European Review for Medical and Pharmacological Sciences 2018; 22: 2029-2035

Clinical diagnosis of cerebral vasospasm after subarachnoid hemorrhage by using transcranial Doppler sonography

D.-D. LI[1], J.-Y. CHANG[2], C.-X. ZHOU[2], J.-B. CUI[3]

[1]Department of Ultrasound, Weifang People's Hospital, Weifang, P.R. China
[2]Department of Neurosurgery, Weifang People's Hospital, Weifang, P.R. China
[3]NICU, Weifang People's Hospital, Weifang, P.R. China

Abstract. – OBJECTIVE: To investigate the diagnosis of cerebral vasospasm (CVS) after subarachnoid hemorrhage (SAH) by transcranial Doppler (TCD) sonography.

PATIENTS AND METHODS: 90 patients with SAH admitted to the Department of Neurosurgery of Weifang People's Hospital from January 2016 to December 2016 were selected. TCD and digital subtraction angiography (DSA) were used to diagnose the prevalence of CVS in patients. The severity of disease was evaluated (improved-Fisher grading). Correlations between neurological status (Hunt-Hess grading) and the prevalence of CVS were analyzed. It turned out that the prevalence of CVS was 87.78% detected by DSA and was 83.33% detected by TCD, no significant difference was found between them ($p > 0.05$).

RESULTS: The results of TCD showed that the gender, age, smoking, alcoholism, and history of hypertension had no significant correlations with the prevalence of CVS ($p > 0.05$). Blood flow velocity of patients was significantly higher at 4-6 days after the occurrence of SAH compared with the level at 1-3 days, reached the peak at 7-9 days, and decreased at 10-12 days after occurrence. Significant differences in the severity of the disease were found between patients with different improved-Fisher grades and different Hunt-Hess grades ($p < 0.05$). The prevalence of CVS was significantly increased after SAH ($p < 0.05$).

CONCLUSIONS: TCD can dynamically detect the blood flow velocity of SAH patients, and can be used for the prediction and diagnosis of CVS after SAH.

Key Words:
Transcranial Doppler sonography, Subarachnoid hemorrhage, Cerebral vasospasm.

Introduction

As one of the major complications after subarachnoid hemorrhage (SAH), the occurrence of cerebral vasospasm (CVS) can significantly increase the disability and mortality rates of patients with SAH[1,2]. It's generally believed that digital subtraction angiography (DSA) is the main method for the diagnosis of CVS. Although DSA is the gold standard for the diagnosis of vasospasm, the technique usually cannot be applied repeatedly to patients due to its invasive nature. Also, DSA cannot be used to observe cerebral blood flow, clinical changes cannot be monitored, so the application of DSA is limited[3,4]. Transcranial Doppler (TCD) sonography is a novel and noninvasive technique with the advantages of low cost, high reliability, and low risk. TCD can be used to monitor cerebral blood flow, so as to determine the changes in vascular caliber and identify the onset of CVS[5-7]. This method has been widely used in neurosurgery[8,9]. In this work, TCD was performed in 90 patients to determine the correlations between the severity of disease, neurological status and the prevalence of CVS, so as to provide evidence for the prediction and early diagnosis of CVS in patients with SAH.

Patients and Methods

Patients

Ninety patients with SAH were selected from the Department of Neurosurgery of Weifang People's Hospital from January 2016 to December 2016.

Inclusion criteria: 1- Patients with SAH confirmed by CT and intracranial aneurysm confirmed by DSA; 2- 30 to 70 years old without restriction on gender; 3- with responsible aneurysm in internal carotid artery system; 4- patients signed informed consent.

Corresponding Author: Jinbei Cui, MM; e-mail: cuijinbeibei@163.com

Exclusion criteria: 1- blood flow signals cannot be detected due to personal reasons; 2- patients with posterior circulation aneurysm; 3- patients combined with severe diabetes or other basic disease; 4- patients quitted study or refused to cooperate with the researchers.

