Cerebral Angiographic Findings in Patients with Non-Traumatic Subarachnoid Hemorrhage

Amnat Kitkhuandee MD*,
Jureerat Thammaroj MD**, Waranon Munkong MD**,
Pichayen Duangthongpon MD*, Chaiwit Thanapaisal MD*

** Division of Neurosurgery, Department of Surgery, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand*
*** Department of Radiology, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand*

Background: *Even though ruptured intracranial aneurysm is a major cause of non-traumatic subarachnoid hemorrhage (SAH), non-aneurysmal SAH has a good prognosis with few neurologic complications. The gold standard for detecting the vascular pathology is digital subtraction cerebral angiography (DSA).*
Objective: *The primary objective of the present study was to clarify cerebral angiographic findings in patients with non-traumatic subarachnoid hemorrhage (SAH); to define the incidence of nonaneurysmal SAH. The secondary aim was to review the clinical data of all of the patients diagnosed with non-traumatic SAH in order to determine the associated etiology.*
Material and Method: *This retrospective, descriptive study, was conducted at Srinagarind Hospital, Faculty of Medicine, Khon Kaen University, between January 2005 and November 2010. The authors reviewed the medical records, cranial computed tomography (CT) and DSA of patients with non-traumatic SAH. The DSA findings were assessed and the correlation with clinical data and CT pattern determined.*
Results: *The author included and analyzed the records of 118 non-traumatic SAH patients (66 females, 52 males). The DSA discovered vascular lesions in 62.6% of cases (57.6% aneurysm, 4.2% arteriovenous malformation (AVM) and 0.8% Moyamoya disease). A total of 76 aneurysms were found in 68 cases. The location of the aneurysms included: 35.5% anterior communicating artery, 17.1% posterior communicating artery, 15.7% middle cerebral artery, 11.8% internal carotid artery, 2.6% basilar artery, 1.3% vertebrobasillar junction and 10.5% others. The prevalence of nonaneurysmal SAH was 42.4% (50/118). In the multivariate analysis, hypertension was the factor most strongly associated with aneurysmal SAH on the DSA (p = 0.029). The location of SAH on Sylvian fissure was most frequently associated with the cause of aneurysms. In another way, tentorial cerebelli SAH was most commonly associated with a non-aneurysm cause.*
Conclusion: *The present study revealed that the major cause of non-traumatic SAH is cerebral aneurysm. The factors associated with aneurysmal SAH included: hypertension and Sylvian fissure SAH. Tentorial cerebelli SAH was most commonly associated with a non-aneurysm cause.*

Keywords: *Cerebral angiographic finding, Non-traumatic subarachnoid hemorrhage, Associated factors*

J Med Assoc Thai 2012; 95 (Suppl. 11): S121-S129
Full text. e-Journal: *http://jmat.mat.or.th*

Non-traumatic subarachnoid hemorrhage (SAH) is a neurologic emergency characterized by the extravasation of blood into the space surrounding the central nervous system, which is usually filled with cerebrospinal fluid. The major determinant of non-traumatic SAH is rupturing of an intracra-nial aneurysm (80% of cases), which has a high rate of death and complications[1]. A major cause for a poor outcome among patients with aneurysmal SAH is re-bleeding, resulting in a mortality rate of 80%[2]. Aneurysmal re-bleeding occurs in 4% of patients within 24 hours, 20% within 2 weeks and 50% within 6 months after the initial occurrence[3]. Non-aneurysmal SAH including isolated perimesencephalic subarachnoid hemorrhage (20% of cases) has a good prognosis with some uncommon neurologic complications[4]. The diagnosis of non-traumatic SAH requires the physician to ascertain its etiology, since the cause of SAH guides its management[5].

The global incidence of non-traumatic SAH varies from country to country; ~10.5 cases per 100,000 per year[1]. Population-based incidence rates for SAH vary between 6.5 and 23.9 per 100,000 for all age groups[6,7], but this increases with age. The mean age

Correspondence to:
Kitkhuandee A, Division of Neurosurgery, Department of Surgery, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand.
Phone: 043-363-252
E-mail: amnat811@yahoo.com

is ~50 years and the greatest frequency is in persons over 35 years. It is higher in women than in men[8]. The incidence of aneurysmal SAH is between 9.3 and 10.8 per 100.000. The value is slightly lower in the USA and Canada, between 6 and 7 per 100.000[9]. The incidence of SAH in the Middle East, Africa and some Far Eastern countries is low and discriminative studies undertaken in Malaysia, Singapore, Hong Kong, Nigeria, India, the Philippines and Thailand seem to confirm a low incidence of cerebral aneurysms in these countries. Not with standing, under developed medical knowledge and lack of diagnostic equipment in these countries probably mean that most patients go undiagnosed[4].

