

OUTCOME OF SURGICAL MANAGEMENT OF 347 INTRACRANIAL ANEURYSMS IN 305 CASES**İhsan SOLAROĞLU, Erkan KAPTANOĞLU, Özerk OKUTAN, Etem BEŞKONAKLI, Yamaç TAŞKIN****Ankara Numune Research and Education Hospital, Department of Neurosurgery, ANKARA****SUMMARY**

Subarachnoid hemorrhage as a result of intracranial aneurysm rupture has high mortality and morbidity rate. Overall management results in intracranial aneurysm management influence by several factors and predicting the outcome in aneurysm surgery is difficult because of variable characteristics of patients. In this study we aimed to investigate the relationship between initial WFNS and Fisher grade and outcome in 305 patients that underwent intracranial aneurysm surgery between 1992-2001. Relationship between initial Fisher grade and vasospasm is also analyzed.

Key words: Aneurysm, prognosis, subarachnoid hemorrhage, treatment.

305 VAKADA 347 İNTRAKRANİAL ANEVİRİZMANIN CERRAHİ SONUÇLARI

İntrakranial anevrizma rüptürüne bağlı subaraknoid kanama yüksek mortalite ve morbidite oranına sahiptir. İntrakranial anevrizmaların tedavi sonuçları birçok faktör tarafından etkilenir ve anevrizma cerrahisinde sonuçları önceden tahmin etmek hastaların değişken özelliklerinden dolayı güçtür. Bu çalışmada 1992-2001 yılları arasında intrakranial anevrizma cerrahisi uygulanmış 305 hastada başvuru WFNS skoru ve Fisher grade'i ile tedavi sonuçları arasındaki ilişkinin araştırılması amaçlanmıştır. Beraberinde başvuru Fisher grade'i ile vazospazm ilişkisi analiz edilmiştir.

Anahtar Sözcükler: Anevrizma, prognoz, subaraknoid kanama, tedavi.

INTRODUCTION

Cerebral aneurysms occur in approximately 1-5% of the general population (1, 2). Despite the recent developments in microsurgical techniques in aneurysm surgery and the treatment of cerebral vasospasm, the prognosis for patients who suffer a subarachnoid hemorrhage (SAH) remains unsatisfactory (3, 4).

SAH as a result of a intracranial aneurysm rupture results in a complex clinical picture that often associated with many interrelated complications such as cerebral edema, diffuse cerebral ischemia, obstructive hydrocephalus, focal cerebral ischemia or infarction (5). Therefore, outcome following SAH is influenced by many variables.

Several factors that influence overall management results, such as patient age, initial computed tomography (CT) findings, timing of surgical intervention, contemporary medical and surgical management, location of intracranial aneurysm, and preexisting medical illnesses have been studied extensively by many authors (2-12). It is difficult to compare these reports of management results because of differences in patient populations and variations in the timing and methods of aneurysm surgery. Therefore, in this report, we assessed the relationships between

initial CT findings and neurological status and outcome in 305 patients with intracranial aneurysm patients who underwent surgery in our institution. The relationship between Fisher grade and vasospasm were also analyzed.

MATERIAL AND METHODS

Between 1992-2001, 347 intracranial aneurysms in 305 cases were treated surgically at our institution. The patients age, sex, preexisting medical illnesses, presenting symptoms and signs, location of aneurysm were analyzed retrospectively.

SAH was confirmed by computerized tomography (CT) or, in rare instances, by lumbar puncture. World Federation of Neurosurgical Societies (WFNS) score was used to determine the initial neurological status and Fisher grading system (13) was used to assess the initial CT findings. Digital subtraction angiography (DSA) was used to show aneurysm location. Angiographically confirmed vasospasm on DSA was noted.

Patients were cared in the neurosurgery intensive care unit and appearance of symptomatic vasospasm was also noted. Symptomatic vasospasm was defined as deterioration of neurological grade in the presence of focal clinical signs consistent with regional brain ischemia (14). Same preoperative medication including

phenytoin, sedatives, and analgesics were used routinely. Same postoperative management including hypervolemic - hypertensive - hemodilution therapy (insufficient respiration treated with intubation and ventilation to maintain PaO₂ above 100 mmHg and PaCO₂ at 25-30 mmHg) were used for all patients with SAH. Antihypertensive medication was used as required.

