The accuracy of transcranial doppler for predicting cerebral infarction in aneurysmal subarachnoid hemorrhage

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The accuracy of transcranial doppler for predicting cerebral infarction in aneurysmal subarachnoid hemorrhage

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ABSTRACT

The accuracy of transcranial doppler for predicting cerebral infarction in aneurysmal subarachnoid hemorrhage

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The present study evaluated the accuracy of transcranial Doppler (TCD) using different vasospasm criteria for the predicting the presence of cerebral infarction due to symptomatic vasospasm. We retrospectively evaluated the clinical and radiological information of all patients consecutively admitted with acute aneurysmal subarachnoid hemorrhage (SAH) in the anterior cerebral circulation, between January 2001 and June 2002. TCD examinations were performed on alternate day for up to 20 days after admission. Cerebral infarction was defined as a new hypodensity in the vascular distribution by CT with appropriate clinical symptoms. A total of 93 patients with aneurysmal SAH in the anterior cerebral circulation were included. TCD indicated the

presence of vasospasm in 60 patients (64.5%). Cerebral infarction on CT with clinical symptoms occurred in 23 patients (24.7%). Evidence of vasospasm on TCD predicted cerebral infarction occurrence by multivariable logistic regression analysis (OR 3.11, 95% CI 1.46 to 6.59). Thus we calculated the diagnostic accuracy of TCD for predicting the presence of cerebral infarction. When TCD showed vasospasm, its sensitivity was 82.6%, specificity 41.4%, positive predictive value 31.7%, and negative predictive value 87.9%, and when TCD showed severe vasospasm, its sensitivity was 69.6%, specificity 77.1%, positive predictive value 50.0%, and negative predictive value 88.5%. Vasospasm on TCD was found to be predictive of symptomatic cerebral infarction on CT but its positive predictive value was low despite of the adoption of severe vasospasm criteria on TCD.

Key Words : Transcranial Doppler, Cerebral Infarction, Subarachnoid Hemorrhage

Ⅰ. INTRODUCTION

Symptomatic vasospasm represents a major cause of morbidity and mortality in patients with aneurysmal subarachnoid hemorrhage (SAH) and carries a 15% to 20% risk of stroke or death.[1] The incidence of cerebral infarction caused by vasospasm varies according to the imaging technique used for diagnosis. Rates of cerebral infarction after SAH range up to 40% by computed tomography (CT),[2] and may be as high as 81% by magnetic resonance imaging (MRI).[3]

Transcranial Doppler (TCD) is based on the principle that given a constant cerebral blood flow, the flow velocity is inversely proportional to the vessel lumen area. Therefore, TCD seems ideally suited to detect vasospasm due to its noninvasiveness, with repeatability and low associated cost. Indeed TCD has been implemented by many neurosurgical units.[4] Several studies that have assessed the diagnostic accuracy of TCD have used symptomatic vasospasm or cerebral infarction as the primary outcome.[2, 5-7] However much less is known about the accuracy of TCD for predicting cerebral infarction on brain CT due to symptomatic vasospasm. In the present study, we evaluated the accuracy of TCD using different vasospasm criteria for predicting the presence of cerebral infarction due to symptomatic vasospasm.

Ⅱ.PATIENTS AND METHODS

1. Patients

We retrospectively reviewed the clinical and radiological information of all patients consecutively admitted to our hospital with acute aneurysmal SAH between January 2001 and June 2002. Patients were included if they had been admitted within 7 days of SAH onset and had a ruptured aneurysm by angiography. Patients with fusiform, traumatic or mycotic aneurysms were excluded. In all patients included in this study the ruptured aneurysm had to be located in the anterior cerebral circulation. Patient neurologic statuses on admission were classified according to the Hunt and Hess grading system (H&H grade),[8] and patients classified as H&H grade V were excluded. An initial CT scan was performed in each case at admission. The amount of blood clots in the subarachnoid space was classified according to the Fisher grading scale.[9]

2. Methods

TCD examinations were performed by an experienced technician on alternative days during the 20 day period following admission. Mean blood

flow velocities (MBFV, cm/sec) of the major anterior circulation vessels were measured through a transtemporal and transorbital window using a 2-MHz hand-held transducer probe (Trans-Scan, Eden Medizinische Electronic, Ueberlingen, Germany). MBFV ≥ 120 cm/sec in the anterior cerebral artery (ACA), middle cerebral artery (MCA) or intracranial internal carotid artery (ICA) were deemed indicative of vasospasm, and MBFV ≥ 120 cm/sec and < 180 cm/sec in the ACA, MCA or intracranial ICA were defined as mild vasospasm on TCD, and ≥ 180 cm/sec as severe vasospasm on TCD.