In ~15% of patients with spontaneous SAH, the uncertain source of hemorrhage can be visualized using cerebral angiography. The clinical course of these patients has been reported as benign compared with patients with aneurysmal SAH[5]. At the authors' tertiary institution in Northeast Thailand, the authors found that the leading cause of patients with non-traumatic SAH is infrequently due to a ruptured intracranial aneurysm; thus, the authors reviewed all of the authors' cerebral angiographic findings in patients with non-traumatic SAH to determine the incidence of non-aneurysmal SAH. Furthermore, the authors studied the clinical data of all patients diagnosed with non-traumatic SAH to catalogue the associated etiologies.

Material and Method

Subjects

This was a descriptive, single-center, retrospective study of patients with non-traumatic subarachnoid hemorrhage (SAH); admitted to Srinagarind Hospital, Faculty of Medicine, Khon Kaen University, between January 2005 and November 2010. As per the Helsinki Declaration, the confidentiality of the patients was protected and the experimental design and protocol reviewed and approved by the Khon Kaen University Ethics Committee for Human Research. The authors reviewed the medical records, CT and DSA images of consecutive, non-traumatic SAH patients admitted for DSA in order to diagnose SAH.

The inclusion criteria for the present study were: 1) Age > 15 years; and 2) SAH verified by cranial CT. The exclusion criteria were: 1) Age < 15 years; 2) Traumatic origin; 3) Hemorrhaging into a tumor; 4) Hemorrhage considered to be from bloody dyscrasia or abnormal coagulopathy; 5) Unavailable DSA images. A total of 418 patients were admitted to the department of neurosurgery with non-traumatic SAH identified by CT. After reviewing the cerebral angiography, there were 118 non-traumatic SAH patients enrolled in the present study.

The demographic data, clinicals, laboratory investigations, hospitalization information were assembled. The demographic data included age and sex. The clinical data included: 1) Underlying disease (*i.e.*, hypertension (HT), diabetic mellitus (DM), prior stroke and/or dyslipidemia); 2) Risk factors (*viz.*, smoking, alcoholic drinking and drug abuse); 3) Clinical signs and symptoms (characteristics as per the Hunt & Hess scale[26]); 4) Initial physical examination (*i.e.*, Initial blood pressure (BP) and the WFNS grading scale[27]). The reviewed laboratory investigations included complete blood count with absolute eosinophil count.

Imaging techniques

CT studies were available for 118 patients, 83 performed at Srinagarind Hospital and the rest from other hospitals. The CT was performed with a 4 detectors device (Zomatom Plus 4, Siemens, Erlangen, Germany) with a 4 mm axial slice thickness in the posterior fossa and 8 mm axial slice thickness in the suprasellar region.

Each patient with a CT or CSF diagnosis of non-traumatic SAH underwent 3-4 vessels selective DSA (DAR-100, Shimadzu, Tokyo, Japan and Allura Xper FD20, Philips, Florida, USA), according to the standard protocol. With the patient supine, the procedure was performed under sedation under a local or general anesthetic. Right transfemoral catheterization was performed using the modified Selding's technique for bilateral carotid and vertebral artery angiography, using the HN5 catheter Fr. 5 with a J-smooth guide wire No. 035 and a Manie catheter Fr. 5 with a J-smooth guide wire No. 038. The cerebral angiogram was obtained in at least two projections of each vessel: anteroposterior view and lateral view. The technique used to obtain the images was 3 frames/sec for 5 sec and 1 frame/sec for 10 sec.

Imaging analysis

Two experienced neuroradiologists reviewed the CT and DSA images independently and achieved a determination on the definite interpretations by consensus. One neuroradiologist had 13 years experience and the other 3 years. Two-thirds of all of the images were reviewed on CT films or DSA films, and one-third on a PAC workstation.

The CT images were analyzed first apart from the DSA images. The CT images were assessed for the presence of subarachnoid hemorrhage, its location and

hydrocephalus. The appearance of hemorrhage is classified using the Fisher's grade[15]: grade 1 = no evident hemorrhage, grade 2 = subarachnoid hemorrhage < 1 mm thickness, grade 3 = subarachnoid hemorrhage > 1 mm thickness and grade 4 = subarachnoid hemorrhage of any thickness with intraventricular hemorrhage (IVH) or parenchymal extension.

