

## Case Report

# Early detection of cerebral vasospasm in a patient with subarachnoid hemorrhage by transcranial Doppler sonography

Zhen-Xing Ding, Hong Zhang

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

Received February 21, 2016; Accepted May 15, 2016; Epub July 15, 2016; Published July 30, 2016

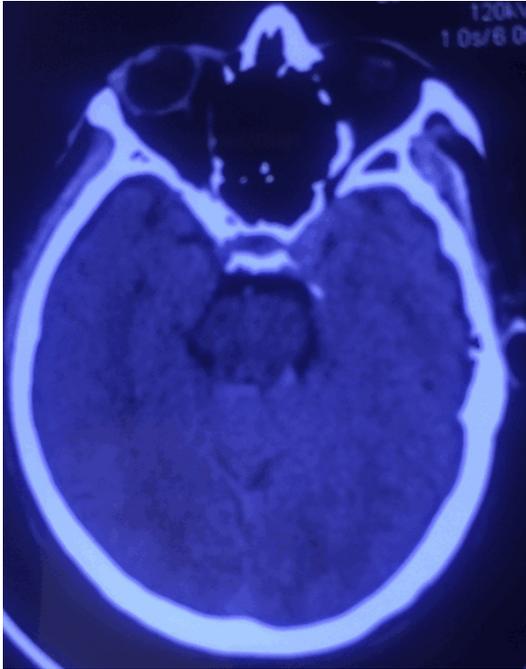
**Abstract:** Cerebral vasospasm is the most common complication in patients following acute subarachnoid hemorrhage (SAH), and results in a high mortality. Although the gold standard for vasospasm diagnosis is conventional angiography, in the intensive care unit (ICU), it is difficult to detect cerebral vasospasm in unconscious patients, in whom transportation for the procedure could be risky. Transcranial Doppler sonography (TCD) is a noninvasive bedside test that allows accurate and sensitive evaluation of cerebral vasospasm before serious neurologic deterioration occurs. Here, we present a critical patient who suffered from vasospasm associated with SAH. His clinical state was continuously poor. On the 6th day of his hospitalization, daily TCD showed a significantly increased peak systolic velocity (PSV) in the right middle cerebral artery (MCA) which was judged to be indicative of mild vasospasm. After daily TCD examinations and urgent aggressive treatment, the patient gradually recovered and was transferred to a general ward. To conclude, TCD is a bedside test and an excellent first-line examination for the early detection and monitoring of patients who are suffering from cerebral vasospasm in ICU.

**Keywords:** Subarachnoid hemorrhage, cerebral vasospasm, transcranial Doppler sonography

## Introduction

Subarachnoid hemorrhage (SAH) can result in vasospasm of the cerebral vessels, and is associated with a high incidence of stroke and death [1]. Cerebral vasospasm is a clinical diagnosis characterized by decreased cerebral blood flow (CBF) and decreased cerebral perfusion after SAH due to the constriction of cerebral arteries [2-4]. The diagnosis of cerebral vasospasm may be based on clinical examination and transcranial Doppler sonography (TCD) indexes, and can be confirmed with cerebral angiography. Cerebral angiography is the gold standard for detecting vasospasm, but carries some risks of complications associated with the invasiveness of the procedure, and generally requires that the patient be transported from the intensive care unit to the radiological imaging department. Meanwhile, in the intensive care unit (ICU), clinical assessments of a patient's neurological condition will only detect

the clinical signs and symptoms that occur only after vasospasm has already had significant effects on the brain. The measurement of CBF velocities with TCD has offered a way of monitoring developing vasospasm, while helping to recognize the development of its process before the onset of its clinical effects [5]. In particular, the sensitivity and specificity of TCD examination for the detection of both angiographically and clinically defined cerebral vasospasm are high. Therefore, TCD has become the most commonly used bedside screening tool for vasospasm monitoring due to its inexpensive, portable and noninvasive nature, and for the ease of repeat testing in the neurocritical ICU [6]. In this study, we present one critical patient who developed vasospasm after SAH in ICU. TCD performed daily correlated with clinical examinations for the diagnosis and monitoring of vasospasm. We also discuss the use of TCD to detect cerebral vasospasm in critical patients after SAH and improve outcomes.



