

# Vessel Wall Magnetic Resonance Imaging in a Case of Post-traumatic Multifocal Striatocapsular Hemorrhagic Infarction

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## Abstract

Post-traumatic striatocapsular infarction is extremely rare and has been described only within the vascular territory of the perforating arteries originating from the middle cerebral artery (MCA). We recently encountered a patient presenting with unilateral multifocal striatocapsular hemorrhagic infarctions following mild head injury. This 25-year-old female was admitted to our trauma center after a motorcycle accident. Initial brain computed tomography and magnetic resonance (MR) imaging showed multifocal acute hemorrhagic infarctions with a clustering in the right caudate head, anterior limb of internal capsule, and globus pallidus. MR angiography and digital subtraction angiography showed suspicious luminal irregularities of the lenticulostriate arteries of the right MCA. Vessel wall MR images (VWI) did neither indicate intramural hematoma nor wall enhancement in the right MCA, suggesting dissection. However, VWI showed the passages of each lenticulostriate artery supplying each infarction site. Therefore, based on both conventional images and VWI, we postulate that this patient's post-traumatic multifocal striatocapsular hemorrhagic infarctions were caused by damage to multiple lenticulostriate arteries.

Key words: craniocerebral trauma, cerebral infarction, hemorrhage, magnetic resonance image, basal ganglia

## Introduction

Trauma can cause multiple vascular complications in and about the head. Some of these complications are well understood, such as occlusion or dissection of the vertebral and carotid arteries.<sup>1,2</sup> Stroke following head trauma, especially in the basal ganglia, is rare. More typical are large or small hemorrhagic lesions, caused by shearing forces in moderate-to-severe head injury by acceleration and/or deceleration forces.<sup>3</sup> Traumatic basal ganglia hemorrhagic lesion is also relatively rare, observed in approximately 0.9–3.0% of head injury cases.<sup>3,4</sup> Post-traumatic striatocapsular infarction is extremely rare and has only been described within the vascular territory of the perforating arteries originating from the middle cerebral artery (MCA).<sup>5–7</sup> In addition, we know little about post-traumatic hemorrhagic infarction of the lenticulostriate artery.

We recently encountered a patient presenting with unilateral multifocal striatocapsular hemorrhagic infarctions confined to the respective territories of multiple lenticulostriate arteries. The patient was diagnosed using conventional images and vessel wall magnetic resonance (MR) images (VWI) after mild head injury from a motorcycle accident. Herein, we present this case with unilateral multifocal striatocapsular hemorrhagic infarctions after mild head injury, along with discussion of the pathogenesis of this infarction. This case report had IRB approval (IRB number H-1810-095-001).

## Case Presentation

A 25-year-old female entered our trauma center with complaints of pain in her right leg and left wrist and hand. The patient was driving a motorcycle and injured in an accident with a car. During her initial hospital visit, she was diagnosed with open fracture of the right femur shaft and multiple closed fractures to the left ulnar, radius, scaphoid, and hamate, and a focal hemorrhage in the right basal ganglia.

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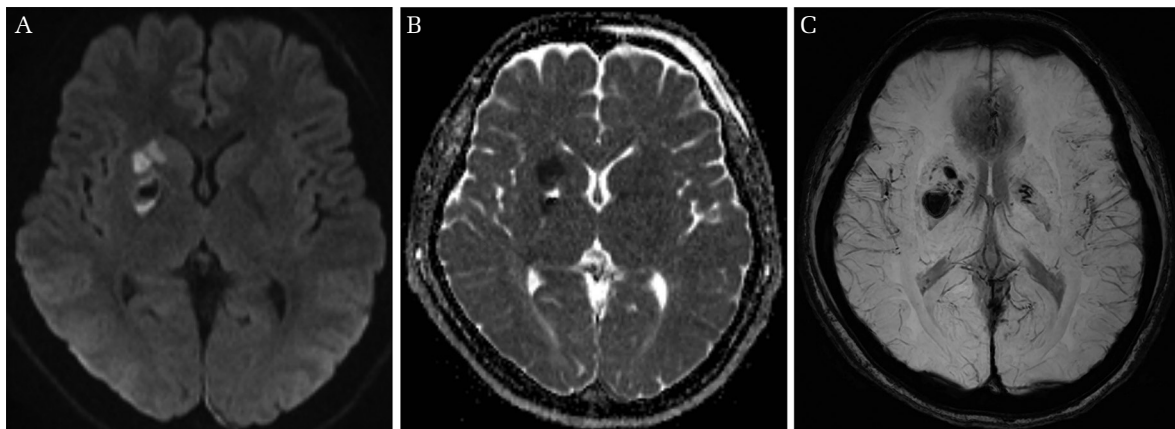
After diagnosis, she was referred to our hospital for comprehensive trauma management. On neurologic examination, she demonstrated alert mentality, right leg immobility due to the femur fracture, and left hand numbness. She demonstrated no neurologic deficit except mild weakness and numbness in her left hand. She showed mild scalp swelling in the left frontal area and no skull fracture.

