Vasospasm and delayed cerebral ischaemia in patients with spontaneous subarachnoid haemorrhage (aneurysmal and pretruncal non-aneurysmal): a centre's perspective

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ABSTRACT

Background: Spontaneous subarachnoid haemorrhage (SAH) is a significant cause of stroke and associated with high morbidity and mortality. One substantial complication of SAH is cerebral vasospasm (CV) and delayed cerebral ischemia (DCI). This study aimed to define the clinical profile in patients with SAH, CV and DCI secondary to spontaneous SAH (aneurysmal and pretruncal non-aneurysmal).

Materials and Methods: We analysed 122 consecutive patients with spontaneous SAH following intracranial aneurysmal and non-aneurysmal information (including patients' pattern characterisation and their possible risk factor association to pre-operative clinical decision and long-term clinical outcome) was documented and analysed.

Results: The main clinical presentations for spontaneous SAH following ruptured intracranial aneurysm and non-aneurysm were headache (77%) and nausea/vomiting (62.3%). The most common sites for SAH following ruptured intracranial aneurysm rupture were the anterior and posterior communicating arteries (57.5%). Hypertension was the most common cause for SAH at 64.8%. It was found 26.2% (n=32) out of the 122 patients developed CV and DCI. The mean day of vasospasm was 6.0 ± 2.8 (range: 1-14 days) Age, length of stay, nausea/vomiting and visual field defect were significantly associated ($p<0.05$) with vasospasm. Mortality rate was also higher in the CV group compared to the group without CV in both at discharge and at 6 months; 281 versus 278 per 1000 and 312 vs 300 per 1000, respectively.

Conclusion: CV and DCI have a significant incidence among local patients with spontaneous SAH following an intracranial aneurysmal and non-aneurysmal rupture and it is associated with substantial morbidity. Prevention, effective monitoring, and early detection are keys to successful management. Regional investigation using a multicentre cohort to analyse mortality and survival rates may aid in improving national resource management of these patients.

KEYWORDS:
Cerebral Vasospasm, Delayed cerebral ischaemia, Malaysia, Outcome, Spontaneous subarachnoid haemorrhage

INTRODUCTION

Spontaneous subarachnoid haemorrhage (SAH) is a significant cause of stroke and may lead to severe neurological deficit or death. It is also associated with high morbidity and mortality for patients despite optimal medical and surgical treatment. Based on the World Health Organization the annual incidence of spontaneous SAH varies in different regions of the world between 2.0-22.5 per 100,000 populations with Finland and Japan having the highest incidence and South and Central America with lowest incidence.[1]

The secondary consequences of SAH – vasospasm (CV) and delayed cerebral ischaemia (DCI), by far one of the most notable complications of SAH carrying a 10-40% risk despite optimal medical treatment leading to permanent deficits in 20-40% of patients in this group.[2] Early recognition and rapid intervention in case of CV and DCI minimises morbidity and mortality in such patients.[3] Hence, identification of patients with spontaneous subarachnoid haemorrhage especially those spontaneous secondary to aneurysm rupture who develop CV is essential towards improving outcomes in patients with SAH.

Cerebral vasospasm (CV) can result in DCI. It was first documented angiographically by Ecker & Riemenschneider in 1951[4] and was associated with delayed focal neurological deficits.[5] Not all patients with CV, however, develop DCI. One third of the patients developed clinical manifestation of CV-DCI.[6,7] Vasospasm typically occurs between 3 to 21 days after the subarachnoid haemorrhage.[3,8] Although DCI is highly associated with CV, there may be other factors including microcirculatory constriction, micro-thrombosis, cortical spreading depression, and delayed cellular apoptosis.[9] Prevention, effective monitoring, and early detection of CV and DCI are the keys to successful management. Current practice involves administration of calcium channel blockers-