Research Methods

Diagnostic methods of CVS: 1- TCD monitoring method: TCD instrument used in this study was from the EME (Neuenhagen, Germany), a continuous-wave Doppler probe with a transmission frequency of 2.4 MHz was used. Detailed steps are as follows: TCD was performed within 24 h after admission. Middle cerebral artery (MCA), anterior cerebral artery (ACA), and basilar artery (BA), were detected by the probe through bilateral transtemporal window and foramen magnum window. Sampling site was 4.0 cm-9.0 cm, and the launch area was 2.0 cm^2. The lateral resolution of sampling size was 1.5 cm, and the vertical resolution was 1.0 cm. Under smooth breathing, the detection time was no less than 1 min to detect changes in blood flow velocity. The focus is on the blood flow velocity of MCA, and the blood flow velocities of vertebral artery (VA), ACA and BA. CVS was diagnosed when blood flow velocity of MCA was beyond 120 cm/s.

DSA detection method: The digital subtraction angiography (DSA) instrument was from Siemens (München, Germany). The procedure was as follows: all patients underwent head CT examination to confirm SAH, and DSA was performed within 72 h after admission. In this study, patients with SAH caused by intracranial aneurysm rupture were subjected to DSA examination, followed by internal carotid artery or bilateral vertebral artery angiography to fully understand the patient's cerebrovascular changes. According to the standard of DSA in determining cerebral artery spasm, CVS was divided into diffuse CVS, peripheral CVS, focal CVS and multifocal CVS.

Grading of the Severity of CVS

CVS in patients with SAH was graded according to the mean blood flow velocity of MCA: no vasospasm, mean blood flow velocity of MCA was lower than 120 cm/s; mild vasospasm, mean blood flow velocity of MCA was higher than 120 cm/s but lower than 140 cm/s, and the degree of vascular stenosis was less than 25%; moderate vasospasm, mean blood flow velocity of MCA was higher than 140 cm/s but lower than 200 cm/s, and the degree of vascular stenosis was more than 25% but less than 50%; severe vasospasm, mean blood flow velocity of MCA was higher than 200 cm/s, and the degree of vascular stenosis was more than 50%.

Improved-Fisher Grading Standards[10]

Grade 0, no bleeding or only intraventricular hemorrhage or intracranial hemorrhage; grade 1, only basal cistern hemorrhage; grade 2, periphery cistern or Sylvian cistern hemorrhage; grade 3, intraventricular hematoma; grade 4, hematomyelia in basal cistern, periphery cistern or Sylvian cistern.

Hunt-Hess Grading Standards[11]

I, asymptomatic/mild headache and neck stiffness; II, severe headache, neck stiffness and oculomotor palsy; III, mild disturbance of consciousness, lethargy, restlessness and mild brain symptoms; IV, hemiplegia, coma, early stage of decerebrate rigidity, and autonomic nerve dysfunction; V, deep coma, decerebrate rigidity, endangered state.

Statistical Analysis

SPSS20.0 statistical analysis software (IBM, Armonk, NY, USA) was used to process the data. Measurement data were expressed as mean ± standard deviation (±s), and comparisons among multiple groups were performed using single-factor analysis of variance or repeated measures analysis of variance, followed by Least Significant Difference (LSD) as its post-hoc test. Count data were expressed as percentage (%), and the comparisons between groups were performed using χ^2-analysis. $p < 0.05$ was considered to be statistically significant.

Results

Comparison of the Prevalence of CVS Determined by two Methods

DSA test identified CVS in 79 out of 90 SAH patients and the prevalence was 87.78%. TCD test identified CVS in 75 out of 90 SAH patients and the prevalence was 83.33%. Although the prevalence detected by TCD was lower than that detected by DSA, no significant difference was found between them ($p > 0.05$) (Table I; notes: all CVS data were from TCD test).

Table I. Comparison of the prevalence of CVS determined by two methods.