SAH was defined as a high-attenuating, amorphous substance that fills the normally dark, CSF-filled subarachnoid spaces around the brain. The following sites were defined: ambient cistern, peri-/pre-pontine cistern, interpeduncular cistern, perimesencephalic (pontine, interpeduncular and ambient cisterns) cistern, quadrigeminal cistern, Sylvian fissure, anterior and posterior interhemispheric fissure, basal cistern, and others. Hydrocephalus is defined as *disproportional dilatation of the ventricular system.*

The cerebral angiogram images were assessed for the presence of any cerebral angiographic abnormality. Giant aneurysm was defined as an aneurysm with a maximum diameter exceeding 2.5 cm. The location of the aneurysm could be: ICA = internal carotid artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; AcoA = anterior communicating artery; PcoA = posterior communicating artery; PCA = posterior cerebral artery; BA = basillar artery: and PICA = posterior inferior cerebellar artery.

Arteriovenous malformation (AVM) was defined as a tightly packed mass of enlarged feeding arteries and dilated, tortuous draining veins with little or no intervening brain parenchyma within a central nidus. Arteriovenous shunting was defined as contrast material appears in draining veins abnormally early in the angiographic sequence. Intracranial vasculitis was defined as multiple short stenoses and constriction of vessels, with vascular dilation alternating with an area of non-visualized vessels.

Vascular spasm was defined as arterial narrowing.

Statistical analysis

Data analyses were performed using SPSS version 10 for Windows. The categorical data (demographics, clinicals and angiographic findings) were analyzed for frequency and percentages while the continuous data were assessed for mean, median and standard deviation.

The prevalence of non-aneurysmal SAH was also defined. The proportion test was used to compare the proportions of the two groups. The t-test and Chi-square test were used to compare averages ($p \leq 0.05$).

Results

Of the 418 non-traumatic SAH patients admitted to Srinagarind Hospital during the study, 300 were excluded because of incomplete data leaving 118 cases enrolled for analysis. Almost all of the subjects lived in Northeast Thailand. The demographic data including sex, age, comorbidities (HT, DM, history of CVA or stroke and dyslipidemia) and risk factors (smoking, alcoholic drinking and drug abuse) are presented in Table 1. The clinical data including onset of SAH, the Hunt and Hess grading system, WFNS grading scale and BP at presentation are presented in Table 2. The associated laboratory investigation including white blood cell and absolute eosinophil count are presented in Table 3. The location of SAH on the CT are pointed out in Table 4, and the associated CT appearance including intraparenchymal hemorrhage (IPH), hydrocephalus, intraventricular hemorrhage (IVH) and the Fisher's grade are presented in Table 5. Of the 118 cases diagnosed as non-traumatic SAH by CT, 68 had cerebral aneurysm detected by DSA with a total of 76 aneurysms, 35 of which were ruptured. The anterior communicating artery aneurysm was intact in 27 patients. The locations of the aneurysms are presented in Table 6.

The prevalence of non-aneurysmal SAH was 42.6%. A normal angiogram was found in 31 (26.3%) of all 118 cases. AVM was found in five patients, four males and one female (average age, 63 years). The mean age of the males was 49.6 years. One male (71 years) had Moya-moya disease, presenting as Fisher's grade IV of SAH. The AVM and aneurysm ratio was 1: 7.5 and the female: male ratio was 1: 0.25. One female patient also had a ruptured left posterior communicating artery aneurysm and combined AVM. The patterns of additional vascular abnormalities of DSA between the aneurysmal and non-aneurysmal SAH are shown in Table 7.

The distribution of demographic data was similar between the aneurysmal and non-aneurysmal subgroups, except for the mean age of females (0.0190) and history of hypertension ($p = 0.0299$) (Table 1). The clinical variables and laboratory investigations; associated with aneurysmal SAH are presented in Table 2 and 3.

