Int J Clin Exp Med 2016;9(6):12313-12317
www.ijcem.com /ISSN:1940-5901/IJCEM0023399

Case Report

Using of transcranial Doppler sonography to guide triple-H therapy in cerebral vasospasm associated with subarachnoid hemorrhage

Zhenxing Ding, Hong Zhang

Department of Emergency and Intensive Care Unit, The First Affiliated Hospital of Anhui Medical University, Hefei 230022, Anhui Province, China

Received January 6, 2016; Accepted March 21, 2016; Epub June 15, 2016; Published June 30, 2016

Abstract: Prophylactic triple-H therapy (the combination of induced hypertension, hypervolemia, and hemodilution) has been suggested to prevent cerebral vasospasm after subarachnoid hemorrhage (SAH) that would cause increased cerebral blood flow (CBF) to the brain, and widely accepted in a number of institutions. It must be noted that triple-H therapy is not completely safe, sometimes carries significant medical morbidity. The measurement of CBF with transcranial Doppler sonography (TCD) has provided a way of early detecting vasospasm after SAH as soon as possible and could guide the timing of triple-H therapy. Here, we present two patients who developed vasospasm associated with Aneurysmal SAH. In both patients, TCD examinations were daily performed to detect vasospasm before the onset of its clinical effects. One patient underwent mild vasospasm, resolved only by the treatment of triple-H therapy. While the second, followed-up triple-H therapy, suffered from vasodilator administered by intra-arterial injecting for its severe vasospasm. In conclusion, our cases suggest that TCD optimize the timing of triple-H therapy in cerebral vasospasm associated with SAH.

Keywords: Transcranial Doppler sonography, vasospasm, subarachnoid hemorrhage, triple-H therapy

Introduction

Cerebral vasospasm is associated with a high incidence of permanent disability and death following acute SAH) [1-4]. Because cerebral vasospasm occurs in a delayed ischemic neurological deficit and could predict, it is an opportunity to give aggressive preventative and therapeutic strategies. In the intensive care unit (ICU), patients after SAH often will be treated with triple-H therapy to prevent and treat cerebral vasospasm that would cause increased CBF to the brain [5, 6]. Prophylactic triple-H therapy has been demonstrated to decrease the frequency and severity of vasospasms and subsequently reduce the incidence of clinical deficits [5, 7, 8]. However, triple-H therapy has serious side effects, such as cerebral hemorrhage, cerebral edema, pulmonary edema and cardiac arrhythmias, especially elderly patients with poor cardiac function may not tolerate induced hypertension with vasopressor agents or volume loading [9].

Although the gold standard for vasospasm diagnosis is cerebral angiography, carries added risks of complications associated with the invasiveness of the procedure. In addition, the procedure requires that the patient be transported (generally from the intensive care unit) to the radiologic imaging department [10]. TCD is a relatively accurate and sensitive, non-invasive tool, allowing for bedside monitoring to determine flow velocities indicative of changes in vascular caliber. In turn, assessment of these velocities contributes valuable index regarding the diagnosis of cerebral vasospasm and, thereby, assists in decision-making regarding the need for therapeutic interventions [11].

In this study we present two patients developed vasospasm after SAH treated by triple-H therapy. TCD done in both patients by the performance of daily studies correlated with clinical examinations and physiologic data for the surveillance and monitoring of vasospasm. We discussed TCD can guide the timing of triple-H

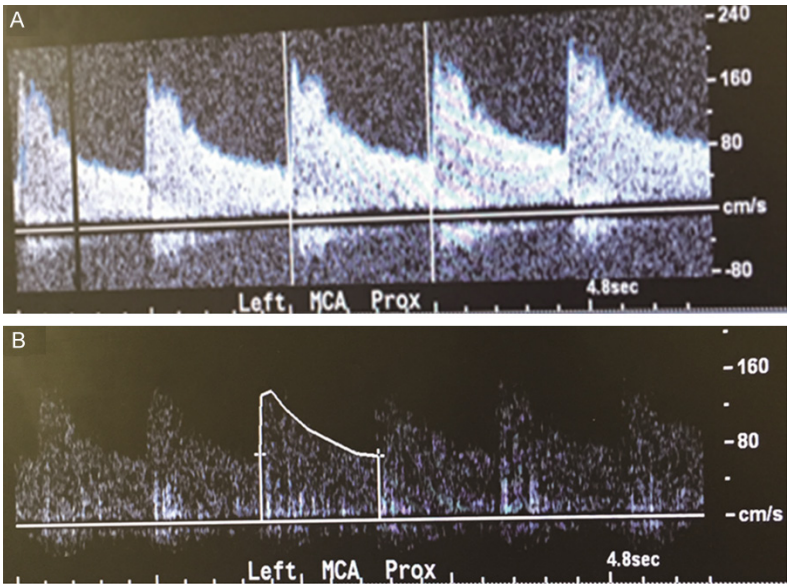

Figure 1. The TCD velocity spectral waveforms obtained from the left MCA of patient 1. A. Increased PSV (230 cm/s) indicated mild vasospasm. B. After the treatment of tripe-H therapy, PSV changed to normal (154 cm/s).

therapy and the tailoring of aggressive treatment regimens after SAH.

