Malaysian Journal of Medical Sciences, Vol. 16, No. 4, Pg 77-78, October - December 2009

LETTER TO THE EDITOR

Non-detection of Acute Angiography-induced Cerebral Vasospasm by Transcranial Doppler Sonography amongst Patients with Subarachnoid Haemorrhage in Kelantan

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Keywords: *neurosciences, neuroradiology, angiography, vasospasm*

Dear Editor,

We conducted a study to determine the proportion of patients with spontaneous subarachnoid haemorrhage (SAH) who developed cerebral vasospasm (CVS) following cerebral intra-arterial digital subtraction angiography (IADSA) and to identify the angiography related risk factors that cause acute CVS following cerebral IADSA by using transcranial Doppler (TCD). A prospective study was conducted over an 18-month period from May 2007 until October 2008 at the Hospital Universiti Sains Malaysia (HUSM). A total of 8 patients above 12 years of age (5 males and 3 females, mean age ± SD: 54.1 ± 13.1) who presented with spontaneous SAH and fulfilled the inclusion criteria were enrolled into this study. Intra-arterial cerebral catheter angiography, even though it is the gold standard modality for assessment of the cause of SAH, is an invasive test with potentially significant neurological complications, with 0.07% to 0.3% risk of permanent stroke and an overall 1.8% risk of systemic complication (1). In patients with intracranial haemorrhage, particularly SAH, it is very difficult to determine whether the development of the neurological complications is part of the natural course of the disease or if it is caused by the diagnostic angiographic examination, which was performed before or during the time when CVS commonly occurs. This raises the question whether cerebral angiography itself could be one of the multiple factors causing the development of CVS among patients with SAH.

This study was undertaken to evaluate whether cerebral IADSA itself is the triggering mechanism for developing CVS in patients with SAH. The TCD was done according to the technique of Aaslid (2). The patients underwent IADSA examinations and the mean blood flow velocity (mBFV) values of the bilateral middle cerebral arteries (MCA) were obtained from the non-invasive TCD examination—which were performed within 6 hours before (the first TCD exam) and after (the second TCD exam) cerebral IADSA. Iso-osmolar non-ionic water-soluble iodinated contrast medium was used during the cerebral IADSA. A mBFV measurement of more than 120 cm/s during the second TCD examination was taken as indicator of angiography-induced CVS development. None of the subjects in this study developed angiography-induced CVS based on the TCD criteria, giving the rate of 0% for CVS. When the patients were compared according to changes in mBFV before and after IADSA, there was no statistical difference in mBFV (P = 0.95 and P = 0.07 for left and right MCA respectively). This study suggests that with the use of iso-osmolar non-ionic contrast medium, combined with improved catheterization techniques and the application of digital technology, cerebral IADSA can be performed with a zero rate of angiography-induced vasospasm in patients with SAH in HUSM. Thus the patients were not put at risk from the diagnostic procedures. Cerebral IADSA will continue to be an important diagnostic imaging modality in managing patients with SAH in the years to come.

To the authors' best knowledge, there is only one study with a similar methodology by Arslantas et al. (3) that has been published in the English-language literature. In that study, which was conducted in Turkey, the authors compared the changes in cerebral blood flow velocity before and after cerebral angiography by using TCD in patients with SAH undergoing IADSA. They used low-osmolar non-ionic contrast medium (CM). In that

study, 30 patients underwent TCD examination immediately before and after an angiography using continuous TCD monitoring in the angiography suite. They included only SAH patients with good clinical Hunt and Hess grades (grade I, Glasgow Coma Scale (GCS) 14 – 15 and grade II, GCS 12 – 13). However, they could not find a statistically significant difference between pre- and post-angiography mBFV of the MCA. Also, they found no significant difference in the clinical course before and after cerebral angiography as seen by changes in GCS (4).

In our study, we used iso-osmolar non-ionic CM and performed TCD of bilateral MCA in the neurology intensive care ward within 6 hours before and after cerebral IADSA to detect any possible angiography-induced CVS. All the clinical World Federation of Neurological Surgeon Scale (WFNSS) grades were included in this study. None of the patients in this study developed CVS after IADSA within the interval between IADSA and the second TCD examination. Similarly, we also could not find significant difference between mBFV before and after IADSA in this study.

Despite its wide acceptance, TCD ultrasonography can be affected by many variables, including a hyperdynamic state on the part of the patient. It is operator-dependent and the cerebral circulation is very complex (2,5). It may be more useful to follow trends than absolute numerical results. Generally, increases of 25 to 50 cm/s/day are considered ominous (5,6). Therefore, in future study of this kind, studying the difference in the TCD readings before and after IADSA would provide more accurate identification of acute angiography-induced CVS by TCD in patients with spontaneous SAH. The small sample size was another limitation of this study, which was due to strict patient selection criteria. A larger sample size is required for future study in order to gain a stronger degree of statistical significance. Based on the prevalence of post-angiography neurological complication of 0.3% reported by Cloft et al. (1), the sample size should include at least 30 subjects.

Author's contributions

Conception and design, darfting of the article: RJ, MSA
Data collection: NANJ
Data analysis and interpretation: RJ
All authors read and approved the final manuscript.

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