The location of SAH at the Sylvian fissure was the factor associated with the cause aneurysm; while the tentorial cerebelli SAH was associated with

Table 1. Demographic data

Characteristics	Number (%)	Aneurysm (n = 68)	Non-aneurysm (n = 50)	p-value
Sex				
Male	52 (44.1)	25 (36.8)	27 (54.0)	0.0624
Female	66 (55.9)	42 (63.2)	23 (46.0)	0.0624
Mean age ± SD (years)	50.71 ± 13.60	52.35 ± 12.96	48.48 ± 14.26	0.1273
(range)	(15-80)	(20-80)	(15-75)	
Male	47.12 ± 13.35	46.48 ± 11.19	47.37 ± 15.30	0.7157
Female	53.55 ± 13.22	55.56 ± 12.95	49.78 ± 13.16	0.019
Comorbidities				
Hypertension	42 (36.5)	30 (44.8)	12 (25.0)	0.0299
Diabetes mellitus	8 (7.0)	6 (9.0)	2 (4.2)	0.3196
History of CVA or stroke	6 (5.2)	4 (6.0)	2 (4.2)	0.668
Dyslipidemia	1 (7.0)	5 (7.5)	3 (6.3)	0.801
Risk factor				
History of smoking	10 (8.7)	8 (11.9)	2 (4.2)	0.1446
Alcoholic drinking	8 (7.0)	7 (10.4)	1 (2.1)	0.0821
Drug abuse	0	0	0	-

Table 2. The Hunt & Hess grading system, WFNS scale and Blood pressure

Variables	Number (%) (n = 68)	Aneurysm (n = 50)	Non-aneurysm	p-value
Onset of SAH (days)				
0-3	57 (49.6)	32 (47.8)	25 (52.1)	0.6476
3-7	29 (25.2)	19 (28.4)	10 (20.8)	0.3595
7-14	14 (12.2)	7 (10.4)	7 (14.6)	0.5036
>14	15 (13.0)	9 (13.4)	6 (12.5)	0.8835
Hunt & Hess scale				
Grade 1	8 (7.0)	5 (7.5)	3 (6.3)	0.801
Grade 2	79 (68.7)	42 (62.7)	37 (77.1)	0.1006
Grade 3	24 (20.9)	17 (25.4)	7 (14.6)	0.1603
Grade 4	4 (3.5)	3 (4.5)	1 (2.1)	0.4896
Grade 5	0	0	0	
Blood pressure at presentation				
Systolic	142 ± 26 (90-219)	144 ± 27	139 ± 25	0.3568
Diastolic	81 ± 17 (10-121)	82 ± 17	79 ± 16	0.3098
WFNS scale at presentation				
Grade 1	68 (59.6)	35 (53.0)	33 (68.8)	0.0912
Grade 2	23 (20.2)	14 (21.2)	9 (18.8)	0.7464
Grade 3	7 (6.1)	5 (7.6)	2 (4.2)	0.4541
Grade 4	13 (11.4)	10 (15.2)	3 (6.3)	0.1399
Grade 5	3 (2.6)	2 (3.0)	1 (2.1)	0.7551

non-aneurysmal causes (Table 4).

Discussion

SAH is an acute and potentially life-threatening condition. The population incidence rate varies between 6.5 and 23.9 per 100.000 for all age groups[17]. Approximately 10-30% of SAH patients die before getting to medical care. For those reaching a hospital alive, mortality rates for non-traumatic SAH are between 30 and 60%[10]. In-hospital mortality is lower

Table 3. Associated laboratory investigation

Variable	Number (%)	Aneurysm	Non-aneurysm	p-value
White blood cell count	11,768 ± 5,019 (3,700-40,000)	(n = 60)	(n = 46)	
< 4,000 cell/mm^3	1 (0.9)	0 (0.0)	1 (2.2)	0.2512
4,000-12,000 cell/mm^3	62 (58.5)	34 (56.7)	28 (60.9)	0.6634
12,000 cell/mm^3 (leukocytosis)	43 (40.6)	26 (43.3)	17 (37.0)	0.5075
Eosinophils count(cell/uL)	206 ± 415 (0-2,507)	(n = 66)	(n = 50)	
≤ 500	103 (88.8)	59 (89.4)	44 (88.0)	0.8137
> 500	13 (11.2)	7 (10.6)	6 (12.0)	0.8137

