นิพนธ์ต้นฉบับ

Cerebral vasospasm evaluated by Transcranial Doppler Sonography in aneurysmal subarachnoid hemorrhage patients

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Objective : *To study the correlation of blood flow velocity of the middle cerebral artery in patients with subarachnoid hemorrhage, vasospasm and clinical outcome.*

Design : *Prospective study*

Setting : *Neurosurgical intensive care unit, King Chulalongkorn Memorial Hospital*

Subjects : *11 patients with aneurysmal subarachnoid hemorrhage*

Methods : *Blood flow velocity of the middle cerebral artery measured by Transcranial Doppler Ultrasound and clinical outcome were compared.*

Results : *Four patients had clinical vasospasm. Two had high flow velocity in the middle cerebral artery since admission. The others had steep rising of the velocity few days prior to neurological deficit. Of these four patients, three had poor outcome. One patient with good outcome had reversible deficit and transient increased flow velocity. Seven patients did not develop clinical vasospasm and all had good outcome. One of them had transient rising of blood flow velocity.*

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Conclusions : *High flow velocities of proximal MCA in the first 4 days after aneurysmal subarachnoid hemorrhage is associated with poor outcome. After that, the acute increased maximal flow velocity is as important as the absolute values of maximal flow velocities for prediction of the development of cerebral infarction secondary to vasospasm. Rapidly decreased flow veloctiy within 2 - 4 days after a steep rising indicates good prognosis.*

Key words : *Vasospasm, Subarachnoid hemorrhage, Transcranial Doppler.*

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วัตถุประสงค์ : *เพื่อศึกษาความสัมพันธ์ระหว่างความเร็วของการไหลของโลหิตในหลอดเลือด Middle cerebral artery เปรียบเทียบกับการเกิดหลอดเลือดสมองหดตัวและอาการของผู้ป่วย*

รูปแบบการวิจัย : *การศึกษาแบบไปข้างหน้า*

สถาบันการศึกษา : *สาขาประสาทวิทยา และประสาทศัลยศาสตร์ คณะแพทยศาสตร์ จุฬาลงกรณ์-มหาวิทยาลัย*

ผู้เข้าร่วมการศึกษา : *ผู้ป่วยหลอดเลือดแตกในชั้น Subarachoid ที่เกิดจากหลอดเลือดโป่งพอง จำนวน 11 ราย*

วิธีการวิจัย : *ศึกษาความเร็วของการไหลของโลหิตในหลอดเลือดสมอง โดย Transcranial Ultrasound เทียบกับอาการผู้ป่วย*

ผลการวิจัย : *ผู้ป่วย 4 รายมีอาการที่เกิดจากหลอดเลือดสมองหดตัว ในจำนวนนี้ 2 รายมีความเร็วของอาการไหลของโลหิตสูงตั้งแต่แรกรับ อีก 2 รายมีความเร็วเพิ่มขึ้นอย่างรวดเร็ว 2-3 วันก่อนจะมีอาการผิดปกติทางระบบประสาทที่เกิดจากหลอดเลือดหดตัว ในผู้ป่วย 4 รายนี้ 3 ราย มีอาการไม่ดี อีก1 รายอาการดีขึ้น ผู้ป่วยอีก 7 รายไม่มีอาการผิดปกติที่เกิดจากหลอดเลือดสมองหดตัว ในจำนวนนี้ 1 รายมีความเร็วของโลหิตเพิ่มขึ้นชั่วคราวและลดลงได้เอง*

วิเคราะห์-สรุปผล : *การมีความเร็วของการไหลขงโลหิตสูงใน 4 วันแรก บ่งบอกว่าผู้ป่วยน่าจะมีผลลัพธ์ที่ไม่ดี หลังจาก 4 วันแล้วการติดตามค่าความเร็วการไหลของโลหิตก็ยังมีประโยชน์ ในผู้ป่วยที่มีความเร็วเพิ่มขึ้นอย่างรวดเร็วเป็นข้อบ่งชี้ว่าจะมีการหดตัวของหลอดเลือดขึ้น ในทางตรงกันข้ามถ้ามีความเร็วของการไหลของโลหิตลดลงอย่างรวดเร็วจะบ่งถึงการพยากรณ์โรคที่ดี*

Cerebral vasospasm is one of the most important causes of morbidity and mortality in patients with aneurysmal subarachnoid hemorrhage (SAH). Generally, demonstration of arterial narrowing by angiography is the gold standard for diagnosis of intracranial vasospasm. However, angiography is an invasive procedure and cannot be repeated at frequent intervals. Another non-invasive way to monitor the vasospasm is Transcranial Doppler Ultrasound (TCD).[1] In western countries, TCD has been widely used for evaluation of vasospasm in patients with subarachnoid hemorrhage. It can be repeated as often as necessary at the bedside. Doppler technology allows for accurate measurement of blood flow velocity in large arteries around the circle of Willis. In vasospasm, arterial narrowing causes increased velocity of flow through the affected segment which can be demonstrated by TCD. It can also help physicians for early detection of vasospasm in asymptomatic patients.