This article was accepted: 23 November 2020
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nimodipine and hypertensive therapy[10] as per AHA guideline. There has been long standing controversy over the use of magnesium sulphate infusion in the management of spontaneous SAH to prevent vasospasm. Current studies in the literature do not suggest a role for magnesium sulphate in mortality reduction after SAH.[11] Also, intravenous Clazosentan (an endothelin receptor antagonist that effectively reduces CV), and cholesterol lowering agent statins have also been studied in randomised trials with negative results.[12]

The purpose of this hospital based retrospective study was to define the clinical profile in patients with spontaneous SAH (aneurysmal and non-aneurysmal) at University Malaya Medical Centre (UMMC). UMMC, an urban 1200-bed teaching hospital serving the Kuala Lumpur - Petaling Jaya area with the population of 2.3 million. It is also to provide a better insight into the causal and relationship of the natural history of disease and socioeconomic impact. This information would serve as a reference in socioeconomic loss / planning for management of patients suffering from haemorrhagic stroke in future.

MATERIALS AND METHODS

Patients and data source

This study was approved by the University of Malaya Medical Centre Human Research Ethics Committee and performed in accordance with institutional ethics committee guidelines (MRECID: 968.12). Data was collected retrospectively for the duration of 10 years (January 2003 and December 2012) and analysed. There was a total of 122 consecutive patients (43 males and 79 female) diagnosed to have spontaneous subarachnoid haemorrhage (aneurysmal and non-aneurysmal) between the stated time frame. Demographics, clinical indications and radiological and treatment-related information were noted as well. The spontaneous SAH was confirmed by computerised tomography (CT) scanning and aneurysm identified by CT angiography.

CV can be angiographic vasospasm and clinical (symptomatic) vasospasm.[13] Angiographic vasospasm was considered to be present when there was unequivocal narrowing of the lumen of the artery on angiograms. Not all patients with CV, however, develop symptomatic vasospasm-hence delayed ischaemia. DCI was defined as documented arterial vasospasm that was consistent with new neurological deterioration, either transient or permanent, between 4 and 21 days after the onset of spontaneous SAH, when all other potential causes such as surgery, hydrocephalus, intracranial re-bleeding, seizure, infection, cardiopulmonary dysfunction, electrolyte imbalance and metabolic disturbances have been excluded. Patients with traumatic and mycotic aneurysms were excluded. Associated risk factor of hypertension is defined as a history of treatment with antihypertensive medication and /or systolic and diastolic blood pressure levels of greater than 160mmHg and 90mmHg documented during previous out-patient reviews.

Patients were treated according to standard local protocol for the management of spontaneous subarachnoid haemorrhage. The patient's Glasgow Coma Scale (GCS) and World Federation of Neurosurgical Societies (WFNS) scale were recorded. The WFNS classification is a specialised scoring system used to prognosticate patients with spontaneous subarachnoid haemorrhage. It uses Glasgow Coma Scale (GCS) and focal neurological deficits to gauge severity of symptoms. The WFNS scoring system[14] is used with the following categories, Grade 1 = GCS 15 with absent neurological deficit, Grade 2 = GCS 13-14 with absent neurological deficit, Grade 3 = GCS 13-14 with focal neurological deficit, Grade 4 = GCS 7-12 with or without focal neurological deficit and Grade 5 = GCS <7 with or without focal neurological deficit. Clinical (symptomatic) cerebral vasospasm was assessed clinically by the presence of acute onset of a focal neurologic deficit or a persistent (>1 hour) decline in the patient's Glasgow Coma Scale score by 2 or more points according to a recent consensus definition.[15,16] All suspected cases of cerebral vasospasm would then be confirmed by Transcranial Doppler (TCD). Transcranial Doppler vasospasm is defined as a mean blood flow velocity greater than 125cm/s in the anterior circulation or greater than 100cm/s in the posterior circulation in addition to a Lindegaard ratio (mean middle cerebral artery flow velocity/mean cervical internal carotid artery flow velocity) of greater than 3.[17] There was a standard protocol that all patients remain in follow up and TCD was consistently performed throughout the hospitalisation stay on day 1, day 3, day 5 and day 7 interval of admission.