**Figure 1.** CT scan of patient showed SAH.

### Case report

A 53-year-old man was admitted to hospital on September 20, 2015, with no medical history, about 2 hours after being injured in a traffic accident. On admission, his Glasgow coma scale (GCS) was 9 while computed tomography (CT) head in the emergency department showed SAH (**Figure 1**). The patient was admitted to the ICU for further management and care. 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.

Few hours after admission, the patient was intubated because of loss of consciousness likely related to hydrocephalus. TCD accompanied with physical exam was performed daily for monitoring cerebral vasospasm. No evidence of vasospasm in the anterior and posterior circulation was found during the first couple of days of hospitalization. Six days later, the patient's clinical state remained poor, and extubation failed. Meanwhile, TCD of the right MCA showed an elevated PSV (**Figure 2**), which was indicative of mild underlying vasospasm [7]. At that time, triple-H therapy (the combination of induced hypertension, hypervolemia, and hemodilution) was initiated. The target sys-

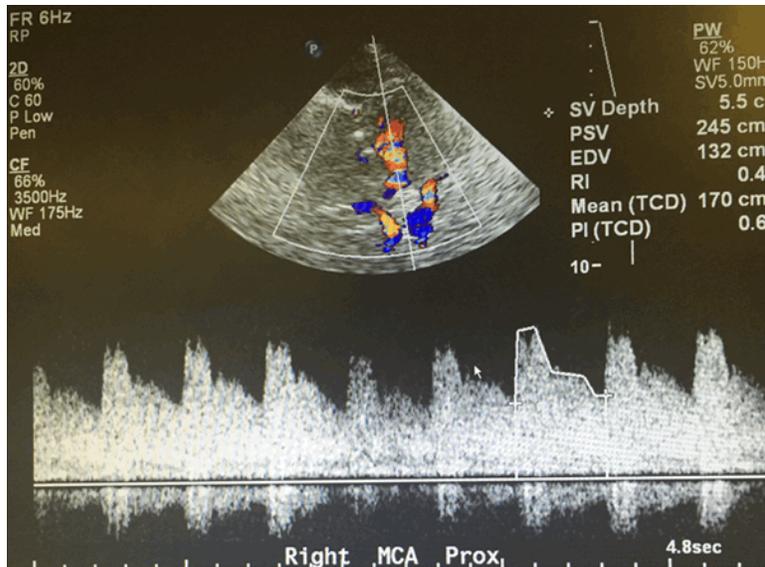
toxic blood pressure was moved to 160-200 mmHg, vasopressors (noradrenaline) were started, and the patient was administered extra sodium chloride to enable positive fluid balance. Forty-eight hours after the treatment, TCD assessment was repeated, which showed normal PSV in the right MCA. The patient was weaned from the triple-H therapy over 2 days and successfully extubated. On the 11<sup>th</sup> day, he was transferred to general ward prior to returning home.

### Discussion

SAH is a major cause of morbidity and mortality. Twenty-five percent of individuals die within 24 hours of experiencing an SAH, and 40% to 60% of all patients with SAH will die within 1 month [8]. Cerebral vasospasm, the most common complication of SAH, usually develops 3-5 days after SAH, peaks between days 6 and 12, which can be gradual or precipitous, and resolves over a period of 2-4 weeks after the onset of hemorrhage [9-12]. Angiographic vasospasm, as seen on digital subtraction angiography (DSA) and computed tomography angiography (CTA) occurs in up to 50% to 70% of patients of SAH, with about half of them suffering from clinical symptoms [4]. Symptomatic vasospasm is commonly characterized by a global decline of the level of consciousness, or accompanied by focal neurological deficits corresponding to the arterial territories involved. However, sometimes it is difficult to detect the clinical deficits and symptoms of vasospasm in critical patients, which occur only after vasospasm has already manifested significant effects on the brain.

