

## Cerebral Vasospasm in Intracerebral Hemorrhage-Case Report

### Abstract

**Background:** Cerebral vasospasm is commonly seen in subarachnoid hemorrhage. However the vasospasm in spontaneous intracerebral hemorrhage without subarachnoid extension has not been described.

**Report:** We report a patient who developed intracerebral hemorrhage associated with cerebral vasospasm demonstrated by conventional angiography. The vasospasm involved the superior and inferior divisions of the middle cerebral artery on the side of intracerebral hemorrhage. The vasospasm resolved in six days as documented by a repeat angiography.

**Conclusion:** Cerebral vasospasm can be rarely seen in patients with intracerebral hemorrhage. Further elaboration is required to understand the pathophysiology and subsequent impact on outcome in such patients.

**Keywords:** intracerebral hemorrhage, cerebral vasospasm, angiography, calcium channel blockers

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Intracerebral hemorrhage (ICH) occurs because of bleeding into the parenchyma of brain that may extend into the ventricles. The incidence of ICH varies from 10 to 20 cases per 100,000 population.<sup>1</sup> Each year, approximately 37,000 to 52,400 people in the United States have an ICH.<sup>2,3</sup> Advancing age and hypertension are the most important risk factors for ICH.<sup>3</sup> Cerebral vasospasm is common in subarachnoid hemorrhage (SAH). Regional cerebral blood flow changes have been noted in ICH as a direct effect of hematoma, however, there is no description of cerebral vasospasm.<sup>4</sup> We report a case of cerebral vasospasm associated with ICH which resolved spontaneously.

### Case Report

A 47 year old man presented with sudden onset of slurred speech and right sided weakness. On initial examination his blood pressure was 165/110 mm of Hg and pulse of 77 beats per minute. His neurological examination showed right sided hemiparesis with decreased sensory perception. He had a five years history of hypertension and 10 pack year history of cigarette smoking. He was non-compliant with his antihypertensive medications. No history of illicit drug use or sympathomimetic agents was elicited. His computed tomographic (CT) scan of the head (as shown in Figure 1a and 1b) showed left putaminal ICH with no evidence of ventricular extension or subarachnoid hemorrhage. Because of his young age, he underwent a cerebral angiogram to determine the etiology of hemorrhage. Angiogram did not reveal any underlying etiology of hemorrhage, however, it demonstrated moderately severe vasospasm in the superior and inferior divisions (M2 segments of left middle cerebral artery). (Figures 2a and 3a). He was started on nimodipine 60 mg every 4 hours orally. He was also started on hydrochlorothiazide and enalapril to manage his hypertension. A repeat angiogram was done on day 6 which showed complete resolution of vasospasm. (Figures 2b and 3b). Nimodipine was discontinued. His neurological deficit had completely resolved at the time of discharge and his blood pressure was well controlled.

### Discussion

ICH is a medical emergency with frequent early neurological deterioration or death.<sup>3</sup> CT of the head is imaging procedure of choice in the initial evaluation of suspected ICH, and angiography should be considered for all patients without a clear cause of hemorrhage or those who are surgical candidates, particularly young, normotensive patients who are clinically stable.<sup>3</sup> The cerebral angiogram revealed an unexpected finding of vasospasm ipsilateral to the ICH. Vasospasm has not been previously reported with primary ICH without subarachnoid hemorrhage. We treated the vasospasm using oral nimodipine. His vasospasm completely resolved by day 6 and we discontinued the nimodipine. Patient did not develop any subsequent neurologic deterioration.

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Figure 1a. CT scan of head showing large ICH with normal subarachnoid spaces.



Figure 1b. A more caudal transaxial section of CT scan in the region of cerebral peduncles and quadrigeminal cistern showing the absence of subarachnoid blood.

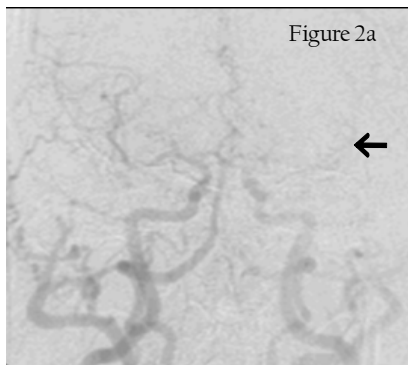
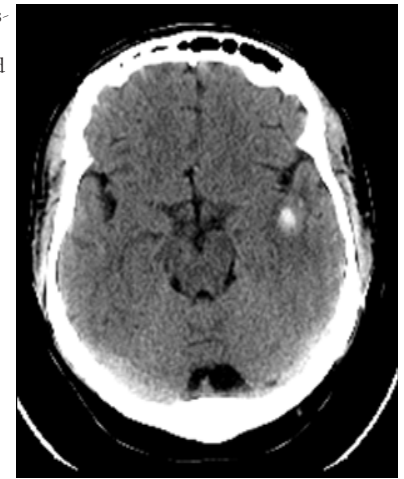