Methods	Cases (n)	Non-CVS (n)	CVS (n)	Prevalence (%)
DSA	90	15	75	83.33
TCD	90	11	79	87.78
χ^2	–		1.035	
p	–		> 0.05	

Effects of Age and Gender on Prevalence of CVS

In this study, 90 patients with SAH were included: 48 were males and 42 were females; 39 patients were between 30-49 years old, and 51 patients were between 50-70 years old. TCD test results showed that the prevalence of CVS was 81.25% in male patients and 85.71% in female patients. No significant difference in the prevalence of CVS was found between patients with different ages and genders ($p > 0.05$) (Table II).

Effects of Smoking, Alcoholism and Hypertension on the Prevalence of CVS

In this work, history of smoking, alcoholism and hypertension were found in 46 cases, 35 cases and 38 cases of 90 patients with SAH, respectively. TCD test results showed that smoking, alcoholism and hypertension had no significant effects on the prevalence of CVS ($p > 0.05$) (Table III).

Changes in Blood Flow Velocity in Different Time Periods

Blood flow velocity monitoring showed that the blood flow velocities of MCA, ACA, BA and VA increased gradually after the occurence of SAH, and blood flow velocities at 4-6 days after the prevalence were significantly higher than those at 1-3 days after the occurrence, and blood flow velocities reached the peaks at 7-9 days after the prevalence, and decreased at 10-12 days after the prevalence. Significant differences in blood flow velocities of MCA, ACA, BA and VA were found between different time periods ($p < 0.05$) (Table IV).

The Relationship Between the Improved-Fisher Grades and the Occurrence of CVS

Improved-Fisher grading results showed that 15, 30, 26 and 19 patients were graded as 1, 2, 3 and 4, respectively. Patients with grade 1 and 2 were mostly found without spasm or with mild spasm, while patients with grade 3 and 4 were mostly found with moderate and severe spasm. Significant differences were found in patients with different improved-Fisher grades ($p < 0.05$) (Table V).

Table II. Effects of age and gender on prevalence of CVS.

Items		Cases (n)	CVS (n, %)	χ^2	*p*
Gender	Male	48	39 (81.25)	1.237	> 0.05
	Female	42	36 (85.71)		
Age (years)	30-49	39	33 (84.62)	1.505	> 0.05
	50-70	51	42 (82.35)		

Table III. Effects of smoking, alcoholism and hypertension on the prevalence of CVS.

Items		Cases (n)	CVS (n, %)	χ^2	*p*
Smoking	Yes	46	38 (82.61)	2.115	> 0.05
	No	44	37 (84.09)		
Alcoholism	Yes	35	29 (82.86)	1.034	> 0.05
	No	55	46 (83.64)		
Hypertension	Yes	38	31 (81.58)	1.136	> 0.05
	No	52	44 (84.62)		

Table IV. Changes in blood flow velocity in different time periods.

Time periods	MCA (cm/s)	ACA (cm/s)	BA (cm/s)	VA (cm/s)
1-3d	144.67 ± 15.34	112.61 ± 10.28	69.58 ± 7.46	63.05 ± 5.39
4-6d	178.69 ± 12.89*	130.56 ± 9.77*	76.43 ± 8.12*	70.15 ± 5.97*
7-9 d	200.24 ± 11.48*	144.29 ± 10.36*	85.27 ± 7.93*	77.19 ± 6.37*
10-12 d	106.48 ± 12.38*	83.63 ± 10.71*	50.86 ± 6.25*	55.70 ± 5.11*
F-value	10.335	11.276	18.169	10.667
p	< 0.05	< 0.05	< 0.05	< 0.05

Notes: *Compared with 1-3 days after the prevalence, $p < 0.05$.

Table V. The relationship between the improved-Fisher grades and the prevalence of CVS.