Radiographic findings often precede such clinical signs, and thus offer the opportunity to intervene and prevent neurologic injury. Cerebral angiography is currently the gold standard for the diagnosis of cerebral vasospasm. However, this test is expensive and invasive, and does not allow for bedside evaluation, making it difficult to apply regularly in critical patients. To this effect, since it was first used by Aaslid and colleagues in 1982, TCD has evolved into an effective bedside test to follow the progression of vasospasm. More work has been done on the use of this technology in the evaluation of CBF velocities, due to its relative inexpensiveness, bedside availability, and non-invasive nature [5, 13]. McGirt and his colleagues demonstrated that in about 70% of

## Detection of cerebral vasospasm by TCD



**Figure 2.** The patient's TCD result of MCA on the right side showed mild vasospasm associated with an increased PSV.

SAH patients, cerebral vasospasm was detected with TCD on average 2.5 days before the appearance of delayed neurological deficits [14]. This recommends that earlier monitoring for vasospasm by using TCD could result in earlier interventional treatment, with the potential for improved patient outcomes. In this report, TCD detected the cerebral vasospasm earlier and also facilitated the decision regarding triple-H therapy.

### Conclusion

In summary, in neurocritical patients, TCD is an excellent first-line examination, and has become an integral part of early monitoring in the management of patients with 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: hongzhang-dc@163.com

### References

[1] Marshall SA, Nyquist P and 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.

[2] White H and Venkatesh B. Applications of transcranial Doppler in the ICU: a review. *Intensive Care Med* 2006; 32: 981-994.

[3] Armonda RA, Bell RS, Vo AH, Ling G, DeGraba TJ, Crandall B, Ecklund J and Campbell WW. Wartime traumatic cerebral vasospasm: recent review of combat casualties. *Neurosurgery* 2006; 59: 1215-1225.

[4] Keyrouz SG, Diringner MN. Clinical review: Prevention and therapy of vasospasm in subarachnoid hemorrhage. *Crit Care* 2007; 11: 220.

[5] 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.

[6] Daffertshofer M, Gass A, Ringleb P, Sitzer M, Sliwka U, Els T, Sedlacek O, Koroshetz WJ and Hennerici MG. Transcranial low-frequency ultrasound-mediated thrombolysis in brain ischemia: increased risk of hemorrhage with combined ultrasound and tissue plasminogen activator: results of a phase II clinical trial. *Stroke* 2005; 36: 1441-1446.

[7] Kirsch JD, Mathur M, Johnson MH, Gowthaman G and Scoutt LM. Advances in transcranial Doppler US: imaging ahead. *Radiographics* 2013; 33: E1-14.

[8] Ingall TJ and Whisnant JP. Epidemiology of subarachnoid hemorrhage. In: Yanagihara T, Piepgras DG, Atkinson JLD, editors. *Subarachnoid Hemorrhage*. New York, NY: Marcel Dekker; 1998. pp. 63-78.

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

[10] Kassell NF, Peerless SJ, Durward QJ, Beck DW, Drake CG and Adams HP. Treatment of ischemic deficits from vasospasm with intravascular volume expansion and induced arterial hypertension. *Neurosurgery* 1982; 11: 337-343.

[11] Mayberg MR, Batjer HH, Dacey R, Diringner M, Haley EC, Heros RC, Sternau LL, Torner J, Adams HP Jr, Feinberg W, 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. *Circulation* 1994; 90: 2592-2605.

## Detection of cerebral vasospasm by TCD

- [12] Rasulo FA, De Peri E and Lavinio A. Transcranial Doppler ultrasound in intensive care. *Eur J Anaesthesiol Suppl* 2008; 42: 167-173.
- [13] Bleck TP. Rebleeding and vasospasm after SAH: new strategies for improving outcome. *J Crit Illn* 1997; 12: 572-582.
- [14] McGirt MJ, Blessing RP and Goldstein LB. Transcranial Doppler monitoring and clinical decision making after subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis* 2003; 12: 88-92.