Table 4. Location of SAH on CT

Location of SAH on CT	Number (%) 118	Aneurysm (%) 68	Non-aneurysm (%) 50	p-value
Sylvian fissure	73 (61.86)	51 (69.86)	22 (30.14)	0.001
Basal Cistern	53 (44.92)	31 (58.49)	22 (41.51)	0.987
Interpeduncular Cistern, Prepontine Cistern	15 (12.71)	8 (55.33)	7 (46.67)	0.796
Perimesencephalic Cistern	46 (38.98)	25 (54.35)	21 (45.65)	0.7
Ambient Cistern	15 (12.71)	9 (60.00)	6 (40.00)	0.936
Quadrigeminal Cistern	29 (24.58)	18 (62.07)	11 (37.93)	0.733
Cerebral Cortical Sulci	19 (16.10)	7 (36.84)	12 (63.16)	0.08
Anterior Interhemisphreric Fissure	34 (28.81)	22 (64.71)	12 (35.29)	0.433
Posterior Interhemisphreric Fissure	12 (10.17)	4 (33.33)	8 (66.67)	0.137
Tentorial Cerebelli	14 (11.86)	4 (28.57)	10 (71.34)	0.04
Perimedullary Cistern	4 (3.39)	3 (75.00)	1 (25.00)	0.841
Others	9 (7.63)	7 (77.78)	2 (22.22)	

Table 5. Associated CT appearance

CT appearance	Number (%) (118)	Aneurysm (68)	Non-aneurysm (50)	p-value
Intraparenchymal hemorrhage	32 (27.8)	19 (28.4)	11 (23.9)	0.5991
Frontal	16 (50.0)			
Parietal	5 (15.6)			
Temporal	5 (15.6)			
Occipital	3 (9.4)			
Corpus callosum	1 (3.1)			
Ant limb of internal capsule	1 (3.1)			
Pons	1 (3.1)			
Hydrocephalus	26 (22.0)	19 (27.9)	7 (14.0)	0.114
Intraventricular hemorrhage	42 (25.6)	28 (41.2)	14 (28.0)	0.200
Fisher's grade 1	1 (0.8)	1 (1.5)	0 (0.0)	0.3892
Fisher's grade 2	3 (2.5)	1 (1.5)	2 (4.0)	0.3884
Fisher's grade 3	58 (49.2)	32 (47.1)	26 (52.0)	0.5957
Fisher's grade 4	56 (47.5)	34 (50.0)	22 (44.0)	0.5189

because of interventional neuroradiology[11].

Computed tomography (CT) scanning without intravenous contrast administration-is the favored primary diagnostic imaging tool, with cerebral angiography being the next procedure of choice[12]. At most institutions in the USA, conventional angiography

remains the standard for evaluating patients with SAH. If a cranial CT scan is negative and strong clinicals suggest SAH is present, a lumbar puncture may be used to confirm this diagnosis[13]. The present study of cerebral aneurysms has long been the exclusive territory of DSA an invasive procedure with low risk for the patient, but serious complications[13,14].

In previous studies, the mean age of non-aneurysmal SAH ranged between 36 and 64 years[20-24]. There was a significant predominance of males with aneurysmal SAH[20,22,25]. The present study confirmed these reports: as the mean age non-aneurysmal SAH was 48.48 $\pm$ 14.26 and the male to female ratio was 1 to 0.85. The majority of patients generally presented with low clinical grading in the non-aneurysmal group (94% Hunt & Hess grades I and II) compared to a high clinical grade in the aneurysmal group (31% WFNS grade IV-V)[20,22]. By contrast, the authors' non-aneurysmal SAH group presented 83.4% Hunt & Hess grades I and II, while authors' aneurysmal group was 70.2% Hunt&Hess grades I and II and 81.3% WFNS grade I-II.

The true incidence of non-aneurysmal SAH in authors' situation could not be defined due to a number of conducible factors. Consequently, authors' examined its prevalence among hospital admissions, which the authors found was 24.6%. The authors found that of the 57.6% of patient cause by aneurysms The others (42.6%) was AVM (4.2%), Moya-moya (0.8%), normal angiogram (26.3%) and irregularities or narrowing and spasm of vessels (11.9%). This result is lower than previously reported, which range between 41 and 82.5% for aneurysm[16,18,28]; thus, the prevalence of aneurysm (57.6%) is also lower than reported in other studies.

In a series organized by Ammar et al, in which the prevalence of SAH was 8/10,000: 41% of the patients suffered from ruptured aneurysms, 23% from AVM, 15% spontaneous SAH, 2% Moya-moya, 4% bleeding disorders and 2% were neonates and the remaining 17% had a venous malformation. The AVM to aneurysm ratio was 1 to 1.76 and the female to male ratio was 1 to 2.65; which is considerably lower than the global average[16].