Grades	Cases (n)	CVS (n, %)			
		Non-spasm	Mild spasm	Moderate spasm	Severe spasm
Grade 1	15	7 (46.67)	4 (26.67)	2 (13.33)	2 (13.33)
Grade 2	30	4 (13.33)	15 (50.00)	6 (20.00)	5 (16.67)
Grade 3	26	3 (11.54)	6 (23.08)	10 (38.46)	7 (26.92)
Grade 4	19	1 (5.26)	2 (10.53)	6 (31.58)	10 (52.63)
F-value		16.251	12.695	15.027	11.329
p		< 0.05	< 0.05	< 0.05	< 0.05

The Relationship Between the Improved-Fisher Grades and Blood Flow Velocity of MCA

Blood flow velocity of MCA was 46.15 ± 15.22 cm/s in patients with grade 1. Along with the increase in improved-Fisher grades, the blood flow velocity of MCA was gradually increased, and blood flow velocity of patients with grade 4 reached 175.74 ± 12.19 cm/s. Significant differences in blood flow velocity of MCA were found between patients with different improved-Fisher grades ($p < 0.05$) (Table VI).

The Relationship Between Hunt-Hess Grades and the Occurrence of CVS

Hunt-Hess grading results showed that 14, 25, 20, 16 and 15 patients were classified as grade I, II, III, IV and V, respectively. Patients with grade I were usually found without spasm or with mild spasm, patients with grade II were usually found with mild or moderate spasm, and patients with grade III-V were usually found with moderate or severe spasm. Significant differences in the severity of spasm were found between patients with different Hunt-Hess grades ($p < 0.05$) (Table VII).

The Relationship Between Hunt-Hess Grades and Blood Flow Velocity of MCA

Blood flow velocity of MCA was 145.67 ± 15.16 cm/s in patients with Hunt-Hess grade I. Along with the increase in Hunt-Hess grades, the blood flow velocity of MCA was gradually increased, and blood flow velocity in patients with Hunt-Hess grade V reached 183.85 ± 16.17 cm/s. Significant differences in blood flow velocity of MCA were found between patients with different 183.85 ± 16.17 grades ($p < 0.05$) (Table VIII).

Table VI. The relationship between the improved-Fisher grades and blood flow velocity. of MCA.

Grades	Cases (n)	MCA (cm/s)
Grade 1	15	146.15 ± 15.22
Grade 2	30	151.49 ± 14.78
Grade 3	26	165.33 ± 10.67
Grade 4	19	175.74 ± 12.19
F-value	–	10.269
p	–	< 0.05

Table VII. The relationship between the improved-Fisher grades and the prevalence of CVS.

Grades	Cases (*n*)	CVS (*n*, %)			
		Non-spasm	Mild spasm	Moderate spasm	Severe spasm
Grade I	14	7 (50.00)	4 (28.57)	2 (14.29)	1 (7.14)
Grade II	25	3 (12.00)	10 (40.00)	11 (44.00)	1 (4.00)
Grade III	20	2 (10.00)	7 (35.00)	5 (25.00)	6 (30.00)
Grade IV	16	2 (12.50)	4 (25.00)	2 (12.50)	8 (50.00)
Grade V	15	1 (6.67)	2 (13.33)	4 (26.67)	8 (53.33)
F-value	–	10.205	12.601	12.928	11.527
p	–	< 0.05	< 0.05	< 0.05	< 0.05

Discussion

As one of the severe complications of SAH, the prevalence and mortality rate of CVS are both relatively high[12]. Some neurosurgical specialists interpret CVS as “persistent contraction of the intracranial artery”, while other researchers believed CVS was the delayed stenosis of brain aorta, which was usually combined with the decrease in the infusion of the distal region of the affected vessel[13-15]. Clinical symptoms and results of cerebral angiography of the patients are the main diagnostic basis for CVS. At present, the golden standard for the diagnosis of CVS is DSA. However, this approach is an invasive examination and cannot be performed repeatedly on patients[16,17]. With the further development of ultrasound imaging technology, TCD, as a novel and noninvasive inspection technology, has been gradually developed. This method can be used to dynamically monitor the intracranial blood flow in patients, and predict the occurrence and development of CVS before the emergence of clinical symptoms. In recent years, TCD has been widely used in the diagnosis of CVS after SAH[18]. The advantages of TCD include easy operation, high safety, noninvasive nature, low-cost and high repeatability. It is noteworthy that DSA is still the golden standard for SAH and CVS induced by SAH[19,20]. DSA test showed that the prevalence of CVS in 90 patients was 87.78%, while TCD test showed that the prevalence was 83.33%. No significant difference was found between them, indicating the high reliability of TCD in the diagnosis of CVS.

