Visualizing Impending Cerebral Circulatory Arrest Caused by Intracranial Hypertension Following Aneurysmal Subarachnoid Hemorrhage

To JNA Readers:

Intracranial hypertension may represent an important complication during the early phase following aneurysmal subarachnoid hemorrhage.[1] Timely diagnosis of intracranial hypertension is essential to avoid secondary brain ischemia; however, intracranial pressure (ICP) monitoring requires the insertion of catheters either within the brain ventricles or parenchyma, and hence, invasive ICP monitoring is not frequently utilized.[2] Transcranial Doppler can be used for noninvasive ICP estimation through calculation of the pulsatility index (PI).[3] We describe a case where noninvasive ICP monitoring with transcranial color-coded Doppler (TCCD) rapidly identified a condition of severe intracranial hypertension, which led to a life-saving treatment.

A 38-year-old man was admitted to the emergency department with a World Federation of Neurological Surgeons (WFNS) score of II (Glasgow Coma Score = 14, no motor deficit) following a brief loss of consciousness. An initial brain computed tomographic (CT) scan showed a subarachnoid hemorrage (Fisher grade 3) with mild hydrocephalus of the ventricular temporal horns (Fig. 1). A brain CT angiogram showed a 5-mm saccular aneurysm at the right A1-A2 anterior cerebral artery junction. Shortly after the admission to the neurosurgical ward, he became restless and agitated with further deterioration of his neurological condition (WFNS = IV, Glasgow Coma Score = 12, mild left upper limb hemiparesis, slight right > left anisocoria with preserved pupillary reactivity to light) and no evidence of seizures. A second brain CT was unchanged, but urgent TCCD of the right middle cerebral artery demonstrated a critically low diastolic blood flow velocity of 18.2 cm/s, yet normal systolic flow velocity of 130 cm/s, indicating prevailing cerebral blood flow during the systolic phase of the cardiac cycle (Fig. 1B). Middle and anterior cerebral arteries were opening during cardiac systole and then rapidly closing during diastole, indicating severe intracranial hypertension with impending cerebral circulatory arrest. (Supplemental Digital Content 1, http://links.lww.com/JNA/A31. Ultrasound Video clearly demonstrating the arteries opening during systole and closing during diastole before external ventricular drain [EVD] placement.) The PI was 2.64, corresponding to an estimated ICP of 40 mm Hg. An EVD was immediately placed, which showed an ICP reading of 35 mm Hg measured after rapid loss of 5 mL of cerebrospinal fluid during the procedure. Following further drainage of 20 mL, ICP reduced to a value of 10 mm Hg. The second TCCD examination showed diastolic flow velocity (41.9 cm/s) and PI (1.05) had returned to normal (Fig. 1C). After EVD placement, the neurological condition rapidly improved with resolution of the neurological deficits within 6 hours. After successful embolization of the aneurysm, the patient was transferred to the neurosurgical ward and after 1 week was eventually discharged to a rehabilitation center where he fully recovered.

The use of TCCD allowed rapid detection of severe intracranial hypertension causing a critical reduction of the cerebral blood flow. Direct visualization of middle and anterior cerebral arteries with use of TCCD showed the vascular lumen closing during the diastolic phase of the cardiac cycle and opening only during the systolic phase. After exclusion of rebleeding, the hydrocephalus appeared to be the likely cause leading to emergent EVD placement. Invasive monitoring confirmed the estimated ICP that was well above established threshold for treatment. Rapid improvement of the neurological condition after cerebrospinal fluid drainage definitely confirmed the diagnosis of hydrocephalus-associated intracranial hypertension.

Intracranial hypertension due to rebleeding and hydrocephalus occurs in nearly 50% of aneurysmal subarachnoid hemorrhage patients, including those presenting with good clinical grades, and can be associated with secondary cerebral infarction and worsening of outcome.[4] However, ventricular volume may not always correlate with increased ICP causing delays in decision making. In fact, as shown in this case, acute hydrocephalus may lead to a critical reduction of cerebral blood flow dispite apparently unchanged cerebral ventricle volumes.

Transcranial Doppler can be used for noninvasive ICP estimation, both through use of PI, indicating intracranial hypertension if above 1, and with estimation of specific ICP values.[5] Importantly, TCCD provides a unique method for directly visualizing major cerebral arteries of the circle of Willis and their acute modification due to intracranial hypertension.