The location of aneurysms varies. In a study of 295 patients undergoing cerebral DSA for acute non-traumatic SAH, Duong et al demonstrated that 27.6% were on the AcoA, 20.2% the MCA, 15.6% the PcoA, 12.8% the ICA, 11.3% multiple sites, 5.9% the basilar tip, 5.4% posterior circulation for a total of 203 patients. The remaining 92 patients (31%) were negative on their initial angiogram[22].

Using DSA, Uysal et al found a total of 34 aneurysms in 28 of the 32 cases involved in the study, while no aneurysm was detected in 4 cases. Of the 5 cases that had multiple aneurysms, four had two and one had three. Thirteen aneurysms (38%) were detected at the ACoA, 11 (32%) at the MCA, 6 (17%) at the ICA, 1 (3%) at the ACA, 1 (3%) at the PCA, 1 (3%) at superior cerebellar artery and 1 (3%) at the BA[18].

In present study, 27 (35.5%) of the aneurysms

Table 6. Location of all aneurysms

Site	Total (n = 76)
Anterior cerebral artery	
A-com	27 (35.5)
A1	1 (1.3)
pericallosal	1 (1.3)
Middle cerebral artery	
MCA bifurcation	12 (15.7)
M1	2 (2.6)
Internal carotid	
ICA	9 (11.8)
P-com	13 (17.1)
PCA	1 (1.3)
Basilar	2 (2.6)
Basilar trunk	1 (1.3)
AICA	2 (2.6)
PICA	2 (2.6)
Vertebral artetry	2 (2.6)
Vertebral-basilarr junction	1 (1.3)

Table 7. Additional vascular abnormalities of aneurysmal and non-aneurysmal patients

Variable	Aneurysm (68)	Non Aneurysm (50)	p-value
Spasm	14	10	0.878
Irregularity of Vessel	5	7	0.383
Hypoplastic Artery	8	1	0.104
Occlusion	2	0	0.616
Dissecting Art	1	0	0.877

were on the AcoA, 12 (15.7%) on the MCA, 13 (17.1%) on the PcoA, 9 (11.8%) on the ICA and 2 (2.6%) on the basilar artery, which accords with previous reports.

In the present study, hypertension at presentation was the clinical factor most related to the cause of aneurysm. The location of the SAH at the Sylvian fissure was related to the cause aneurysm as previously reported. The location of SAH was related to the non-aneurysmal cause, perhaps because the tentorial cerebelli area has no major cerebral artery.

Since ours was a retrospective study, it has some inherent limitations. First, the particulars of information depended on the accuracy and consistency of recording patient information by attending clinicians. Second, due to the method of patient selection, the present study may underestimate the reliability of the outcomes by excluding patients without SAH in CT and recording CSF analysis. Notwithstanding, the present study populations were quite large which may offset some of the weaknesses. The present patients were essentially inhabitants of Northeast Thailand; hence the authors' results will be useful when planning the management of the patients in this region and potentially neighboring countries with similar circumstances and ethnicities.

Conclusion

The present study unveiled that the major cause of non-traumatic SAH is cerebral aneurysm. The factors associated with aneurysmal SAH are hypertension and Sylvian fissure SAH. In another way, tentorial cerebelli SAH is associated with non-aneurysmal causes.

Acknowledgement

The authors wish to thank (a) the Center of Cleft Lip-Cleft Palate and Craniofacial Deformities, Khon Kaen University in association with the Tawanchai Project for support and (b) Mr. Bryan Roderick Hamman and Mrs. Janice Loewen-Hamman for assistance with the English-language of the manuscript.

Potential conflicts of interest

None.