Table VIII. The relationship between Hunt-Hess grades and blood flow velocity of MCA.

Grades	Cases (*n*)	MCA (cm/s)
Grade I	14	145.67 ± 15.16
Grade II	25	150.78 ± 12.33
Grade III	20	164.59 ± 13.72
Grade IV	16	170.06 ± 14.20
Grade V	15	183.85 ± 16.17
F-value	–	15.148
p	–	< 0.05

Results of TCD test showed that the gender, age, smoking, alcoholism, and hypertension had no significant correlation with the prevalence of CVS ($p > 0.05$). Hemodynamic tests showed that blood flow velocities of MCA, ACA and BA and VA were gradually increased after SAH; the blood flow velocities at 4-6 days after SAH were significantly higher than those at 1-3 days after SAH; the peaks were reached at 7-9 days after SAH, followed by a decrease at 10-12 days after SAH. Those data indicate a rule in the changes of blood flow velocity. It can be concluded that TCD can be used to determine the changes of cerebral vascular hemodynamics in patients with SAH, and thus play a role in the diagnosis of CVS after SAH. The results provided by TCD can be used as important basis for follow-up treatment.

There are a variety of clinical grading methods for patients with SAH, and those grading methods are usually based on meningeal irritation symptoms, headache, neurological status and state of consciousness[21]. Clinical grading is extremely important for the evaluation of surgical risk and medical outcomes. Hunt-Hess grading is the most commonly used method in the clinical practice[22,23]. Results of this study showed that there were significant differences in severity of disease between patients with different Hunt-Hess grades ($p < 0.05$). Those data suggest that poor neurological function of patients with SAH can increase the prevalence of primary and secondary brain damages after intracranial aneurysm ruptures. In addition, cerebrovascular self-regulation will be seriously affected, eventually leading to the occurrence of CVS through a series of complex pathophysiological mechanisms. After SAH, erythrocytes and blood disintegration products in subarachnoid can accelerate the prevalence of CVS[24]. It's generally believed that bleeding can affect the development of CVS in patients with SAH. At present, Fisher grading method is mostly used in the grading of bleeding of patients with SAH[25,26]. In this investigation, there were significant differences in the severity of CVS between patients with different improved-Fisher grades ($p < 0.05$), and the prevalence of CVS after SAH was significantly increased with the increase in improved-Fisher grades ($p < 0.05$). The possible explanation is that the production of endothelin and other vasoconstrictor substances after SAH can stimulate the blood vessels, which in turn lead to the occurrence of vasospasm[27]. TCD and CT examinations are needed to be performed immediately after the occurrence of SAH. The combination of CT scanning results and the changes in cerebral blood flow velocity can be used to guide clinical practices. Proper treatment should be performed for patients with severe subarachnoid hemorrhage to delay the development of CVS and reduce the risk of the prevalence of other complications.

Conclusions

We observed that TCD is a novel and noninvasive technique that can be used to monitor CVS. TCD can be used to monitor blood flow velocity of patients with SAH, so as to determine the spasm of the affected cerebral blood vessels and can be used for the prediction and diagnosis of CVS after SAH.

Conflict of Interest
The Authors declare that they have no conflict of interests.