The authors report a prospective study of TCD findings in patients with aneurysmal subarachnoid hemorrhage who were admitted at the Neurosurgical intensive care unit, King Chulalongkorn Memorial hospital during July to November 1998. The objective of this study was to compare the differences of flow velocities in the proximal middle cerebral arteries in patients with symptomatic and asymptomatic vasospasms.

Clinical material and methods

From July to November 1998, 11 cases of ruptured intracranial aneurysms were admitted and selected for the study. Their clinical grades at presentation, according to the classification of Hunt and Hess, and the dates of admission are shown in Table 1. All patients had spontaneous SAH proven by CT scan or by bloody and/or xanthochromic cerebrospinal fluid. Cerebral aneurysm was verified as the source of bleeding by immediate angiography after admission. Location of the 11 aneurysms are summarized in Table 2. All patients underwent aneurysms clipping in acute period. Dates of those operations are summarized in Table 3. All cases were treated with standard regimen for subarachnoid hemorrhage including analgesics, calcium antagonists (Nimodipine) and prophylactic Triple-H therapy. No steroid or antifibrinolytic drugs were used.

Table 1. Clinical grades and days of admission in 11 patients.

Clinical grade	Days after SAH				
I	0	0	1	0	0
II	0	2	2	0	0
III	0	1	2	0	1
IV	0	2	0	0	0

Table 2. Location of 11 ruptured aneurysms in these series.

Artery	No. of cases
Posterior communicating	6
Anterior communicating	4
Anterior choroidal	1

Table 3. Clinical grades and dates of operative in 11 patients.

Patient No.	Clinical grade	Date of operation (Days after SAH)
1	2	1
2	4	1
3	1	3
4	3	3
5	2	2
6	2	2
7	2	2
8	3	3
9	4	2
10	3	7
11	3	5

Flow velocities of proximal MCA on the affected sides or the craniotomy sides were recorded by TCD on alternate day basis for at least 14 days in all patients. In case of clinical deterioration, CT scans were repeated to exclude rebleeding or hydrocephalus and to evaluate the size of the cerebral ischemia.

New focal neurological deficits found in the postoperative period were defined as clinical vasospasm after they were excluded for other causes such as rebleeding or hydrocephalus. Patients with new cerebral infarction diagnosed by CT scan was also defined as clinical vasospasm.

For final analysis, maximum and mean flow velocities of the middle cerebral arteries in patients with good outcome and without clinical vasospasm were compared to those with poor outcome and clinical vasospasm. Poor outcomes were defined as death or major neurological deficits such as aphasia or hemiplegia.

Results

Findings in all patients are summarized in Table 4.

Table 4. Maximal flow velocities of proximal MCA in ruptured aneurysmal SAH patients (cm/s) Days after SAH.

Case No.	Grade	Location	2	4	6	8	10	12	14	16	18	20	22	24	Result
1	2	Acom.	94	137	151	148	160	164	157	183					good
2	4	Ant.chor	96	102	162	153	118	122	140	104	102				good
3	1	Pcom.		135	130	148	130	120	138	116	93	114			good
4	3	Acom.	97	136	110	121	138	155	109	93					good
5	2	Pcom.	68	90	112	138	140	127	166	89	87	86			poor
6	2	Pcom.	183	191	199	188	192	196	160	145	178				poor
7	2	Pcom.		74	86	88	86	84	86						good
8	3	Acom.	54	112	127	135	94	53	64	59					good
9	4	Pcom.		143	157										poor
10	3	Pcom.			122	211	122	146	82	90	74	75	64	69	good
11	3	Acom.			128	118	114	109	119	128					good

There were 4 patients with clinical vasospasm, three of them had poor outcomes and the other had good outcome.The three patients with bad outcome (case No. 5,6,9) suffered from cerebral infarction diagnosed by clinical and confirmed by CT scan. One of them died form massive cerebral infarction despite a decompressive lobectomy. The patient with good outcome (case No.8) had a reversible neurological deficit without CT evidence of rebleeding.