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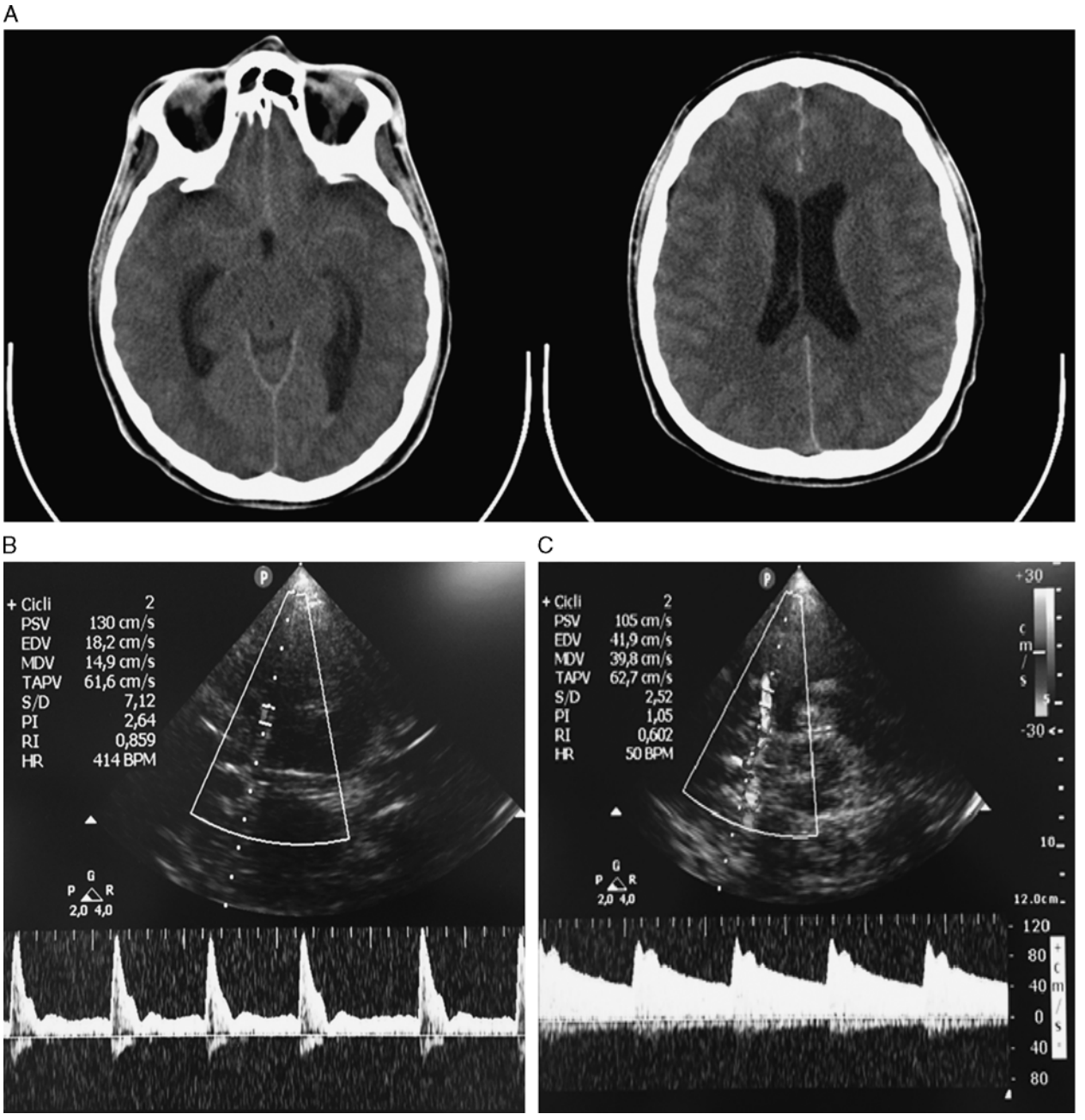

FIGURE 1. A, Bilateral subarachnoid hemorrhage in the cerebral sulci, basal cisterns, perivascular spaces, left posterior ventricular horn and in the third ventricle; initial signs of mild hydrocephalus of the temporal horns. B, Transcranial color-coded Doppler (TCCD) examination showing signs of intracranial hypertension, as demonstrated by the presence of a low diastolic flow velocity of 18.2 cm/s, a pulsatility index (PI) of 2.64, and noninvasive intracranial pressure (ICP) estimation was 40 mm Hg. C, TCCD showing both systolic and diastolic flow velocity returning to normal values after EVD placement, with a net reduction of the PI (1.05) and the noninvasive ICP estimation value (10 mm Hg).

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