We used the following criteria to decide the timing of surgical intervention; as soon as possible if patient have WFNS score on preoperative neurological examination ≤ 3 . In patients that have WFNS score 4 or 5, the operation was delayed until the patient's condition could become stabilized.

Standart right pterional approach was used in majority of cases. All aneurysms were operated on under the operating microscope and by one surgeon or a member of the resident staff with that surgeon acting as the first asistant. Aneurysms were repaired by clipping, wrapping or combination of each other. Multiple aneurysms that approachable through the same operative exposure were routinely repaired during the same operation.

The follow-up time ranged from 6 to 53 months. Neurological outcome was evaluated by Glasgow Outcome Scale (GOS), and GOS I and GOS II were accepted as functional recovery (FR+).

The relation between mortality and morbidity rates and initial WFNS score and Fisher grade were analyzed. The interrelationship between the Fisher grade and vasospasm was also analyzed.

Statistical analysis was performed using Pearson Chi-square test. A p value <0.05 was considered to indicate a statistically significant difference.

RESULTS

There were 162 male (53.1%) and 143 female (46.9%), ranging in age from 14 to 76 years, with a mean of 53.5 years (Table I). The most common presenting symptom was headache (69.8%) and the sign was stiffness of the neck (59.8%). The ratio of hypertension as a preexisting medical illness was 33.4%. Distribution of initial WFNS scores and Fisher grades are shown in Table II and Table III. Unruptured intracranial aneurysms (WFNS=0) were found in eight patients.

Three-hundred-fourty-seven aneurysms were found in 305 patients on DSA. Distribution of location of aneurysms is shown in Table IV. Anterior circulation aneurysms were noted in

Table I. Distribution of patients age.

Age	No of cases	Ratio (%)
0-10	-	-
11-20	3	1.0
21-30	7	2.3
31-40	44	14.4
41-50	87	28.5
51-60	99	32.5
61-70	53	17.4
71 and more	12	3.9
Total	305	100

Table II. Distribution of initial WFNS score.

WFNS	No of cases	Ratio (%)
0	8	2.6
1	92	30.2
2	101	33.1
3	62	20.3
4	42	13.8
5	-	-
Total	305	100

Table III. Distribution of initial Fisher grade.

Fisher Grade	No of cases	Ratio (%)
1	62	20.3
2	104	34.1
3	92	30.2
4	47	15.4
Total	305	100

Table IV. Location of intracranial aneurysms.

Location of aneurysm	No	Ratio (%)
MCA	122	35.2
ACoA	128	36.9
ACA	16	4.6
PICA	2	0.6
ICA		
Cavernous	4	1.2
Ophthalmic	5	1.4
Bifurcation	14	4.0
Basillar tip	3	0.9
PcoA	52	14.9
Sup.Cerebellar	1	0.3
Total	347	100

Table V. Relationship between initial WFNS score and outcome.

WFNS	Functional Recovery				Exitus		Total
	+		-		n	%	
	N	%	n	%			
0	8	100	0	0	0	0	8
1	88	95.7	4	4.3	2	2.2	92
2	94	93.1	7	6.9	2	2.0	101
3	53	85.5	9	14.5	3	4.8	62
4	24	57.1	18	48.9	7	16.7	42
5	0	0	0	0	0	0	0
Total	267	87.5	38	12.5	14	4.6	305

$\chi^2=45.34; df=4 p<0.001^{***}$

Table VI. Relationship between initial Fisher grade and outcome.

Fisher Grade	Functional Recovery				Exitus		Total
	+		-		n	%	
	N	%	n	%			
1	59	95.2	3	4.8	1	1.6	62
2	97	93.3	7	6.7	2	1.9	104
3	85	92.4	7	7.6	2	2.2	92
4	26	55.3	21	44.7	9	19.1	47
Total	267	87.5	38	12.5	14	4.6	305

$\chi^2=53.16; df=3 p<0.001^{***}$

Table VII. Relationship between initial Fisher grade and vasospasm.