Patient No.5 had a left posterior temporal infarction proven by CT scan on day 14 post-op. He had a steep rising of the flow velocity in the MCA two days before the cerebral infarction was detected.

Patient No. 6 had a left frontotemporal infarction diagnosed by CT scan on day 12. She had a relatively high maximal flow velocities of the middle cerebral artery since the day of admission and continued to have high flow velocity throughout the course.

Patient No.8 had a new onset of hemiparesis on day 7 after SAH. She had a steep rising and a rapid decreasing of the velocity curve. With medical treatment and triple H therapy, the hemiparesis was completely reversible within 3 days.

Patent No.9 had a massive cerebral infarction and died on day 2 post-operatively. The flow velocity in this patient was higher than those of patients without clinical vasospasm since admission.

Seven patients without clinical vasospasm had moderately increased maximal and mean flow velocities postoperatively on day 6 - 10. Among them, patient No.10 had an acute increased and rapidly decreased flow velocity without new focal neurological deficit. He had good outcome.

Maximal flow velocities in patients without clinical vasospasm, compared with those with clinical vasospasm, are presented in figure 1 - 5.

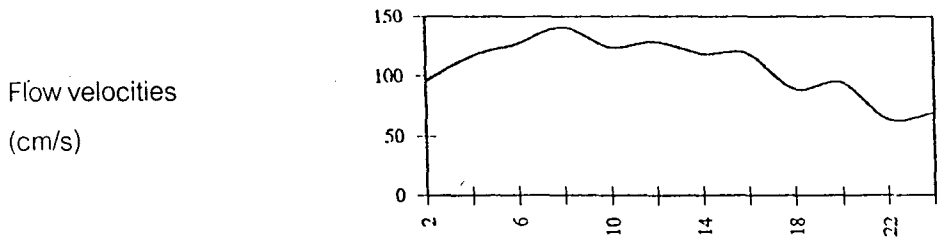

Figure 1. Maximal flow velocities of 7 patients without clinical vasospasm.

