

Case report

Two cases of delayed perforating artery infarction adjacent to intracranial hemorrhage



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Dear Editor,

Acute intracerebral hemorrhage (ICH) is rarely complicated by concomitant cerebral infarction. Some studies have reported observing new ischemic lesions together with ICH, but mainly as subclinical lesions located in different vascular territories [1,2]. However, to the best of our knowledge, symptomatic perforating artery infarction adjacent to an ICH has not been reported previously. We present herein the first descriptions of delayed perforating artery infarction adjacent to hematoma and discuss the possible pathophysiological mechanisms.

1. Case 1

The patient was a 52-year-old man who complained of sudden-onset right hemiplegia. He had a past history of hypertension, and blood pressure on admission was 199/122 mmHg. No abnormalities of the cranial nerves were apparent. Manual muscle testing (MMT) score was 3 on the right upper extremity and 4 on the right lower extremity. Neither sensory impairment nor cerebellar ataxia were observed. The patient displayed normal reflexes in all four limbs, and showed no pathological reflexes or pyramidal tract signs. National Institutes of Health Stroke Scale (NIHSS) score on admission was 4. The initial magnetic resonance imaging (MRI) revealed intracranial hemorrhage in the left putamen (Fig. 1A). Vasospasm in the proximal large vessels was not detected on MR angiography (data not shown). Arrhythmia was not detected on the electrocardiogram. Routine blood tests revealed a high

level of low-density lipoprotein (144 mg/dl) without any other abnormalities in blood cell count, serum protein, liver function markers, coagulation markers, or vitamins. In addition, tests for autoimmune disorders such as anti-SS-A/SS-B antibodies and proteinase-3 (PR3) – / myeloperoxidase (MPO)-ANCA yielded negative results. Based on these findings, we diagnosed hypertensive ICH. He was subsequently treated with intravenous nicardipine and glycerol, but developed a decline in neurologic status on hospital day 3, with MMT score in the right distal extremity worsening from 3 to 1. Diffusion-weighted imaging (DWI) revealed new cerebral infarction along with perforating artery adjacent to the hematoma (Fig. 1B).

2. Case 2

A 77-year-old man presented with sudden-onset left hemiparesis. He had a past history of hypertension, and blood pressure in the emergency room was 206/99 mmHg. He showed mild dysarthria, left facial palsy, and left hemiparesis (MMT: 4/5). NIHSS score was 4 on admission. Routine blood tests revealed a low level of high-density lipoprotein (29 mg/dl), without any other abnormalities including blood cell count, serum protein, or coagulation markers. Initial MRI showed intracranial hemorrhage in the right putamen (Fig. 1C). After we diagnosed hypertensive ICH, anti-hypertensive therapy and intravenous glycerol were initiated. On hospital day 5, the patient experienced secondary neurological deterioration, showing left complete hemiplegia. DWI revealed acute perforating artery infarction adjacent to the ICH (Fig. 1D).