Fisher Grade	Vasospasm				Total
	+		-		
	n	%	n	%	
1	9	14.5	53	85.5	62
2	17	16.4	87	83.6	104
3	31	33.7	61	66.3	92
4	5	10.6	42	89.4	47
Total	62	20.3	243	79.7	305

$\chi^2=15.19; df=3 p=0.002^{**}$

98.2% of the patients. The most common aneurysm location was anterior communicating artery with the ratio of 36.9%. Thirty-two of 305 patients had multiple aneurysms. Twenty-two patients had double, ten patients had three aneurysms. Thirty-seven aneurysms were wrapped, four aneurysms were wrapped and clipped, and the remaining aneurysms were occluded by application of a single or more aneurysm clips. McFaden (Codman Co., Randolph MA, USA) and Yaşargil (Aesculap Co., Tutlingen, Germany) clips were used to repair aneurysms. The mean time interval between bleeding and operation was 4.7 days in patients

with SAH in our institution.

The overall mortality rate was 4.6% and the morbidity rate was 7.9%. The overall FR rate was 87.5%.

The relationship between WFNS score and outcome is shown in Table V. There were significant differences among the mortality and morbidity rates when patients were categorized according to WFNS score ($p<0.05$). The rates of mortality and functional recovery for the 42 patients who have WFNS score 4 were 16.7% and 57.1%, respectively. There was a trend towards poorer outcome in the cases that have increased WFNS score.

The relationship between Fisher grade and outcome is shown in Table VI. There were significant differences among the mortality rates and functional recovery rates when patients were categorized according to Fisher grade ($p<0.05$). The rates of mortality and functional recovery for the 42 patients who have a Fisher grade 4 were 19.1% and 55.3%, respectively. Fisher grade 4 was associated with poorer outcome.

The relationship between Fisher grade and vasospasm is shown in Table VII. The ratio of vasospasm in patients with a Fisher grade 3 was 33.7%. There was statistically significant difference between the vasospasm ratio when the cases were analyzed according to Fisher grade ($p<0.05$). Fisher grade 3 was associated with increased rate of vasospasm.

DISCUSSION

The male: female ratio and the mean patient age were similar with reported series in the literature (3, 11, 15, 16). However, distribution of aneurysms according to location was not similar. Although the ratio of posterior circulation aneurysms was lower than expected ratio, most of the patients with posterior circulation aneurysms were referred to embolization.

There is no consensus on the timing of ruptured intracranial aneurysm surgery (17). Rebleeding is the leading cause of morbidity and mortality in addition to the initial bleed and vasospasm, producing unfavorable results in 7.5% patients (3). It has been reported that the incidence of rebleeding in the early-management group was significantly lower when compared with the late-management group (16). Miyaoka et al (16) suggested that early surgery appears to be beneficial in Grade III and IV patients according

to Hunt and Hess classification. Hernesniemi et al (15) suggested that patients in Grade I-III according to Hunt and Hess classification could be operated during the first 72 hours safely with good results. However, early surgery do not prevent delayed ischemic deficits (15). Many authors reported that the mortality rate associated with early surgery is significantly higher than in conventional delayed surgery (18, 19). It is difficult to compare these reported results and formulate a precise protocol for the timing of aneurysm surgery. We delayed the operative intervention in patients with poor grades until the patient's condition could become stabilized. Patients that have initial WFNS score ≤ 3 were operated as soon as possible. The correlation between patients neurological status on admission and outcome was reported by Sundt et al (5). They were used the modified Bottarell classification for grading neurological status of the patient and reported significantly high mortality and morbidity rates in patients with poor neurological grades. There were significant differences among the mortality and morbidity rates when patients were categorized according to WFNS score ($p < 0.05$) in our study. Our results are in line with those of Sundt et al (5), and provide further evidence that initial poor neurological status is one of the main predictors of outcome in cases of aneurysmal SAH.