The clinical status of each of the 122 surgical patients was documented at 6 months following surgery. A good outcome was defined as a modified Rankin Scale (MRS) score of 0 or 1; adverse outcomes were considered relevant if they produce a change in MRS of more than 1 as a result of intervention. The clinical condition assigned at last follow-up was used to determine the MRS. The MRS is the most widely used clinical measure for stroke clinical trials; measuring the degree of disability or dependence in the daily activities of people who have suffered stroke or other causes of neurological disability. The scale runs from 0-6; 0-No symptoms, 1-No significant disability, 2-Slight disability, 3-Moderate disability, 4-Moderately severe disability, 5-Severe disability and 6-Dead.[18]

Statistical Analysis

The analyses were performed with IBM SPSS Statistics for Windows software (Version 20.0. Armonk, NY: IBM Corp). Descriptive statistics were utilized for participating demographic, risk factor, clinical sign/symptoms, lateralising sign and physical examination data. Continuous variables are expressed as mean ± standard deviation and categorical variables as percentages.

Comparing outcome for numerical data between two groups was analysed using the independent t-test and Mann-Whitney test if the assumptions were not met. The Wilcoxon signed-rank test was used to compare two sets of modified Rankin scale (MRS) that come from the same participants. The Chi-square test was used to compare differences for categorical data among groups and Fisher's exact test was used as another alternative if the assumptions for chi-square test are not met. The McNemar test was used to test the difference between paired proportions. Two-tailed tests were used for all analyses, and the probability value of less than 0.05 (p-value<0.05) was considered as statistically significant.

RESULTS

Demographic and Clinical profile

Spontaneous subarachnoid haemorrhage

In the 10 years covered by this study, there were a total of 122 patients with spontaneous SAH treated at UMMC. Patients who were operated or initially received treatment in other centres were excluded as there was insufficient information on presenting symptoms. There were 43 men (35.2%) and 79 women (64.8%) with a male to female ratio of 1:2. The mean age was 56.6±14.17 years (range 27-83).

Patient's ethnicity was Chinese; 49.2% (n=60), Malay 32.0% (n=39), Indian 15.6% (n=19) and other ethnic; 3.3% (n=4). The most common clinical presentations for spontaneous SAH (aneurysmal rupture and non-aneurysmal) were headache (77%), nausea and vomiting (62.3%), loss of consciousness (52.5%) and seizures (12.3%). Other related presenting complaints were weakness (17.2%), visual disturbances (13.9%), speech disturbances (6.6%) and behavioural changes (2.5%).

There were multiple risk factors related to spontaneous SAH in this series. Hypertension was the most common associated risk factor for SAH which accounted for 79 patients (64.8%). However only 26.2% (n=32) were documented smokers.

The location of the aneurysm was determined following CT or invasive cerebral angiography. 106 patients (86.9%) were subjected to CT or Cerebral angiography. The remainder 16 patients were deemed unstable to undergo such a procedure. From the group of patients who underwent an angiography, an intracranial cerebral aneurysm was detected in 96 patients (90.6%). Most of the intracranial cerebral aneurysms were located at the anterior and posterior communicating arteries which account for more than half (57.5%) of the aneurysm locations. Other sites were middle cerebral artery 25.5% (n=27), internal carotid artery 5.7% (n=6), posterior inferior cerebral artery 1.9% (n=2), and one each for distal anterior cerebral artery, vertebral artery, anterior choroidal artery and basilar artery. Patients' presented with a poor WFNS grade (3, 4, 5) were marginally higher than a good WFNS grade, 54.9% (n=67) and 45.1% (n=55) respectively. However, there was no an association between WFNS grading and treatment (Table I).