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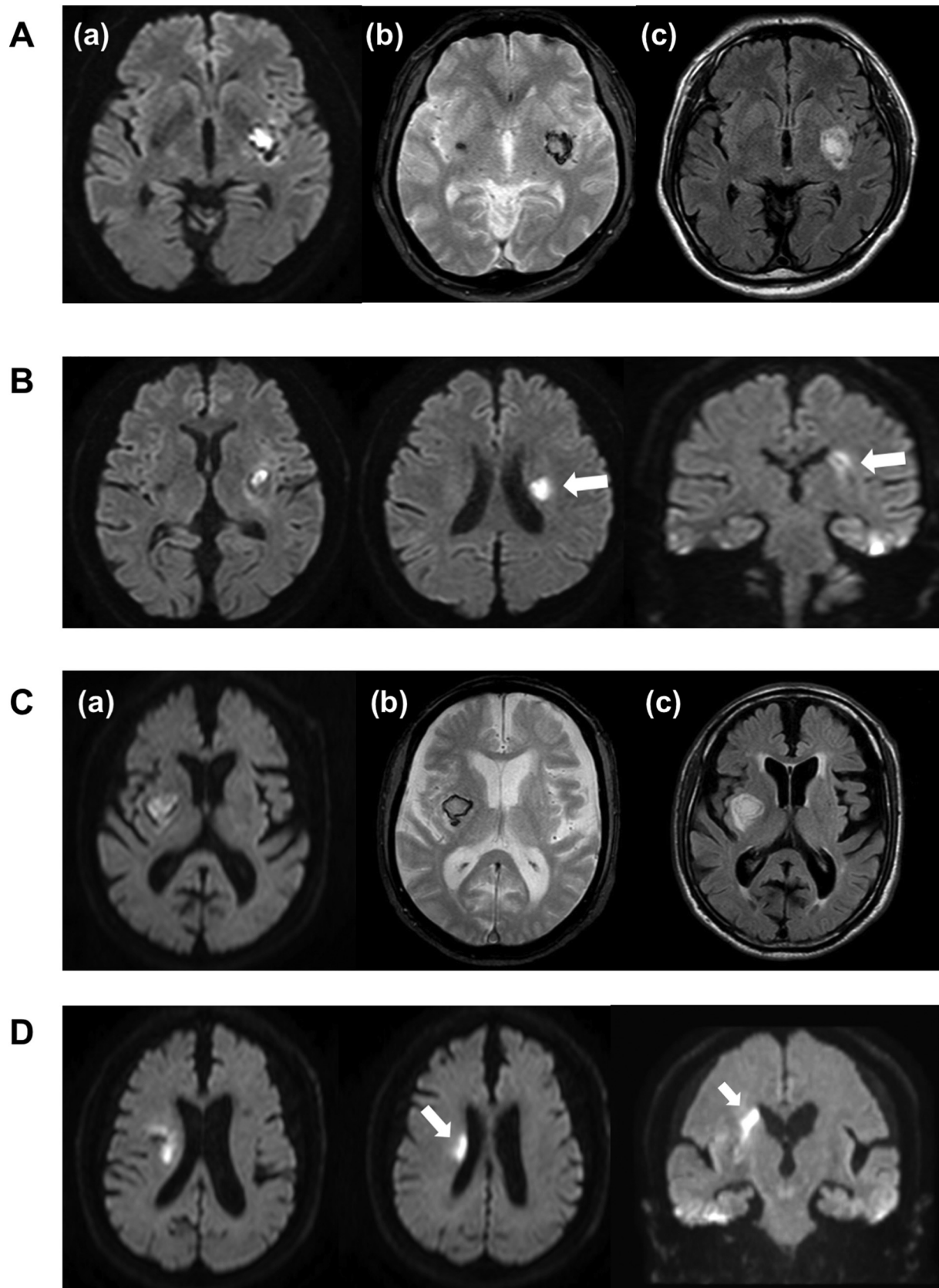


Fig. 1. A) Brain magnetic resonance imaging (MRI) on admission of Patient 1 shows intracranial hemorrhage in the left putamen. a) Diffusion-weighted image (DWI); b) T2*-weighted image; and c) Fluid-attenuated inversion recovery image (FLAIR). B) DWI of Patient 1 on hospital day 10. White arrows indicate acute corona radiata infarction adjacent to the hematoma. C) Brain MRI on admission of Patient 2 demonstrates intracranial hemorrhage in the right putamen. a) DWI; b) T2*-weighted imaging; c) FLAIR imaging. D) DWI of Patient 2 on hospital day 8. White arrows indicate acute corona radiata infarction adjacent to the hematoma.

3. Discussion

We report a novel clinical manifestation of symptomatic perforating artery infarction adjacent to hypertensive ICH, which was detected on DWI.

A previous study reported cases of large ischemic stroke due to mechanical compression of the ICH against large vessels [3]. In addition, a previous cross-sectional study demonstrated acute cerebral infarctions in more than one-fifth of spontaneous ICH patients. The majority of infarct lesions in ICH patients were subcentimeter, subcortical, and subclinical [2]. However, details of positional relationships between hematoma and cerebral infarctions remain elusive. Moreover, symptomatic perforating artery infarction adjacent to ICH has not been reported previously. Here, we provided the first two reports of delayed perforating artery infarction adjacent to ICH.

Possible mechanisms of cerebral infarction in the present cases were as follows: first, the hematoma itself and/or perihematomal edema could theoretically compress the adjacent perforators, causing cerebral ischemia. In line with this hypothesis, previous experimental studies have demonstrated decreased cerebral blood flow after ICH was associated with a mass effect, leading to cerebral ischemia in rats [4]. Second, cerebral vasospasm may have been induced in the perforating artery in our cases. Cerebral vasospasm is frequent after subarachnoid hemorrhage (SAH), resulting in severe complications [5]. However, cerebral vasospasm is not exclusive to SAH, and has been reported in a patient with ICH [6]. In patients with ICH, blood comes into direct contact with the cerebrospinal fluid and/or diffuses along the Virchow-Robin perivascular spaces. Hemoglobin degradation products also inhibit endothelial relaxation in the cerebral arteries, leading to

vasoconstriction [5]. Finally, aggressive lowering of blood pressure beyond the lower limits of cerebral autoregulation might induce cerebral ischemia in hypertensive ICH patients.

In conclusion, the present cases demonstrated that ICH can be followed by perforating artery infarction adjacent to the hematoma. Although further accumulation of reports is warranted to determine whether and how ICH induces perforating artery infarction, blood pressure should be lowered with care during the acute phase of ICH.

Declaration of Competing Interest

The authors declare no conflicts of interest.

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