References

1. van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. Brain 2001; 124: 249-78.
2. Rosenorn J, Eskesen V, Schmidt K, Ronde F. The risk of rebleeding from ruptured intracranial aneurysms. J Neurosurg 1987; 67: 329-32.
3. Eskesen V, Rosenorn J, Schmidt K. The impact of rebleeding on the life time probabilities of different outcomes in patients with ruptured intracranial aneurysms. A theoretical evaluation. Acta Neurochir (Wien) 1988; 95: 99-101.
4. Adams HP Jr, Gordon DL. Nonaneurysmal subarachnoid hemorrhage. Ann Neurol 1991; 29: 461-2.
5. Friedman AH. Subarachnoid hemorrhage of unknown etiology. In: Wilkins RH, Rengachary SS, editors. Neurosurgery update II. New York: McGraw-Hill; 1990: 73-77.
6. Sivenius J, Heinonen OP, Pyorala K, Salonen J, Riekkinen P. The incidence of stroke in the Kuopio area of East Finland. Stroke 1985; 16: 188-92.
7. Kurtzke JF. The current neurologic burden of illness and injury in the United States. Neurology 1982; 32: 1207-14.
8. Ingall TJ, Wiebers DO. Natural history of subarachnoid hemorrhage. In: Whisnant JP, editor. Stroke, population, cohorts and clinical trials. Boston: Butterworth-Heineman; 1993: 174-86.
9. Ostergaard KM. Increased incidence of bleeding intracranial aneurysms in Greenlandic Eskimos. Acta Neurochir (Wien) 1983; 67: 37-43.
10. Ingall T, Asplund K, Mahonen M, Bonita R. A multinational comparison of subarachnoid hemorrhage epidemiology in the WHO MONICA stroke study. Stroke 2000; 31: 1054-61.
11. Johnston SC. Effect of endovascular services and hospital volume on cerebral aneurysm treatment outcomes. Stroke 2000; 31: 111-7.
12. Bederson JB, Connolly ES Jr, Batjer HH, Dacey RG, Dion JE, Diringer MN, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 2009; 40: 994-1025.
13. Gershon A, Feld RS, Twohig MT. Imaging in subarachnoid hemorrhage. Medscape reference [Internet]. 1994-2012 [cited 2011 May 27]. Available from: http://emedicine.medscape.com/article/344342-overview
14. El Khaldi M, Pernter P, Ferro F, Alfieri A, Decaminada N, Naibo L, et al. Detection of cerebral aneurysms in nontraumatic subarachnoid haemorrhage: role of multislice CT angiography in 130 consecutive patients. Radiol Med 2007; 112: 123-37.

15. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery 1980; 6: 1-9.
16. Ammar A, al Rajeh S, Ibrahim AW, Chowdhary UM, Awada A. Pattern of subarachnoid haemorrhage in Saudi Arabia. Acta Neurochir (Wien) 1992; 114: 16-9.
17. Milenkovic Z, Babic M, Radenkovic S. Nontraumatic (spontaneous) subarachnoid hemorrhage. Medicine and Biology 1998; 5: 12-17.
18. Uysal E, Yanbuloglu B, Erturk M, Kilinc BM, Basak M. Spiral CT angiography in diagnosis of cerebral aneurysms of cases with acute subarachnoid hemorrhage. Diagn Interv Radiol 2005; 11: 77-82.
19. Karttunen AI, Jartti PH, Ukkola VA, Sajanti J, Haapea M. Value of the quantity and distribution of subarachnoid haemorrhage on CT in the localization of a ruptured cerebral aneurysm. Acta Neurochir (Wien) 2003; 145: 655-61.
20. Dupont SA, Lanzino G, Wijdicks EF, Rabinstein AA. The use of clinical and routine imaging data to differentiate between aneurysmal and nonaneurysmal subarachnoid hemorrhage prior to angiography. Clinical article. J Neurosurg 2010; 113: 790-4.
21. Levy LF. Subarachnoid haemorrhage without arteriographic vascular abnormality. J Neurosurg 1960; 17: 252-8.
22. Duong H, Melancon D, Tampieri D, Ethier R. The negative angiogram in subarachnoid haemorrhage. Neuroradiology 1996; 38: 15-9.
23. Barlow P. Incidence of delayed cerebral ischaemia following subarachnoid haemorrhage of unknown cause. J Neurol Neurosurg Psychiatry 1985; 48: 132-6.
24. Shephard RH. Prognosis of spontaneous (non-traumatic) subarachnoid haemorrhage of unknown cause. A personal series 1958-1980. Lancet 1984; 1: 777-9.
25. Cioffi F, Pasqualin A, Cavazzani P, Da Pian R. Subarachnoid haemorrhage of unknown origin: clinical and tomographical aspects. Acta Neurochir (Wien) 1989; 97: 31-9.
26. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968; 28: 14-20.
27. Teasdale GM, Drake CG, Hunt W, Kassell N, Sano K, Pertuiset B, et al. A universal subarachnoid hemorrhage scale: report of a committee of the World Federation of Neurosurgical Societies. J Neurol Neurosurg Psychiatry 1988; 51: 1457.
28. Urbach H, Zentner J, Solymosi L. The need for repeat angiography in subarachnoid haemorrhage. Neuroradiology 1998; 40: 6-10.