Focal acute hemorrhage in the right basal ganglia was observed on initial brain computerized tomography (CT) performed on 2 h post-accident (Fig. 1). Brain MR imaging with diffusion-weighted and apparent diffusion coefficient map (Figs. 2A and 2B) performed on 14 h post-accident showed a few acute

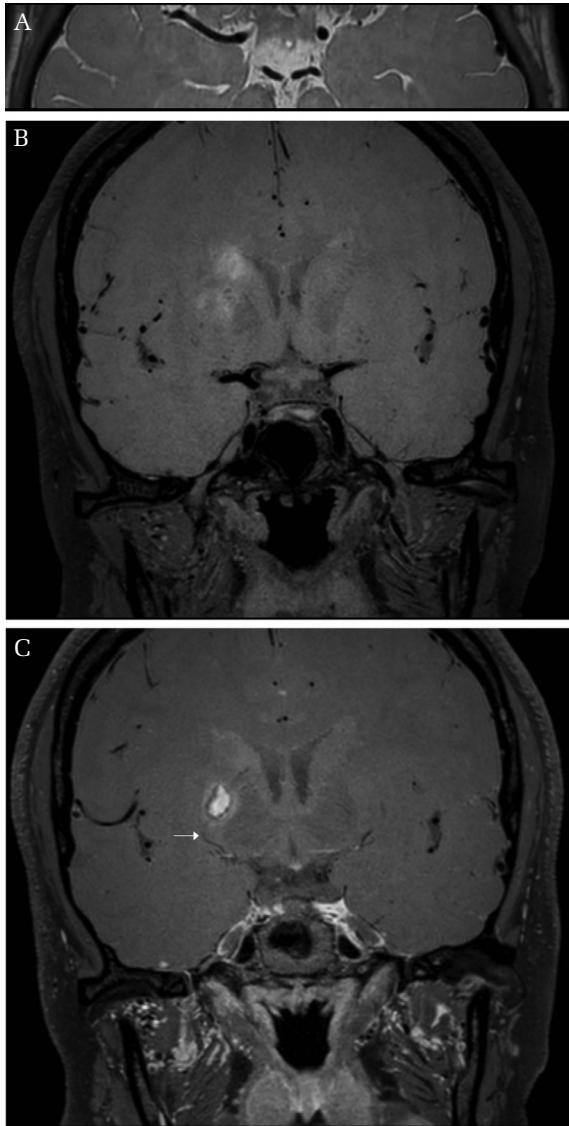


**Fig. 1** Non-contrast brain CT showing focal acute hemorrhage in the right basal ganglia.

focal infarctions and hemorrhagic infarctions in the right caudate head, the anterior limb of the internal capsule, and the globus pallidus. Susceptibility-weighted images (SWI), MR angiography, and VWI were performed at 4 and 19 days following the trauma. 3T MR imaging was performed (Achieva 3.0T, Philips Healthcare, Best, The Netherlands) with a 32-channel coil. VWI was composed of 3D proton density weighted images and both pre- and post-contrast-3D blood-suppression fast spin echo T<sub>1</sub>-weighted images. Voxel size was 0.6 mm isotropic dimension for 3D acquisitions, and peripheral pulse gating was applied during scans. There was no significant difference between the two VWIs, performed 15 days apart. SWI (Fig. 2C) shows multifocal small hemorrhages with clustering in the right basal ganglia area. The M1 segment in the right MCA does neither show stenosis, wall hematoma, nor wall enhancement suggesting dissection on VWI (Fig. 3). Each lenticulostriate artery running on the acute infarction or hemorrhagic infarction area can be seen on VWI, suggesting striatocapsular infarctions of trauma-based stroke type. However, VWI was unable to demonstrate directly any thrombus or rupture of the lenticulostriate arteries in the right MCA. Intracranial time of flight MR angiography (Fig. 