Cerebral infarction was defined as a new hypodensity located in a vascular distribution on CT with clinical symptoms, including an insidious onset of confusion, a decline in level of consciousness, or focal deficits not explained by other causes (rebleeding, hydrocephalus, electrolyte disturbances, hypoxia, or seizure). Cerebral infarction possibly related to complications of surgery or angiography was excluded from analysis. A single neuroradiologist, who unaware of clinical information, reviewed all CT scans and divided each cerebral infarction into its corresponding vascular territory using a validated arterial territory map.[10]

On admission, all patients were treated with intravenous fluids to maintain euvolemia. Before aneurysms were secured, systolic blood pressures were kept < 160 mmHg. Oral calcium channel blocker - nimodipine (Bayer, Germany) was administered to all patients for prophylaxis of cerebral vasospasm from the time of admission. An intensive care protocol with

'HHH' therapy (induced hypertension, hypervolemia, hemodilution) was mandated for those patients with symptomatic vasospasm.

3. Statistic Analysis

SPSS software for window was used throughout (version 12.9, SPSS, Inc., Chicago, Illinois, USA). Categorical variables were compared using the chi-square test (Pearson, Yate's continuing correction or Fisher's exact test), and continuous variables were compared between groups using the Student's T-test. Predictors of cerebral infarction were evaluated by multivariable logistic regression analysis. Variables included were; age, sex, ruptured aneurysm location, HH grade, Fisher grading scale, treatment, vasospasm of TCD and extraventricular drainage. These variables were selected based on their presumed biological and clinical importance. Sensitivity, specificity, positive predictive value and negative predictive value of TCD for cerebral infarction after aneurysmal SAH in the anterior cerebral circulation were calculated. The predictive performance of the TCD was assessed with the receiver operating characteristic (ROC) curve which plots versus false positive test results at various cut-ff points. *P* values of <0.05 was considered significant.

Ⅲ.RESULTS

A total of 93 patients (56 females, 37 males) with aneurysmal SAH in the anterior cerebral circulation were included in this study. Mean patient age was 53.6 years (range 21 to 77 years). The most common location of a ruptured aneurysm was the anterior communicating artery. Seventy-eight patients underwent surgical clipping and 15 patients were treated with endovascular coil embolization. Mean time from SAH onset to aneurysm treatment was 4 days (range 0 to 28 days). All patients had at least 1 CT scan after aneurysm treatment and two more CT scans after cerebral infarction symptom onset. TCD indicated presence of vasospasm in 60 (64.5%) of the 93 study subjects. Among these 60 patients, mild vasospasm on TCD was detected in 28 and severe vasospasm on TCD in 32 (Table 1).

Table 1. Distribution of demographic, clinical, radiological and treatment characteristics in 93 patients with aneurysmal subarachnoid hemorrhage

	Total (n = 93)	Cerebral infarction Absent	Cerebral infarction Present	*P*
Age, years, mean ± SD	53.6 ± 12.7	54.8 ± 12.7	49.9 ± 12.	> 0.05
Sex				> 0.05
Male	37	27	10	
Female	56	43	13	
Ruptured aneurysm location				> 0.05
Anterior communicating	37	25	12	
Posterior communicating	14	12	2	
Middle cerebral	24	17	7	
Anterior cerebral	6	5	1	
Anterior choroidal	6	6	0	
Internal carotid	6	5	1	
Hunt and Hess Grade				> 0.05
I	5	5	0	
II	40	30	10	
III	31	21	10	
IV	17	14	3	
Fisher Grade				> 0.05
I	6	6	0	
II	15	12	3	
III	65	46	19	
IV	7	6	1	
Treatment				> 0.05
clipping	78	58	20	
coiling	15	12	3	
Vasospasm on TCD				< 0.05
Absent	33	29	4	
Present Mild	28	25	3	
Severe	32	16	16	
Extraventricular drainage				> 0.05
Not done	79	59	20	
Done	14	11	3	

Cerebral infarction on CT scan with clinical symptoms occurred in 23 patients. (24.7% of the study population) Using multiple logistic regression models, age, sex, ruptured aneurysmal location, clinical grade using the H&H grade, Fisher grading scale, surgical clipping and extraventricular drainage during the acute stage were not found to be significantly associated with the occurrence of cerebral infarction, but vasospasm on TCD remained predictive of a cerebral infarction (Table 2).