Case report

Patient 1

A 61-yr-old female was admitted to the hospital on January 5th, 2015, with no significant past medical history, no medications. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Anhui Medical University. Written informed consent was obtained from all participants. Patient awoke with worst headache of life with R sided weakness at 3:30 am on the morning of admission. According to EMS (Emergency Medical Service System), patient had transient right side facial droop, right side grip weakness, right side arm drift, with abnormal speech, and then were resolved on the Emergency Department. Of note patient took aware this AM. Patient did mention multiple small sharp headaches that would last a few seconds and go away over the past couple of weeks, denied any other history of headaches. The CTA (computed tomography angiography) on admission demonstrated dysplastic basilar tip aneurysm measuring 7 × 5 mm and two tiny nipple-like protrusions. Noncontrast CT demonstrated diffuse SAH, which completely fills the basal cisterns and extends through the foramen magnum into the upper spinal canal. The ventricles were mildly dilated suggesting early developing hydrocephalus. Therefore, he was admitted to the ICU for observation.

A few hours into his admission, Patient was intubated and EVD (external ventricular drainage) placed for decreasing level of consciousness likely related to hydrocephalus. In addition, she was treated with standard hemodynamic management (Goal euthermia, euvolemia, normonatremia, systolic blood pressure goal <140 mmHg), and completely coiled embolization on the second day. Her neurological exam improved significantly, then successfully extubated to aerosol mask. Daily TCD assessment was performed, no evidence of vasospasm in anterior and posterior circulation on the first couple days of her hospitalization. On the 6th day, patient's neurological condition deteriorated. She was lethargy and had a very mild RUE (right upper extremity) pronator drift. Meanwhile, the TCD result of the MCA (middle cerebral artery) on the left side showed an increased Lindegaard ratio and increased PSV (peak systolic velocity) in the proximal left MCA (**Figure 1A**). This was judged to be indicative of mild vasospasm [10], and tripe-H therapy was initiated. The gold of systolic blood pressure changed to 160-200 mmHg, started gentle pressor (noradrenaline). Simultaneously, the patient was administrated extra sodium chloride for positive fluid balance. Twenty-four hours after the intervention, TCD measurement was repeated, and showed normal velocities in the left MCA (**Figure 1B**). The patient was weaned from the tripe-H therapy over 2 days and continued to do well. On the 10th day, he was transferred to gentle ward prior to returning home.

Patient 2

A 69-yr-old female was admitted to the hospital on May 17th, 2015, presented with headache and sudden collapse when having dinner with