ลักษณะภาพถ่ายทางรังสีในผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ที่ไม่ได้เกิดจากบาดเจ็บที่ศีรษะ

อำนาจ กิจควรดี, จุรีรัตน์ ธรรมโรจน์, วรานนท์ มั่นคง, พิชเยนทร์ ดวงทองพล, ไชยวิทย์ ธนไพศาล

ภูมิหลัง: *เลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ในผู้ป่วยที่มิใช่บาดเจ็บที่ศีรษะ มักเกิดจากการแตกของหลอดเลือดสมองโป่งพอง ซึ่งใช้การฉีดสารทึบแสงเส้นเลือดในสมองเพื่อทำการวินิจฉัย การแตกของหลอดเลือดสมองโป่งพองมีอัตราตายและทุพพลภาพสูง แต่ในขณะที่สาเหตุที่ไม่ได้เกิดจากการแตกของหลอดเลือดสมองโป่งพองเป็นไปในทางตรงกันข้าม การพยากรณ์ของโรคในผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์จากสาเหตุอื่นดีกว่าจากการแตกของหลอดเลือดสมองโป่งพอง ดังนั้นลักษณะอาการทางคลินิกและลักษณะภาพถ่ายทางรังสี จึงอาจมีความแตกต่างในสองกลุ่มนี้*

วัตถุประสงค์: *เพื่อศึกษาอุบัติการณ์การแตกของหลอดเลือดสมองโป่งพอง ปัจจัยทางคลินิกและลักษณะทางภาพรังสีที่แตกต่างกันในกลุ่มผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ที่เกิดจากการแตกของหลอดเลือดสมองโป่งพองเปรียบเทียบกับกลุ่มที่ไม่ได้เกิดจากการแตกของหลอดเลือดสมองโป่งพอง*

วัสดุและวิธีการ: *เป็นการศึกษาย้อนหลังจากข้อมูลโรงพยาบาลศรีนครินทร์ มหาวิทยาลัยขอนแก่น ระหว่าง มกราคม พ.ศ. 2548 ถึง พฤศจิกายน พ.ศ. 2553 สืบค้นเวชระเบียน ภาพถ่ายเอกซเรย์คอมพิวเตอร์สมอง และภาพถ่ายการฉีดสาร ทึบแสงหลอดเลือดแดงสมอง โดยรังสีแพทย์สองท่าน อ่านแยกกันอ่านอย่างอิสระ และนำข้อมูลมาวิเคราะห์ร่วมกับข้อมูลทางคลินิก*

ผลการศึกษา: *พบว่าผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ 118 ราย เกิดจากการแตกของหลอดเลือดสมองโป่งพอง 68 ราย (ร้อยละ 57.6) ตำแหน่งของหลอดเลือดสมองโป่งพองส่วนใหญ่พบที่ เส้นเลือดแดง anterior communication ร้อยละ 35.5 เส้นเลือดแดง posterior communicating ร้อยละ17.1 เส้นเลือดแดง middle cerebral ร้อยละ 15.7 เส้นเลือดแดง internal carotid ร้อยละ 11.8 ปัจจัยที่เกี่ยวข้องกับผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์จากการแตกของหลอดเลือดสมองโป่งพองคือความดันโลหิตสูง และลักษณะทางภาพรังสีคือพบตำแหน่งเลือดที่ Sylvian fissure แต่ในทางกลับกันการพบตำแหน่งเลือดออกที่ tentorial cerebelli มีความเกี่ยวข้องว่าเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ในรายนั้น ไม่ได้เกิดจากการแตกของหลอดเลือดสมองโป่งพอง*

สรุป: *ผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ส่วนใหญ่เกิดจากจากการแตกของหลอดเลือดสมองโป่งพอง (ร้อยละ 57.6) ปัจจัยที่เกี่ยวข้องกับปัจจัยที่เกี่ยวข้องกับผู้ป่วยเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์จากการแตกของหลอดเลือดสมองโป่งพองคือความดันโลหิตสูง และลักษณะทางภาพรังสีคือพบตำแหน่งเลือดที่ Sylvian fissure แต่ในทางกลับกันการพบตำแหน่งเลือดออกที่ tentorial cerebelli มีความเกี่ยวข้องว่าเลือดออกชั้นใต้เยื่อหุ้มสมองอะแร็กนอยด์ในรายนั้นไม่ได้เกิดจากการแตกของหลอดเลือดสมองโป่งพอง*