Cerebral vasospasm (CV) and delayed cerebral ischaemia (DCI)

Thirty-two (26.2%) out of 122 patients developed CV and DCI which was documented with cerebral angiography and TCD. The mean for days for patient with spontaneous subarachnoid haemorrhage developed CV and DCI was 6± 2.8 (range 1 -14 days). We found that younger age, length of stay, nausea/vomiting and visual field defect were common presenting symptoms and significant associated factors as a result from vasospasm. The mean age for the non-DCI group (59.6±11.7 years) was higher than the mean age for the DCI group (50.6±10.9 years). Meanwhile, the length of stay for DCI group (Median = 24.0, IQR = 17.0) was longer compared to non-DCI group (Median = 16.0, IQR = 19.0). In DCI group, the nausea/vomiting (87.5%) and visual field defect (25.0%) showed significantly higher prevalence than in the non-DCI group. Other variables such as mean arterial pressure (MAP), gender, race, hypertension, smoker, headache, level of consciousness (LOC), fits, speech disturbances, weakness, inappropriate behaviour and WFNS grade were not statistically significant (Table II). Based on the TCD study in patients with spontaneous SAH who developed CV and DCI, it is observed that the median for Lindegaard ratio was 3.5 while the mean for velocity of MCA was 111.8±38.1 m/s.

Outcome

Thirty-four patients with spontaneous SAH following intracranial aneurysm and non-aneurysmal rupture died in hospital prior to discharge. The mortality rate was higher in the CV-DCI group compared to the group without the CV-DCI at discharge and 6 months, 28.1% vs. 27.8% and 31.2% vs. 30% respectively. The difference was not significant (Table III).

Patient's functional outcome was assessed with MRS and 75% (n=24) patients suffered a downgrade in function (MRS>1) as a result of CV-DCI based on noted in nine patients who died. There was no significant association between MRS upon discharge and 6 months with CV-DCI (Table III). Table IV and Table V showed WFNS grades, MRS at discharge and MRS at 6 months did not show any statistical significance to the outcome in patients who developed CV-DCI following spontaneous SAH (aneurysmal and non-aneurysmal rupture)

DISCUSSION

Demography

Spontaneous subarachnoid haemorrhage (SAH) is a dreadful cerebrovascular disease and contribute substantially to morbidity and mortality. Cerebral vasospasm is common sequelae that occurs in up to 70% of patients with spontaneous SAH.[19,20] In the 10 years covered by this study, a total of 122 patients were analysed. The mean age was 56.6 ±14.17 years (range, 27-83). There were total 43 male and 79 females. There is female preponderance and this is comparable to a study by Juvela which showed women,

Table I: Association between WFNS grade and treatment modality

Variables (n=122)	Treatment						p-value
	Interventional Coiling		Surgical Aneurysm Clipping		Conservative		
	n	(%)	n	(%)	n	(%)	
WFNS Grade							0.265[a]
1 and 2	16	(50.0%)	26	(49.1%)	13	(35.1%)	
3, 4 and 5	16	(50.0%)	27	(50.9%)	24	(64.8%)	

[a]Pearson's chi-square test; WFNS = World Federation of Neurosurgical Societies

Table II: Characteristics of 122 SAH patients according to variables and cerebral vasospasm (CV)-delayed cerebral ischaemia (DCI)