Table 2. Predictors of cerebral infarction by multivariate logistic regression analysis in 93 patients with acute aneurysmal subarachnoid hemorrhage

	Odds Ratio	95% CI	*P*
Age (10-year increase)	1.00	0.63-1.59	0.99
Male vs female	0.88	0.28-2.70	0.81
Ruptured aneurysm location	0.81	0.56-1.18	0.27
Hunt and Hess Grade	1.21	0.60-2.41	0.60
Fisher Grade	0.94	0.40-2.21	0.88
Clipping and coiling treatment	1.36	0.67-2.77	0.39
Vasospasm on TCD	3.11	1.46-6.59	0.03
Extraventricular drainage	0.68	0.13-3.45	0.64

The diagnostic accuracy of TCD was then studied for the prediction of cerebral infarction. When TCD showed vasospasm, its sensitivity was 82.6%, specificity 41.4%, positive predictive value 31.7%, and negative predictive value 87.9%, and when TCD showed severe vasospasm, its sensitivity was 69.6%, specificity 77.1%, positive predictive value 50.0%, and negative predictive value 88.5% (Table 3).

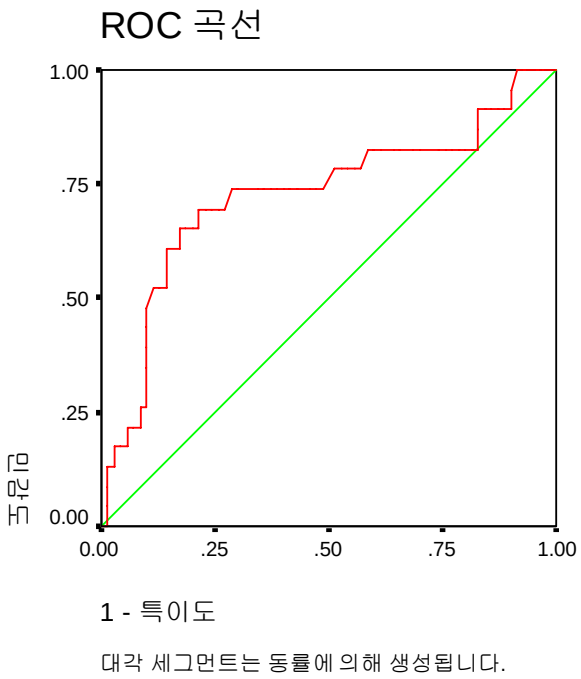
Table 3. Sensitivity (Sn), specificity (Sp), positive predictive value (PPV), and negative predictive value (NPV) of transcranial Doppler to detect infarction in 93 patients with aneurysmal subarachnoid hemorrhage

	Cerebral infarction Present	Cerebral infarction Absent	Total
Vasospasm on TCD	(Sn 82.6%, Sp 41.4%, PPV 31.7%, NPV 87.9%)		
120 cm/sec ≥	19	41	60
120 cm/sec <	4	29	33
Severe Vasospasm on TCD	(Sn 69.6%, Sp 77.1%, PPV 50.0%, NPV 88.5%)		
180 cm/sec ≥	16	16	32
180 cm/sec <	7	54	61

The areas under the ROC curve were equal to 0.725 (95% CI 0591-0.860) (Figure 1). The MBFV threshold value for TCD that discriminated best between patients with and without cerebral infarction giving the fewest false negative classification was 179 cm/sec. For the thresholds value, the sensitivity and specificity of TCD to predict cerebral infarction were 73.9% and 71.4%.

Cerebral infarctions occurred in 10 patients in the ACA territory and in 13 patients in the MCA territory. Cerebral infarction in a branch territory of the ICA, such as, anterior choroidal artery, was not observed. Vasospasm on TCD was not documented in 3 patients (30%) in ACA territory and in only 1 patient (7.7%) in MCA territory. Cerebral infarctions without vasospasm on TCD were more frequent in patients with ACA territory than those with MCA territory involvement, but this was not significant ($P>0.05$).

Figure 1. Detection of cerebral infarction by the vasospasm on TCD. ROC-curve with a reference line.



Ⅳ. DISCUSSION

As with any diagnostic test, the choice of a reference standard against which to evaluate the test accuracy is of central importance. The majority of studies to date have assessed the diagnostic accuracy of TCD using angiographic vasospasm[11] or cerebral infarction due to symptomatic vasospasm as the primary outcome measure.[2, 5-7] Moreover, several authors were unable to find any correlation between TCD and angiographic results in patients with vasospasm. A recent meta-analysis performed on 26 trials comparing TCD with cerebral angiography to detect vasospasm in patients with SAH suggested that the TCD of the MCA has a high specificity (99%) and positive predictive value (97%) but a low sensitivity (67%). This study also found that TCD of other arteries lacks evidence of accuracy.[11] In addition cerebral infarction may occur in patients without apparent vasospasm on angiography. Conversely, the presence of a documented vasospasm does not always portend neurologic deterioration. Among those patients who developed an angiographic vasospasm, only 50% showed neurologic deficits.[12] In fact, the presence of a cerebral infarction on CT, but not angiographic vasospasm, has been found to be correlated with elevated mortality and a poor functional outcome.[2] Therefore, in the present study we chose cerebral infarction due to symptomatic vasospasm as the primary

outcome.