4A) and digital subtraction angiography (DSA) (Fig. 4B) show suspicious irregularity of the lenticulostriate arteries in the right MCA. The patient underwent reduction and internal fixation for multiple fractures in her right femur and left ulnar, radius, and scaphoid. Her left hand weakness and numbness improved with acute management of the hemorrhagic stroke and trauma; 1 month later, she showed no neurologic deficit.



**Fig. 2** Diffusion-weighted image (A) and apparent diffusion coefficient map (B) on 14 h post-accident show two focal acute infarctions in the right caudate head and anterior limb of the internal capsule, and a focal hemorrhagic infarction in the right globus pallidus. Susceptibility-weighted image on 4 days post-accident (C) shows multifocal small hemorrhages in the right basal ganglia area.



**Fig. 3** High-resolution vessel wall MR images on 19 days post-accident show no evidence of dissection in the right MCA. Axial reconstruction of the proton-density image (A) shows no stenosis of the M1 segment in the right MCA. Coronal precontrast  $T_1$ -weighted image ( $T_1$ WI) (B) shows no dissecting hematoma, intimal flap, or wall enhancement at the M1 segment in the right MCA. Coronal postcontrast  $T_1$ WI (C) demonstrates the lenticulostriate artery continuing the hemorrhagic infarction in the right basal ganglia (arrow: lenticulostriate artery).

## Discussion

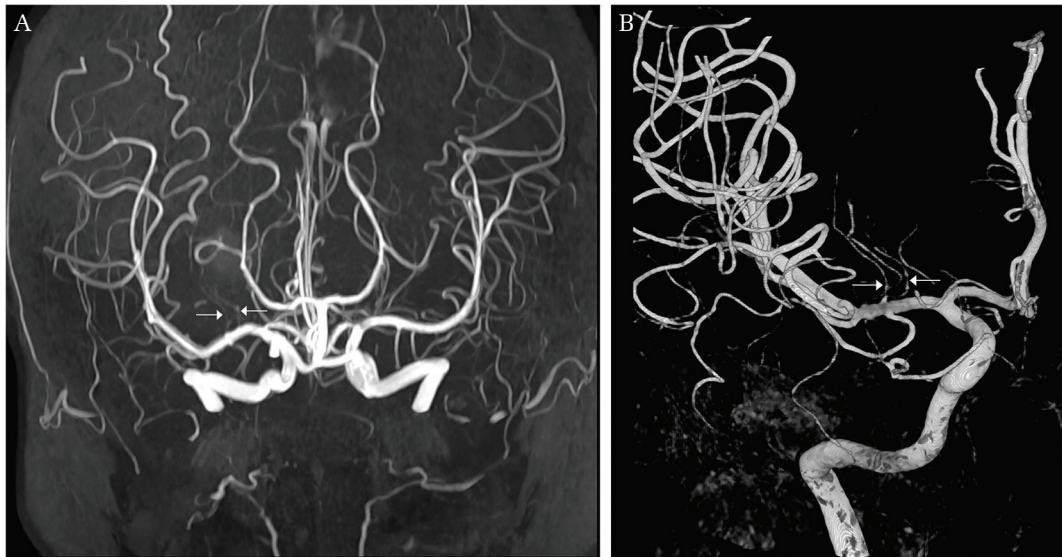
Common image findings in moderate-to-severe traumatic brain injury include skull fractures, shearing injuries, hemorrhages, and infarction/edema. In contrast, post-traumatic cerebral infarction is a relatively rare consequence of head injury; its frequency, causes, and influences on mortality are