SAH-induced vasospasm is a major cause of mortality and neurological morbidity and remains as a significant clinical problem in patients with ruptured intracranial aneurysm. Acute cerebral vasospasm is characterized by increased intracranial pressure (20, 21), decreased cerebral blood flow (21, 22) and decreased cerebral perfusion pressure (21). It is now widely accepted that blood products, especially oxyhemoglobin, contribute to cerebral vasospasm (23). The relationship between the severity of SAH and vasospasm was reported by many authors (13, 24). The ratio of vasospasm in patients with a Fisher grade 3 was 33.7% in our study. Fisher grade 3 was associated with increased rate of vasospasm. It may be related with "the more blood the more spasm" idea. Despite of the increased ratio of vasospasm in patients with a Fisher grade 3, the mortality rate was significantly high in Fisher grade 4 group. There are two possible explanation for the relationship for mortality and vasospasm rates: 1) The ratio of angiographically confirmed vasospasm on DSA was more prominent in Fisher grade 3 and 2) the

ratio of clinical vasospasm was more prominent in Fisher grade 4. Additional studies may required to clarify this issue.

The optimal management of patients with unruptured intracranial aneurysms remains controversial. Treatment of unruptured intracranial aneurysms are influenced by many factors including; 1) patient factors, such as previous aneurysmal SAH, age, and coexisting medical conditions, 2) aneurysm characteristics, such as size, location, and morphology, 3) factors in management, such as the experience of neurosurgeon (25). In our study, eight patients were presented with symptoms such as mass effect on cerebral or brainstem structures or cranial nerve palsies. Eight unruptured intracranial aneurysms were found on DSA in these patients. Seven of them were young patients with a long life expectancy and the aneurysms were suitable for clipping. One patient was old with the age of 70 years but has not coexisting medical problems and considered to be medically suitable for surgery. All patients were treated surgically without any major morbidity. We recommend operative management in patients with unruptured intracranial aneurysms. It seems the most effective treatment strategy to prevent the patient for SAH. However, this decision requires an accurate assessment of the factors that were listed above.

As summary, there is no universal management protocol for SAH and timing of surgery for ruptured intracranial aneurysms. Predictives of outcome are influenced by many factors in patients with aneurysmal SAH. On the basis of our results we suggest that initial WFNS score and Fisher grade are important prognostic factors in aneurysm surgery.

REFERENCES

1. Atkinson JL, Sundt TM, Houser OW, Whisnant JP. Angiographic frequency of anterior circulation intracranial aneurysms. *J Neurosurg* 1989, 70: 551-555.
2. Nakagawa T and Hashi K. The incidence and treatment of asymptomatic, unruptured cerebral aneurysms. *J Neurosurg* 1994, 80: 217-223.
3. Kassell NF, Torner JC, Haley EC, Jane JA, Adams HP, Kongable GL. The international cooperative study on the timing of aneurysm surgery. Part 1: Overall management results. *J Neurosurg* 1990, 73: 18-36.
4. Kassell NF, Torner JC, Jane JA, Haley EC, Adams HP. The international cooperative study on the timing of aneurysm surgery. Part 2: Surgical results. *J Neurosurg* 1990, 73: 37-47.
5. Sundt TM, Kobayashi S, Fode NC, Whisnant JP. Results and complications of surgical management of 809 aneurysms in

722 cases. Related and unrelated to grade of patient, type of aneurysm, and timing surgery. *J Neurosurg* 1982, 56: 753-765.

6. Ropper AH and Zervas NT. Outcome 1 year after SAH from cerebral aneurysm. Management morbidity, mortality, and functional status in 112 consecutive good-risk patients. *J Neurosurg* 1984, 60: 909-915.

7. Ausman JI, Diaz FG, Malik GM, Andrews BT, McCormick PW, Balakrishnan G. Management of cerebral aneurysms: Further facts and additional myths. *Surg Neurol* 1989, 32: 21-35.