Variables	No (n = 90)				Yes (n = 32)				p-value
	n	(%)	Mean	(SD)	n	(%)	Mean	(SD)	
Age, years			59.6	(11.74)			50.6	(10.90)	0.001[a]
Length of Staye			16.0	(19.0)			24.0	(17.0)	0.035[b]
Systolic Blood Pressure			162.9	(27.08)			157.5	(24.23)	0.328[a]
MAP			107.8	(14.55)			110.2	(15.58)	0.461[a]
Gender									0.458[c]
Male	30	(33.3)			13	(40.6)			
Female	60	(66.7)			19	(59.4)			
Race									0.254[d]
Malay	28	(31.1)			11	(34.4)			
Chinese	48	(53.3)			12	(37.5)			
Indian	12	(13.3)			7	(21.9)			
Others	2	(2.2)			2	(6.3)			
Hypertension									0.326[c]
No	34	(37.8)			9	(28.1)			
Yes	56	(62.2)			23	(71.9)			
Smoker									0.514[c]
No	65	(72.2)			25	(78.1)			
Yes	25	(27.8)			7	(21.9)			
Nausea/Vomiting									0.001[c]
No	42	(46.7)			4	(12.5)			
Yes	48	(53.3)			28	(87.5)			
Headache									0.251[c]
No	23	(25.6)			5	(15.6)			
Yes	67	(74.4)			27	(84.4)			
LOC									0.119[c]
No	39	(43.3)			19	(59.4)			
Yes	51	(56.7)			13	(40.6)			
Fits									0.967[c]
No	79	(87.8)			28	(87.5)			
Yes	11	(12.2)			4	(12.5)			
Speech Disturbances									0.453[c]
No	85	(94.4)			29	(90.6)			
Yes	5	(5.6)			3	(9.4)			
Weakness									0.171[c]
No	72	(80.0)			29	(90.6)			
Yes	18	(20.0)			3	(9.4)			
Visual Field Defect									0.035[c]
No	81	(90.0)			24	(75.0)			
Yes	9	(10.0)			8	(25.0)			
Inappropriate Behaviour									0.566[d]
No	87	(96.7)			32	(100.0)			
Yes	3	(3.3)			0	(0.0)			
WFNS Grade									0.860[c]
1 and 2	41	(45.6)			14	(43.8)			
3, 4 and 5	49	(54.4)			18	(56.3)			

[a]Independent t-test; [b]Mann-Whitney test; [c]Pearson's chi-square test; [d]Fisher's exact test; [e]Presented as median (interquartile range);
SD = standard deviation; MAP = mean arterial pressure; LOC = level of consciousness; WFNS = World Federation of Neurosurgical Society

Table III: Mortality rate and cerebral vasospasm (CV) and delayed cerebral ischaemia (DCI)

Variables		No (n = 90)		Yes (n = 32)		p-value
		n	%	n	%	
Mortality rate at discharge		278 per 1,000		281 per 1,000		0.970[a]
	No	65	72.2	23	71.9	
	Yes	25	27.8	9	28.1	
Mortality rate at 6 months		300 per 1,000		312 per 1,000		0.895[a]
	No	63	70.0	22	68.8	
	Yes	27	30.0	10	31.2	

[a]Pearson's chi-square test

Table IV: Association between MRS and cerebral vasospasm (CV) and delayed cerebral ischaemia (DCI)

Variables	No (n = 90)		Yes (n = 32)		p-value
	n	(%)	n	(%)	
MRS at discharge					0.325[a]
0 and 1	31	(34.4)	8	(25.0)	
2 to 6	59	(65.6)	24	(75.0)	
MRS at 6 months					0.231[a]
0 and 1	39	(43.3)	10	(31.2)	
2 to 6	51	(56.7)	22	(68.8)	

[a]Pearson's chi-square test; MRS = modified Rankin scale

Table V: Association between variables and outcome in cerebral vasospasm (CV) and delayed cerebral ischaemia (DCI)

Variables	Outcome								
	Lindegaard Ratio			Velocity			Day of Vasospasm		
	Median	(IQR)	p-value[b]	Mean	(SD)	p-value[a]	Mean	(SD)	p-value
Overall	3.5	(0.8)		111.8	(38.05)		6.0	(2.82)	
WFNS Grade			0.711			0.806			0.711[b,c]
1 and 2	3.4	(1.1)		109.8	(33.61)		6.5	(5.0)	
3, 4 and 5	3.5	(0.8)		113.3	(42.09)		6.0	(3.0)	
MRS at discharge			0.631			0.496			0.971[a]
0 and 1	3.4	(0.8)		103.0	(26.65)		6.0	(2.52)	
2 to 6	3.5	(0.8)		114.5	(41.03)		6.1	(2.97)	
MRS at 6 months			0.114			0.203			0.917[a]
0 and 1	3.2	(0.8)		98.1	(25.99)		6.1	(2.36)	
2 to 6	3.5	(0.8)		117.6	(41.34)		6.0	(3.03)	