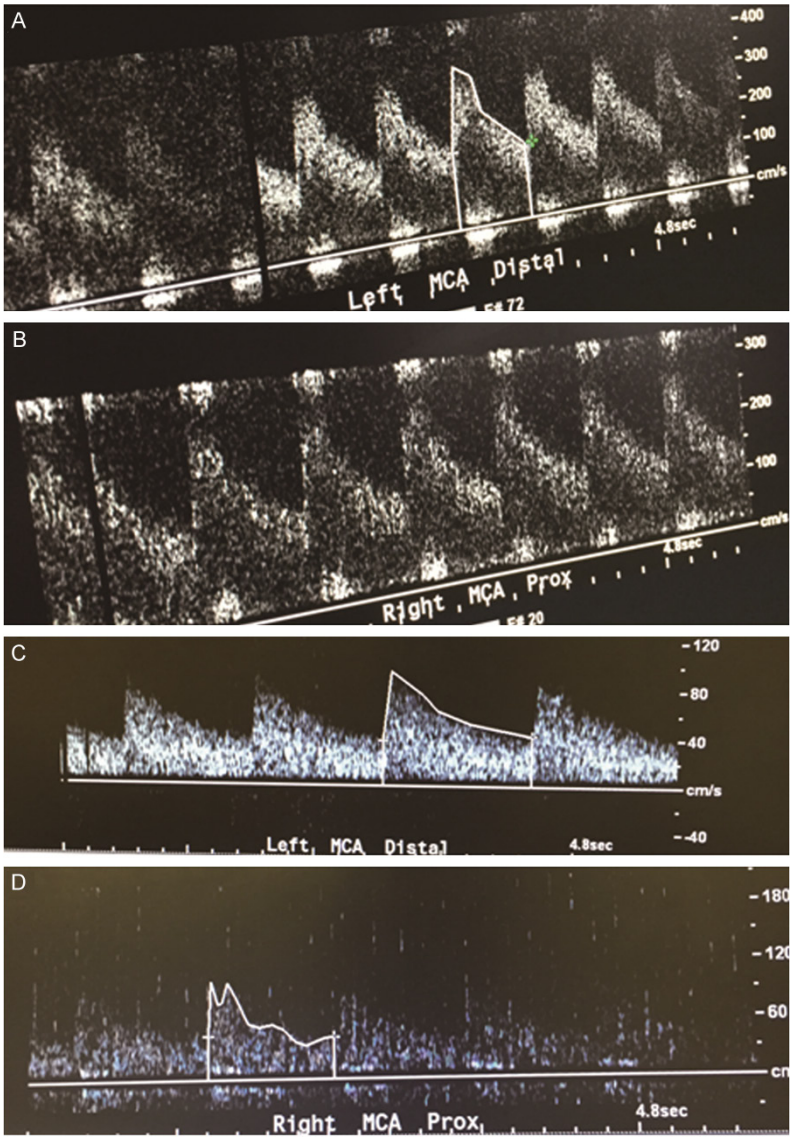

Figure 2. The TCD velocity spectral waveforms obtained from bilateral MCAs of patient 2. A and B: TCD showed bilateral MCAs vasospasm associated with CBF velocities high to 300; C and D: After continued treatment of tripe-H therapy and Verapamil intravascular administrating, both PSV dropped to normal (103 cm/s, 98.3 cm/s, respectively).

friends and presented to hospital. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Anhui Medical University. Written informed consent was obtained from all participants. She subsequently deteriorated in the emergency department. She was intubated and was transferred to ICU for further management. CT demonstrated diffuse SAH. CTA Head found ruptured right posterior communicating artery aneurysm. When admitted to ICU, her Glasgow coma scale (GCS) was 12. The patient underwent the placement of EVD and arterial line due to significant hydrocephalus, then, was treated with standard hemodynamic management (Goal euthermia, euvolemia, normonatremia, systolic blood pressure goal <140 mmHg). TCD assessment was also daily performed for monitoring cerebral vasospasm. On the second hospital day, the right posterior communicating artery aneurysm was successfully coiled. Five days later, the patient deteriorated neurologically to a GCS score of 6, less withdrawing to noxious on exam. At this time, TCD showed bilateral MCAs vasospasm associated with CBF velocities high to 300 (**Figure 2A**, **2B**). Although the tripe-H therapy was initiated, the patient's clinical state remained poor in 48 hours. The repeated TCD demonstrated no obviously improvement in relation to prior values. Patient was brought for CTA (showed moderate vasospasm of the bilateral A1, M1, and M2 segments of the anterior and middle cerebral arteries), subsequently went to angio (mild to moderate vasospasm in right MCA; moderate to severe vasospasm left MCA, not amenable to angioplasty; 20 mg Verapamil was intravascular administrated both to right and left MCA). Approximately 24 hours later, her neurological status improved and TCD index dropped to normal. (**Figure 2C**, **2D**). Continued tripe-H therapy, she recovered slowly. On the 17th day the patient was discharged and transferred to gentle ward.

Discussion

TCD was first reported by Aaslid and colleagues in 1982 [12] as a non-invasive, rapid technique for monitoring cerebral vasospasm, based on

the principle that a decrease in vessel diameter translates into an increase in blood flow velocity. As a non-invasive, bedside test with minimal risk, TCD has become a standard screening tool in Neurocritical Care Unit. The measurement of CBF velocities with TCD has provided a way of detecting developing vasospasm after SAH [12]. The diagnosis of cerebral vasospasm may be suspected on the basis of clinical examination and TCD results, and can be confirmed with cerebral angiography. Meanwhile, the sensitivity and specificity of TCD for the detection of both angiographically and clinically defined vasospasm are high. Therefore, TCD is an excellent first-line examination to identify those patients who may need urgent aggressive intervention [13].