8. Naso WB, Rhea AH, Poole A. Management and outcomes in a low-volume cerebral aneurysm practice. *Neurosurgery* 2001, 48: 91-100.

9. Chung RY, Carter BS, Norbash A, Budzik R, Putnam C, Ogilvy CS. Management outcomes for ruptured and unruptured aneurysms in the elderly. *Neurosurgery* 2000, 47: 827-833.

10. Beskonakli E, Ergungor MF, Ergun R, Akdemir G, Cayli S, Bostanci U, Gul B, Yuksel M, Taskin Y. Spontaneous Subarachnoid hemorrhage and intracranial aneurysms: Clinical analysis in 122 cases. *The Medical Journal of A. Numune Hospital* 1996, 36: 90-95.

11. Gilsbach JM and Harders AG. Morbidity and mortality after early aneurysm surgery- a prospective study with nimodipine prevention. *Acta Neurochir (Wien)* 1989, 96: 1-7.

12. Öhman J and Heiskanen O. Effect of nimodipine on the outcome of patients after aneurysmal subarachnoid hemorrhage and surgery. *J Neurosurg* 1988, 69: 683-686.

13. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomography scanning. *Neurosurgery* 1980, 6: 1-9.

14. Fisher CM, Roberson GH, Ojemann RG. Cerebral vasospasm with ruptured saccular aneurysm- the clinical manifestations. *Neurosurgery* 1977, 1: 245-248.

15. Hernesniemi J, Vapalahti M, Niskanen M, Tapaninaho A, Kari A, Luukkonen M, Puranen M, Saari T, Rajpar M. One-year outcome in early aneurysm surgery: a 14 years experience. *Acta Neurochir (Wien)* 1993, 122: 1-10.

16. Miyaoka M, Sato K, Ishii S. A clinical study of the relationship of timing to outcome of surgery for ruptured cerebral aneurysms. A retrospective analysis of 1622 cases. *J Neurosurg* 1993, 79: 373-378.

17. Kassell NF and Drake CG. Timing of aneurysm surgery. *Neurosurgery* 1982, 10: 514-519.

18. Graf CJ and Nibbelink DW. Cooperative study of intracranial aneurysms and subarachnoid hemorrhage. Report of a randomized treatment study. III. Intracranial surgery. *Stroke* 1974, 5: 559-601.

19. Nishimoto A, Ueta K, Onbe H, Kitamura K, Omae T, Goto F, Ohneda G, Chigasaki H, Tsuru M, Suzuki J, et al. Nationwide co-operative study of intracranial aneurysm surgery in Japan. *Stroke* 1985, 16: 48-52.

20. Asano T and Sano K. Pathogenetic role of no-reflow phenomenon in experimental subarachnoid hemorrhage in dogs. *J Neurosurg* 1977, 46: 454-466.

21. Bederson JB, Levy AL, Ding WH, Kahn R, DiPerna CA, Jenkins AL III, Vallabhajosyula P. Acute vasoconstriction after subarachnoid hemorrhage. *Neurosurgery* 1998, 42: 352-360.

22. Transquart F, Ades PE, Groussin P, Rieant JF, Jan M, Baulieu JL. Postoperative assessment of cerebral blood flow in subarachnoid haemorrhage by means of ^{99m}Tc-HMPAO tomography. *Eur J Nucl Med* 1993, 20: 53-58.

23. Sonobe M and Suzuki J. Vasospasmogenic substances produced following subarachnoid haemorrhage, and its fate. *Acta Neurochir (Wien)* 1978, 44: 97-106.

24. Harders AG and Gilsbach JM. Time course of blood velocity changes related to vasospasm in the circle of Willis measured by transcranial Doppler ultrasound. *J Neurosurg* 1987, 66: 718-728.

25. Bederson JB, Awad IA, Wiebers DO, Piepgras D, Haley EC, Brott T, Hademenos G, Chyatte D, Rosenwasser R, Caroselli C. Recommendations for the management of patients with unruptured intracranial aneurysms. A statement for healthcare professionals from the stroke council of the American Heart Association. *Stroke* 2000, 31: 2742-2750.