Rinkel GJ : The simplified acute physiology score to predict outcome in patients with subarachnoid hemorrhage. **Neurosurgery 57** : 230-236, 2005
16. Seiler RW, Reulen HJ, Huber P, Grolimund P, Ebeling U, Steiger HJ : Outcome of aneurysmal subarachnoid hemorrhage in a hospital population : a prospective study including early operation, intravenous nimodipine, and transcranial Doppler ultrasound. **Neurosurgery 23** : 598-604, 1988

17. Solenski NJ, Haley EC Jr, Kassell NF, Kongable G, Germanson T, Truskowski L, et al : Medical complications of aneurysmal subarachnoid hemorrhage : a report of the multicenter, cooperative aneurysm study. **Crit Care Med 23** : 1007-1017, 1995
18. Taylor CL, Selman WR, Kiefer SP, Ratcheson RA : Temporary vessel occlusion during intracranial aneurysm repair. **Neurosurgery 39** : 893-905, 1996
19. Wartenberg KE, Schmidt JM, Claassen J, Temes RE, Frontera JA, Ostapkovich N, et al : Impact of medical complications on outcome after subarachnoid hemorrhage. **Crit Care Med 34** : 617-623, 2006