Our experience has evolved to use TCD in patients with SAH by the performance of daily studies correlated with clinical examinations and physiologic results. Patients admitted with SAH are tested as soon as possible after digital subtraction angiography and securing of the aneurysm. In the setting of clinical stability, TCD are continued daily while patients are maintained in a state of euvolemia and normonatremia. When a patient with abnormal clinical examination, if TCD velocities increase to generally accepted levels for vasospasm for that vessel, a trial of tripe-H therapy is instituted. Fluid balance is shifted to maintaining a positive fluid state, and patients are allowed to autoregulate blood pressure up to systolic pressures (SBP) of 200 mmHg or mean arterial pressures (MAP) of 120 to 140 mmHg, depending on the clinical status of the patient and other existing comorbidities. If unsuccessful, the patient proceeds to angioplasty. If the physical examination is difficult or obscured, and the TCD results indicate the presence of cerebral vasospasm, a CTA or angiogram is suggested. If vasospasm is confirmed, tripe-H therapy is undertaken, and if unsuccessful, cerebral angiography, as both a diagnostic and therapeutic intervention, may be performed at this stage. TCD followed-up then may be important in assessing the results of therapy and, along with the clinical exam, will assist in the timing of repeat angiography and will guide the hemodynamic management.

In conclusion, TCD has become the most common screening tool for monitoring vasospasm preceded neurologic deficits, prompting the timing of hemodynamic management, and avoiding the blind treatment of prophylactic tripe-H therapy after SAH.

Disclosure of conflict of interest

None.

Address correspondence to: Hong Zhang, Department of Emergency and Intensive Care Unit, The First Affiliated Hospital of Anhui Medical University, No. 218 Jixi Road, Hefei 230022, Anhui Province, China. Tel: +86 551 62922289; E-mail: cnhongzhang@126.com

References

[1] Kassell NF, Sasaki T, Colohan AR and Nazar G. Cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Stroke 1985; 16: 562-572.

[2] Kassell NF, Torner JC, Haley EC Jr, Jane JA, Adams HP and Kongable GL. The international cooperative study on the timing of aneurysm surgery. Part 1: Overall management results. J Neurosurg 1990; 73: 18-36.

[3] Allen GS, Ahn HS, Preziosi TJ, Battye R, Boone SC, Boone SC, Chou SN, Kelly DL, Weir BK, Crabbe RA, Lavik PJ, Rosenbloom SB, Dorsey FC, Ingram CR, Mellits DE, Bertsch LA, Boisvert DP, Hundley MB, Johnson RK, Strom JA and Transou CR. Cerebral arterial spasm-a controlled trial of nimodipine in patients with subarachnoid hemorrhage. N Engl J Med 1983; 308: 619-624.

[4] Heros RC, Zervas NT and Varsos V. Cerebral vasospasm after subarachnoid hemorrhage: an update. Ann Neurol 1983; 14: 599-608.

[5] Bederson JB, Connolly ES Jr, Batjer HH, Dacey RG, Dion JE, Diringer MN, Duldner JE Jr, Harbaugh RE, Patel AB and Rosenwasser RH. 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.

[6] Diringer MN, Bleck TP, Claude Hemphill J 3rd, Menon D, Shutter L, Vespa P, Bruder N, Connolly ES Jr, Citerio G, Gress D, Hänggi D, Hoh BL, Lanzino G, Le Roux P, Rabinstein A, Schmutzhard E, Stocchetti N, Suarez JI, Treggiari M, Tseng MY, Vergouwen MD, Wolf S and Zipfel G. Critical care management of patients following aneurysmal subarachnoid hemorrhage: recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. Neurocrit Care 2011; 15: 211-240.

[7] Hoff R, Rinkel G, Verweij B, Algra A and Kalkman C. Blood volume measurement to guide fluid therapy after aneurysmal subarachnoid hemorrhage: a prospective controlled study. Stroke 2009; 40: 2575-2577.
[8] Meyer R, Deem S, Yanez ND, Souter M, Lam A and Treggiari MM. Current practices of triple-H prophylaxis and therapy in patients with subarachnoid hemorrhage. Neurocrit Care 2010; 14: 24-36.
[9] Raabe A, Beck J, Keller M, Vatter H, Zimmermann M and Seifert V. Relative importance of hypertension compared with hypervolemia for increasing cerebral oxygenation in patients with cerebral vasospasm after subarachnoid hemorrhage. J Neurosurg 2005; 103: 974-981.
[10] Kirsch JD, Mathur M, Johnson MH, Gowthaman G and Scoutt LM. Advances in Transcranial Doppler US: Imaging Ahead. Radiographics 2013; 33: E1-14.
[11] Rigamonti A, Ackery A and Baker AJ. Transcranial Doppler monitoring in subarachnoid hemorrhage: a critical tool in critical care. Can J Anaesth 2008; 55: 112-123.
[12] Aaslid R, Markwalder TM and Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 1982; 57: 769-774.
[13] Kalanuria A, Nyquist PA, Armonda RA and Razumovsky A. Use of Transcranial Doppler (TCD) ultrasound in the Neurocritical Care Unit. Neurosurg Clin N Am 2013; 24: 441-456.