To the best of our knowledge, previous investigators have used an arbitrary TCD mean cut-off velocity to discriminate between those with vasospasm and those without vasospasm. The cut-off values used in MCA are 120 cm/sec, 130 cm/sec or 140 cm/sec, and in ACA and ICA they are 90 cm/sec, 110 cm/sec or 120 cm/sec.[2,6-7,11] Studies using cerebral infarction as the primary outcome have used an MVFV of 120 cm/sec in MCA, ACA and ICA to define a positive TCD. [2,6,] Therefore, we used an MBFV of 120 cm/sec in MCA, ACA or intracranial ICA to determine vasospasm on TCD. In addition, most authors have considered MBFV values exceeding 200 cm/sec to be diagnostic of vasospasms that may subsequently lead to a delayed neurologic deficit, but opinions have recently changed. Mascia et al[5] suggested that an MBFV threshold of 160 cm/sec as calculated by ROC analysis can accurately detect clinical vasospasm. In the present study, the ROC analysis defined a TCD velocity threshold equal to 179 cm/sec for cerebral infarction (sensitivity 73.9%, specificity 71.4%). Therefore we defined an MBFV $\geq$ 180 cm/sec in MCA, ACA or intracranial ICA as severe vasospasm on TCD. Most of previous studies used one cut-off criterion to determine a positive TCD for accuracy calculations. [2,6,7,11] Our study is unique in that we used two different vasospasm criteria on TCD, and we calculated the diagnostic accuracy for predicting the presence of cerebral infarction by using both vasospasm and severe vasospasm on TCD.

In the present study, the incidence of cerebral infarction on CT was 24.7%, which is within the range (16- 40%) reported by other authors. [2, 5-7] TCD indicated the presence of vasospasm in 60 (64.5%) of 93 patients, and severe vasospasm was detected by TCD in 32 patients (34.4%). Evidence of vasospasm on TCD was found to predict cerebral infarction occurrence by multivariable logistic regression analysis. Thus, we calculated the diagnostic accuracy of TCD for the prediction of cerebral infarction. When TCD revealed vasospasm, its sensitivity was 82.6%, its specificity was 41.4%, the positive predictive value was 31.7%, and the negative predictive value was 87.9%. These findings strongly agree with several other reports, except for the low specificity value. Rabinstein et al[2] reported a sensitivity of 90%, a specificity of 44%, a positive predictive value of 55%, and a negative predictive value of 86%. Suarez et al[6] found a sensitivity of 70%, a specificity of 73%, a positive predictive value of 39%, and a negative predictive value of 90%. The overall positive predictive value was low. When the TCD showed severe vasospasm, its sensitivity was 69.6%, its specificity was 77.1%, its positive predictive value was 50.0%, and its negative predictive value was 88.5%. Despite the use of strict TCD criteria, its positive predictive value was suboptimal. The velocity–diameter relationship has three distinct Regimes of 'Spencer Curves'[13]: (1) the 'forward side of the curve where the flow is relatively constant and the velocity is inversely related to diameter; (2) the plateau regime where the flow is reduced while the velocity remains relatively

independent of the diameter, and (3) the 'backside' of the curve where additional diameter reductions result in lower velocities and where the flow is reduced to critical values. In plateau and 'backside' curves where the cerebral autoregulation is ineffective, the arterial blood pressure influences the velocity-diameter relationship.[14] When patients exhibited symptomatic vasospasm, we treated it with "HHH' therapy. The blood volume flow increased and some of cerebral infarction was not evident, which could be one reason for the low positive predictive value.