[a]Independent t-test; [b]Mann-Whitney test; [c]Presented as median (interquartile range); SD = standard deviation; MRS = modified Rankin scale; WFNS = World Federation of Neurosurgical Societies

particularly those over the age 55, are more likely and have a higher risk of SAH from rupture brain aneurysm than men (3:2 ratio). In the study it was shown that female gender was the only significant independent risk factor ($p<0.05$) for *de novo* aneurysm formation for cerebral aneurysm growth.[21] It has been reported ethnicity of African-Americans and Hispanics are about twice as likely to have SAH from brain aneurysms compared to Caucasian.[22] In our study we found that the Chinese accounted for 49.2% of the study cohort. This does not reflect the national demographics of racial distribution in Malaysia which is predominantly of the Malay. This could be explained by the fact that this study was conducted in UMMC which is located in Petaling Jaya which has a higher density of Chinese population in Malaysia. However ethnical discrepancies can be a common affair for this disease based on previous data.[23,24]

It was also observed that more than half of intracranial cerebral aneurysms were located at the anterior and posterior communicating arteries (57.5%) in this cohort. Delgado Almandoz et al., in a previous study observed the size and location of ruptured intracranial aneurysms involving 588 patients with first-time acute SAH treated endovascularly at their centre noticed the similar pattern of aneurysm location over a 16-year time period. Ruptured brain aneurysm is fatal in about 50% of cases.[25] Of those who survived, about two third suffer some permanent neurological deficit.

Monitoring

The extravasation of blood that accumulating in the subarachnoid and perivascular spaces could trigger CV. There are several predictors of CV, including the amount of blood on CT scan (Fisher CT scale), location of aneurysms, age, possible gender, smoking, hypertension and presence of intraventricular blood clot. Of all these factors, location and high Fisher CT scale, are consistently reported as the most powerful predictors of CV.[26] Not all patients with CV, however, develop DCI. In current cohort, there was 26.2% of CV-DCI found in patients following spontaneous SAH which is comparable to other studies. The incidence of angiographic vasospasm reported at 43.3% and symptomatic vasospasm or delayed cerebral ischaemic (DCI) occurred in 32.5% based on a review conducted by Dorsch and King in an Australian population.[27]

Standard protocol is in placed including admitting patients to comprehensive stroke unit with continuous clinical and radiological assessment, as early detection of neurologic deterioration is the mainstay treatment of CV. Nimodipine (60mg every 4 hours) administration with baseline transcranial Doppler (TCD) ultrasound measurement on arrival is acquired and daily thereafter to guide blood pressure hypertensive therapy management. Haemodynamic optimisation (euvolemia plus hypertension) and early aggressive treatment within two hours of neurology deterioration is the best strategy to prevent DCI. We found that age was statistically significant factors with patients with CV-DCI. CV-DCI was seen to be more prevalent in the younger age group (50.6±10.90 versus 59.6±11.74). This result is consistent with a study by Kale and colleagues the risk of CV-DCI in aneurysmal SAH that a younger age group of patients developing CV-DCI (mean 48.51±11.23 years) compared to those did not develop CV-DCI (mean 59.67± 13.30 years) with statistical significance of $p<0.001$.[28] In addition, a stronger interventional radiology (IR) team support is crucial and should made available to patients with DCI from VS. The IR play an important role to make the treatment more robust and strongly conclusive of VS compared to only using TCD.[29]

The treatment of ruptured brain aneurysm is far more costly than the treatment of unruptured aneurysms. Patients who developed CV-DCI were found to have a significantly longer length of hospital stay compared to the group without CV, 24.0±17.0 days versus 16.0±19.0 days, respectively (p=0.035). Yundt and colleagues some time ago had investigated hospital resource utilisation in the treatment of cerebral aneurysms deduced that the total cost per patient was highest in the group of patients who developed CV-DCI which is correlated with hospital stay.[30] Hence hospital resources can be used sparingly if patients with CV are identified early and rapid intervention is instituted. This would also improve patient outcome and length of patient hospital stay.