Figure 2a

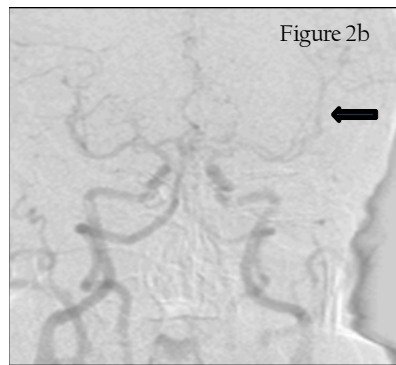


Figure 2b

Figure 2a. Anteroposterior projection of an arch injection, showing decreased perfusion in the distal branches of left middle cerebral artery.

Figure 2b. Anteroposterior projection of an arch injection on day 6, showing improved perfusion in the distal branches of middle cerebral artery.

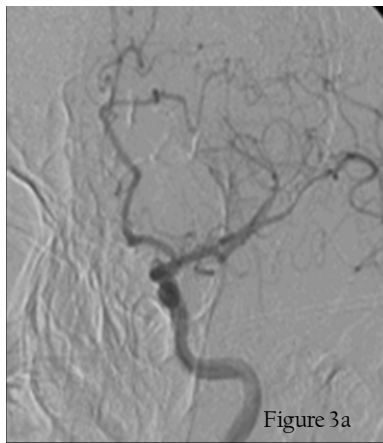


Figure 3a



Figure 3b

Figure 3a. Caldwell oblique view of left ICA injection showing vasospasm in the M2 segment of left middle cerebral artery.

Figure 3b. Caldwell oblique view of left ICA injection on day 6, showing resolution of vasospasm in the M2 segment of middle cerebral artery.

Qureshi et al.<sup>4</sup> have described three phases of changes of cerebral blood flow (CBF) in ICH. The acute period of first 48 hours called the hibernation phase is characterized by a region of hypoperfusion, predominantly in the periphery of hematoma with concomitant reduction in cerebral metabolism. The reperfusion phase starts approximately 48 hours after the onset of ICH. There is an accompanying increase in metabolic activity in previously hypometabolic regions. After approximately 14 days, CBF approaches normal values. Normalization is attributed to resolution of the hematoma and its mass effect. There are no reports of cerebral vasospasm associated with primary ICH. Symptomatic vasospasm has been reported in 22% to 40% of patients with SAH.<sup>5</sup> Symptomatic cerebral vasospasm is a major cause of

morbidity and mortality in patients with aneurysmal SAH. The pathogenesis of cerebral vasospasm is poorly understood. Various hypotheses have been postulated as the possible mechanisms for vasospasm.<sup>6-9</sup> These include the contraction of cerebral arterial smooth muscle cells and impairment of vasodilatory activity due to prostacyclin/thromboxane A<sub>2</sub> imbalance or oxyhemoglobin induced inhibition of acetylcholine-mediated vasodilation. Proliferative vasculopathy has also been postulated as the possible mechanism of vasospasm. Immunoreactive and inflammatory processes are other postulated contributors to vasospasm.<sup>6</sup> The vasospasm results in increased cerebral vascular resistance and decrease in CBF which results in tissue ischemia. Transcranial Doppler ultrasound (TCD) reliably detects vasospasm,<sup>5,10</sup> however, cerebral angiography remains the gold standard for evalu-

ation of vasospasm.<sup>5,11</sup> Cerebral angiography is particularly recommended when clinical deterioration fails to respond to aggressive medical therapy as the symptomatic vessel may be amenable to endovascular therapy.<sup>5</sup> Symptomatic vasospasm risk index has been proposed to identify patients at risk of vasospasm early in the course of SAH.<sup>12</sup> Calcium channel antagonists reduce the proportion of patients with poor outcome and ischemic neurologic deficits after aneurysmal SAH. Nimodipine is widely used calcium channel antagonist in aneurysmal SAH.<sup>13,14</sup>

Our patient did not have any evidence of SAH. This case suggests that cerebral vasospasm is not exclusive to subarachnoid hemorrhage and can be seen in spontaneous ICH. The time course of onset and resolution of vasospasm was different from that expected in SAH. Our patient did not have any neurological worsening after his initial symptoms and hence we conclude that his vasospasm was asymptomatic. We treated the patient with oral nimodipine for few days and it is unclear whether the vasospasm resolved spontaneously or as a result of administration of nimodipine. After this report, it is possible that more cases of vasospasm associated with ICH will be identified and reported, which will help in better understanding of the pathophysiology of ICH.

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