References

1) Moftakhar P, Cooke DL, Fullerton HJ, Ko NU, Amans MR, Narvid JA, Dowd CF, Higashida RT, Halbach VV, Hetts SW. Extent of collateralization predicting symptomatic cerebral vasospasm among pediatric patients: correlations among angiography, transcranial doppler ultrasonography, and clinical findings. J Neurosurg Pediatr 2015; 15: 282-290.
2) Neulen A, Prokesch E, Stein M, Konig J, Giese A. Image-guided transcranial Doppler sonography for monitoring of vasospasm after subarachnoid hemorrhage. Clin Neurol Neurosurg 2016; 145: 14-18.
3) Malhotra K, Conners JJ, Lee VH, Prabhakaran S. Relative changes in transcranial doppler velocities are inferior to absolute thresholds in prediction of symptomatic vasospasm after subarachnoid hemorrhage. J Stroke Cerebrovasc Dis 2014; 23: 31-36.
4) Wachter D, Hans F, Kreitschmann-Andermahr I, Rohde V. Lower occurrence of transcranial doppler and symptomatic vasospasm after aneurysmal subarachnoid hemorrhage and aneurysm clipping in the elderly patient? Neurosurgery 2011; 69: 261-267.
5) Macdonald RL. Delayed neurological deterioration after subarachnoid haemorrhage. Nat Rev Neurol 2014; 10: 44-58.
6) Cossu G, Messerer M, Stocchetti N, Levivier M, Daniel RT, Oddo M. Intracranial pressure and outcome in critically ill patients with aneurysmal subarachnoid hemorrhage: a systematic review. Minerva Anestesiol 2016; 82: 684-696.
7) Spruijt B, Tasker RC, Driessen C, Lequin MH, van Veelen ML, Mathijssen IM and Joosten KF. Abnormal transcranial Doppler cerebral blood flow velocity and blood pressure profiles in children with syndromic craniosynostosis and papilledema. J Craniomaxillofac Surg 44: 465-470, 2016.
8) Kantelhardt SR, Greke C, Keric N, Vollmer F, Thiemann I, Giese A. Image guidance for transcranial doppler ultrasonography. Neurosurgery 2011; 68: 257-266.
9) Toscano M, Vicenzini E, Di Piero V. Comments on 'hemodynamic features of non-aneurysmal subarachnoid hemorrhage in a case of familial moyamoya disease' after a 4-year transcranial doppler ultrasound follow-up. Eur Neurol 2016; 75: 149.
10) Zhu RL, Chen ZJ, Li S, Lu XC, Tang LJ, Huang BS, Yu W, Wang X, Qian TD, Li LX. Statin-treated patients with aneurysmal subarachnoid haemorrhage: a meta-analysis. Eur Rev Med Pharmacol Sci 2016; 20: 2090-2098.
11) Huang Q, Wang G, Hu YL, Liu JX, Yang J, Wang S, Zhang HB. Study on the expression and mechanism of inflammatory factors in the brain of rats with cerebral vasospasm. Eur Rev Med Pharmacol Sci 2017; 21: 2887-2894.
12) Djelilovic-Vranic J, Basic-Kes V, Tiric-Campara M, Djozic E, Kulenovic J. Follow-up of vasospasm by transcranial doppler sonography (TCD) in subarachnoid hemorrhage (SAH). Acta Inform Med 2017; 25: 14-18.
13) Toscano M, Puledda F, Vigano A,Vicenzini E, Guidetti G, Lenzi GL, Di Piero V. Hemodynamic features of

non-aneurysmal subarachnoid hemorrhage in a case of familial moyamoya disease: a transcranial doppler ultrasound study. Eur Neurol 2014; 72: 330-332.

14) Ehrlich G, Kirschning T, Wenz H, Hegewald AA, Groden C, Schmiedek P, Scharf J, Seiz-Rosenhagen M. Is there an influence of routine daily transcranial doppler examination on clinical outcome in patients after aneurysmal subarachnoid hemorrhage? World Neurosurg 2016; 88: 214-221.