Recently, multiple studies have examined early predictors of symptomatic vasospasm, or cerebral infarction on CT or MRI. By using a multivariate analysis Rabinstein et al[2] reported that the evidence of vasospasm on TCD and angiogram is predictive of cerebral infarction on CT. Shimoda et al[15] found that poor SAH grade, advanced age, vasospasm on angiogram, multiple cortical infarcts on MRI consistent with vasospasm, and chronic hydrocephalus were predictive of symptomatic infarction on MRI. Qureshi et al[16] reported that the thickness of the SAH on CT, an early rise in MCA-MBFV, defined as a value ≥ 110 cm/sec recorded on or before post-SAH day 5, a Glasgow Coma Scale score <14, and the rupture of ACA or ICA were all predictive of symptomatic vasospasm. Charpentier et al[17] found that an age < 50 years, good neurologic grade and hyperglycemia were predictive of symptomatic vasospasm. In the present study, evidence of vasospasm on TCD predicted cerebral infarction occurrence by multivariable logistic regression

analysis whereas age, sex, ruptured aneurysm location, HH grade, Fisher grading scale, treatment and extraventricular drainage did not. We excluded H&H grade V but included H&H grade IV. It is difficult to accurately diagnose neurological deterioration caused by vasospasm in such patients, and the relationship between clinical grades and symptomatic cerebral infarction occurrence may be underestimated.

Besides the absolute velocity criteria, an ominous sign of pending complications due to cerebral vasospasm is a large day-to-day increase in the TCD velocity during days 3-7 after SAH. In a study of 121 patients, Grosset et al[18] reported that a rate of increase ≥ 50 cm/sec/day in MBFV was another TCD criterion associated with symptomatic vasospasm. In the present study, we did not mandate daily TCD recordings so this variable was not analyzed.

Previous comparative studies on angiographic vasospasm[11] or cerebral infarction due to symptomatic vasospasm[6], revealed that the diagnostic accuracy of TCD for ACA was lower than that for MCA. We calculated the frequencies of cerebral infarction by vascular territory with or without vasospasm on TCD. Cerebral infarctions without vasospasm on TCD were more frequent in patients with ACA territory (30%) than in patients with MCA territory (7.7%), which was not significant ($P>0.05$).

This study has several limitations. First, our definition of vasospasm did not include a measurement of the Lindegaard ratio for the differentiation of true vasospasm from hyperemia.[19] Second, the number of patient samples was

relatively small. As was found by previous studies,[2,6] our study showed a high negative predictive value but its interpretation is limited.

Ⅴ. CONCLUSION

In conclusion, we evaluated the accuracy of TCD using two different vasospasm criteria to predict cerebral infarction due to symptomatic vasospasm. Vasospasm on TCD is predictive of symptomatic cerebral infarction on CT but its positive predictive value is low despite our adoption of the criteria of severe vasospasm on TCD. Moreover, our observations indicate that TCD data and its interpretation may be correlated with neurologic symptoms.

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국문요약

지주막하 출혈에서의 뇌졸중 발생 예측에 대한 경두개 도플러 초음파의 정확도

이 재 민

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지주막하 출혈에서는 혈관경련에 의해 뇌졸중이 발생할 수 있다. 본 연구에서는 각기 다른 혈관경련 기준을 사용하여 혈관경련에 의한 뇌졸중 발생에 대한 예측인자로서의 경두개 도플러 초음파의 정확도에 대해 알아보고자 한다. 2001년 1월부터 2002년 6월까지 전뇌순환계 (anterior cerebral circulation)의 급성 지주막하 출혈로 입원한 환자들의 의무기록 및 방사선 소견을 후향적으로 조사하였다. 경두개 도플러 초음파는 내원 당일부터 내원 20일까지 시행하였으며, 뇌졸중은 합당한 신경학적 증상과 컴퓨터 단층 촬영 상 새로 발생한 저음영 영역이 있는 경우로 정의하였다. 총 93명의 전뇌

순환계 급성 지주막하 출혈 환자군 중 60명 (64.5%)의 환자에서 경두개 도플러 초음파 검사 상 혈관경련이 발견되었다. 뇌졸중은 23명 (24.7%)에서 발생하였다. 경두개 도플러 초음파 상 혈관경련이 뇌졸중 발생의 예측에 미치는 영향은 다인자 logistic 회귀분석을 통해 증명하였다 (OR 3.11, 95% CI 1.46 to 6.59). 이를 통해 뇌졸중 발생 예측에 대한 경두개 도플러 초음파의 정확도를 계산하였다. 경두개 도플러 초음파 상 혈관경련이 있을 때 뇌졸중에 대한 민감도는 82.6%, 특이도는 41.4%였으며 양성예측률은 31.7%, 음성예측률은 87.9%였다. 경두개 도플러 초음파 상 심한 혈관경련이 있을 때 민감도는 69.6%, 특이도 77.1%였으며, 양성예측률은 50.0%, 음성예측률은 88.5%였다. 본 연구에서 경두개 도플러 초음파 상 혈관경련은 뇌졸중 발생 예측 인자로서의 가치는 있었으나, 심한 혈관경련 기준을 적용해도 양성예측률은 낮았다.

핵심되는 말 : 경두개 도플러 초음파, 뇌졸중, 지주막하 출혈