Outcome

Approximately 15% of people with a spontaneous SAH from ruptured aneurysm die before reaching the hospital. Most of the deaths are due to rapid and massive haemorrhagic stroke. In the current series, more than half of the patients with spontaneous SAH (54.9%) presented to our centre were noted to have poor WFNS grades, i.e., 3, 4 and 5. This raises the question the level of health awareness among the community which leads to this late presentation for treatment once patients are at clinically deteriorated state. Nevertheless, in terms of patient care there was no statistical significance between the good and poor WFNS grade of the patient and the treatment provided, it was observed that both the WFNS groups were treated equally (Table V).

Identically a study conducted by Wostrack and colleagues[31] showed one hundred and three patients with WFNS grade V SAH received maximal treatment and retrospectively evaluated a favourable outcome at discharge 16% (n = 16) of cases, whereas in the follow-up it rose to 26% (n = 27). Their study concluded that a favourable outcome was achieved in 26% of aggressively treated patients, rendering the withdrawal of maximal therapy for WFNS grade V SAH patients unacceptable. Hence, patients with poor WFNS grade (grade V) should also be treated aggressively without any sense of bias as they have been proven to have a favourable outcome following aggressive treatment.[31]

This paper also described the functional outcome of patient with CV-DCI following spontaneous SAH in an urban Malaysian population. There have been numerous studies correlating functional outcome including Glasgow outcome scale (GOS) with in patients with CV-DCI following SAH.[14,32,33] The proportion of patients with a poorer MRS score, i.e., 2-6 was certainly higher in the group with CV-DCI in comparison to the group without CV-DCI at the 2-time point intervals which was at discharge and at 6 months. However, there is no difference between MRS upon discharge and 6 months with vasospasm. Further investigation would be promising.

LIMITATIONS AND FUTURE DIRECTION

The main limitation in this study was sample size due to it being a single centre study. Nonetheless, this report looked at incidence of CV-DCI in a local Malaysian population. An establishment of a multicentre database of patients with spontaneous SAH (aneurysmal and non-aneurysmal) in Malaysia would give a more accurate projection of incidence of CV-DCI, functional outcome and mortality for both treatment and non-treatment cohorts to reduce any potential biasness. In addition, another limiting factor due to insufficient numbers this work did not venture and investigate the well-known predictors and or risk factors of vasospasm or DCI hence worthwhile investigating in the near future with larger numbers of patients. Some of these known predictor or risk factors includes Fisher CT Grade, rebleeding, alcohol intake, leucocytosis, hyperglycaemia, and electrocardiographic QTc prolongation, left ventricular hypertrophy, and ST depression. Moreover, the effect of various treatment types and VS/DCI as well as the timing of these interventions should be investigated in future studies. A database collection with the aforesaid data provides valuable information on patient outcome and this may hopefully improve on patient management.

CONCLUSION

Vasospasm and DCI has a significant incidence among local patients with spontaneous SAH (aneurysmal and non-aneurysmal) and it is associated with substantial morbidity. Prevention, close vigilant neurology and TCD monitoring with haemodynamic optimization (euvolemia plus stepwise hypertensive therapy adjustment) are the keys to successful management. Further investigation using a multicentre cohort to analyse mortality and survival rates may aid in improving national resource management of these patients in Malaysia.

ACKNOWLEDGEMENTS

Part of this work is supported by Fundamental Research Grant Scheme (FRGS) initiative FRGS [FP019-2013B] and Postgraduate Research Fund (PPP) [P0048-2013B] of University of Malaya.

DISCLOSURES

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

CONFLICT OF INTEREST

All the authors declared no conflict of interests.

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