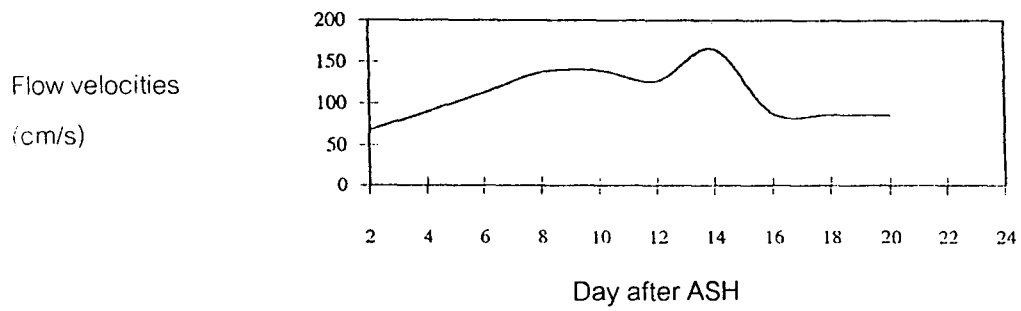

Figure 2. Maximal flow velocities of the patient No.5

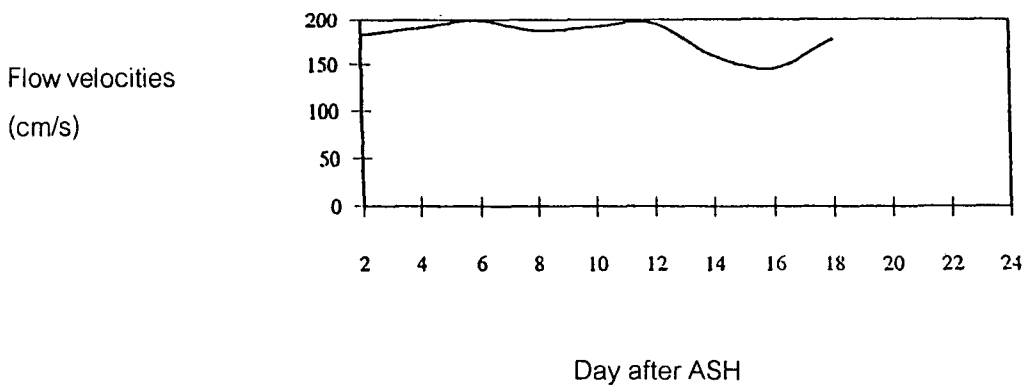

Figure 3. Maximal flow velocities of the patient No.6

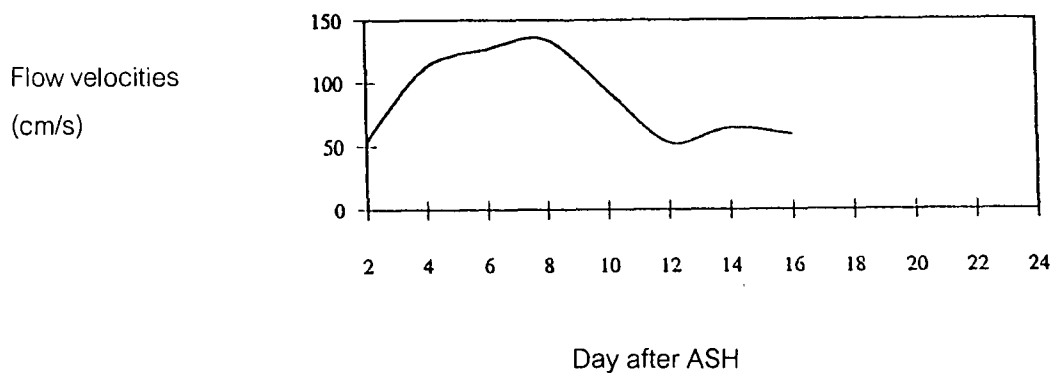

Figure 4. Maximal flow velocities of the patient No.8

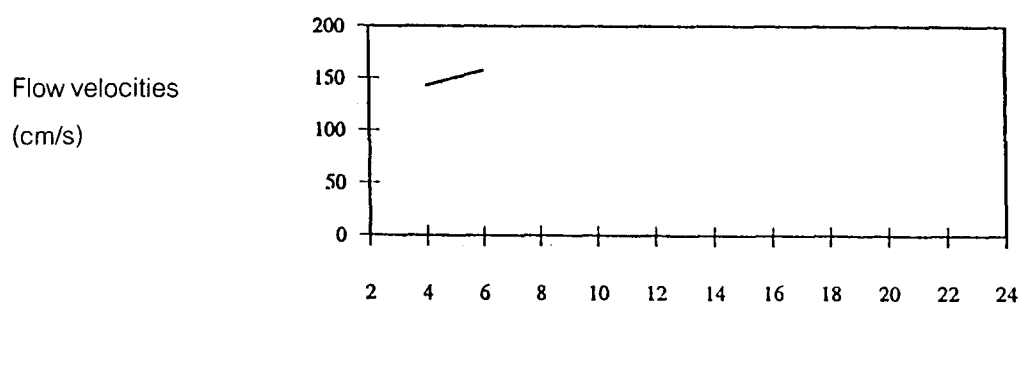

Figure 5. Maximal flow velocities of the patient No.9

Discussion

Former studies have shown the significant change in flow velocity of the cerebral arteries in patients with symptomatic vasospasm after subarachnoid hemorrhage.[2] High flow velocity on the 2nd and 3rd day after SAH as well as a steep increase of flow velocity curve highly suggested vasospasm and predicted poor outcome. This series confirms the findings, two patients with clinical vasospasm had high flow velocities within the first 4 days after SAH. The other two who did not have high flow velocity values in the first 4 days, had a steep increased flow velocity before the development of cerebral infarction. In these patients, absolute flow velocities were not very high. Due to small number of patients in this series, statistical significance could not be established. However, our data suggested that high flow velocities in the middle cerebral artery in the first 4 days after aneurysmal subarachnoid hemorrhage is associated with poor outcome from cerebral infarction. Moreover, close monitoring of the flow velocity is necessary for upto 2 weeks after SAH since one of our patient (No 5) developed a symptomatic vasospasm on day 14. In this patient, gradually increased flow velocity during the first 10 days was observed followed by a rapidly increased velocity two days before the symptoms of vasospasm occurred. Our findings suggest that the acute increased maximal flow velocity is as important as the absolute values of maximal flow velocities for prediction of the development of cerebral infarction secondary to vasospasm. Rapidly decreased flow velocitiy within 2-4 days after a steep rising, however, indicates a better prognosis.

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