15) Barth M, Pena P, Seiz M, Thomé C, Muench E, Weidauer S, Hattingen E, Kasuya H, Schmiedek P. Feasibility of intraventricular nicardipine prolonged release implants in patients following aneurysmal subarachnoid haemorrhage. Br J Neurosurg 2011; 25: 677-683.

16) Etminan N, Macdonald RL, Davis C, Burton K, Steiger HJ, Hanggi D. Intrathecal application of the nimodipine slow-release microparticle system eg-1962 for prevention of delayed cerebral ischemia and improvement of outcome after aneurysmal subarachnoid hemorrhage. Acta Neurochir Suppl 2015; 120: 281-286.

17) Dehdashti AR, Binaghi S, Uske A, Regli L. Intraarterial nimodipine for the treatment of symptomatic vasospasm after aneurysmal subarachnoid hemorrhage: a preliminary study. Neurol India 2011; 59: 810-816.

18) Greke C, Neulen A, Kantelhardt SR, Birkenmayer A, Vollmer FC, Thiemann I, Giese A. Image-guided transcranial Doppler sonography for monitoring of defined segments of intracranial arteries. J Neurosurg Anesthesiol 2013; 25: 55-61.

19) Ulrich CT, Fung C, Vatter H, Setzer M, Gueresir E, Seifert V, Beck J, Raabe A. Occurrence of vasospasm and infarction in relation to a focal monitoring sensor in patients after SAH: placing a bet when placing a probe? PLoS One 2013; 8: e62754.

20) Kumar G, Shahripour RB, Harrigan MR. Vasospasm on transcranial doppler is predictive of delayed cerebral ischemia in aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis. J Neurosurg 2016; 124: 1257-1264.

21) Paschoal FJ, de Almeida LRK, de Lima OM,Nogueira Rde C, Paschoal EH, Teixeira MJ, Figueiredo EG, Bor-Seng-Shu E. Embolic signals during routine transcranial Doppler ultrasonography in aneurysmal subarachnoid hemorrhage. Biomed Res Int 2015; 2015: 153714.

22) Walcott BP, Stapleton CJ, Koch MJ, Ogilvy CS. Diffuse patterns of nonaneurysmal subarachnoid hemorrhage originating from the basal cisterns have predictable vasospasm rates similar to aneurysmal subarachnoid hemorrhage. J Stroke Cerebrovasc Dis 2015; 24: 795-801.

23) Marshall SA, Nyquist P, Ziai WC. The role of transcranial doppler ultrasonography in the diagnosis and management of vasospasm after aneurysmal subarachnoid hemorrhage. Neurosurg Clin N Am 2010; 21: 291-303.

24) Ding W, Gao N, Li MX, Ding LJ, Li FF, Mou L. Clinical evaluation of the efficacy of the combination of aneurysm embolization and cerebrospinal fluid replacement in the treatment of aneurysmal subarachnoid hemorrhage. Eur Rev Med Pharmacol Sci 2015; 19: 402-405.

25) Wilson DA, Nakaji P, Abla AA,Uschold TD, Fusco DJ, Oppenlander ME, Albuquerque FC, McDougall CG, Zabramski JM, Spetzler RF. A simple and quantitative method to predict symptomatic vasospasm after subarachnoid hemorrhage based on computed tomography: beyond the Fisher scale. Neurosurgery 2012; 71: 869-875.

26) Perrein A, Petry L, Reis A, Baumann A, Mertes P, Audibert G. Cerebral vasospasm after traumatic brain injury: an update. Minerva Anestesiol 2015; 81: 1219-1228.

27) Foreman PM, Starke RM, Hendrix P, Harrigan MR, Fisher WS Rd, Vyas NA, Lipsky RH, Lin M, Walters BC, Pittet JF, Mathru M, Griessenauer CJ. Endothelin polymorphisms as a risk factor for cerebral aneurysm rebleeding following aneurysmal subarachnoid hemorrhage. Clin Neurol Neurosurg 2017; 157: 65-69.