not well understood.<sup>3)</sup> Occlusion, spasm, emboli, dissection, and thrombus formation in the major intracranial arteries have all been considered as causes of infarction.<sup>8–10)</sup> Compression and distortion of the brain secondary to brain herniation and increased intracranial pressure may also induce focal ischemic necrosis areas.<sup>11)</sup> Despite this, we identified neither brain herniation nor increased intracranial pressure in this patient. Furthermore, basal ganglia hemorrhagic infarction after mild head injury is rare.

In our case, both MR angiography and DSA showed no abnormalities of the main intracranial arteries, except for suspicious luminal irregularities of the lenticulostriate arteries in the right MCA. The VWIs showed neither intramural hematoma, intimal flap, nor wall enhancement of the MCA, suggesting MCA dissection. On brain MR images and VWI, these cerebral infarctions did not merge; rather, the regions of each lenticulostriate artery are distinct. Therefore, these findings suggest that a few ischemic or hemorrhagic infarctions in the basal ganglia might have been caused by injuries to the affected perforating arteries, not by dissection of the MCA.

It is difficult to diagnose intracranial arterial dissection with conventional luminal imaging such as CT angiography, MR angiography, or DSA. However, intracranial VWI is a useful adjunct to conventional imaging for clinical evaluation of arterial dissection.<sup>12,13)</sup> Although intracranial arterial dissection is not always visible on VWI, the possibility of dissection cannot be ruled out when imaging findings suggesting dissection are not shown on VWI. In our case, a hemorrhagic infarction is more likely, caused by multiple damages to the lenticulostriate artery rather than by an MCA dissection, given the lack of imaging findings suggesting such dissection. On the VWI (Fig. 3), it is apparent that the clustering shape is not from a large confluent infarction but instead from multifocal infarctions. This shape provides indirect evidence that the damage was not from MCA dissections but rather from multiple damages to the lenticulostriate artery. Nor could we identify any other injury to the right MCA territory except for that in the basal ganglia area.

The exact mechanisms of cerebral infarctions in the deep gray matter following head injury remain unclear. The pathogenesis of posttraumatic occlusions of the cerebral artery can be divided into four lesion types:<sup>6,14,15)</sup> emboli from the cervical portion of the carotid artery, vasospasm, traumatic dissection, and posttraumatic thrombosis. Shaffer et al.<sup>16)</sup> have suggested that intermittent arterial spasm might result in recurrent episodes of transient, though reversible,



**Fig. 4** Both MR angiography (A) and digital subtraction angiography (B) demonstrate suspicious focal luminal irregularities (arrows) of multiple lenticulostriate arteries in the right MCA.

hemiparesis following head trauma. Cerebral vasospasm after blunt head trauma is an important secondary posttraumatic insult, which results in thickening of the intima and folding of the internal elastic lamina.<sup>9,17,18)</sup> However, in our case, the hemorrhagic infarction developed within 24 h after trauma. In particular, the right basal ganglia hemorrhage was identified 2 h post-accident. Ischemic infarction was identified on MR image within 14 h after the mild head injury. Thus, arterial spasm might not be the mechanism of hemorrhagic infarction in this patient.

Impact directed toward the tentorium may shift the brain through the tentorial notch, leading to vessel pulling and tearing, which may cause hemorrhage in the basal ganglia.<sup>4)</sup> The basal ganglia and internal capsule are supplied by the lenticulostriate branches of the MCA, which form an acute angle at their origin from the main trunk of the MCA. The lateral perforators are more acute and shorter compared with the medial. The mobile segment of these vessels between the two fixed ends (proximal to the MCA and distal to the brain parenchyma) is vulnerable to any sudden movement induced by stretching or trauma to the intima, which results in vasospasm or thrombosis.<sup>14–16)</sup> Younger patients have a smaller subarachnoid space in the proximal Sylvian fissure compared with older patients, who are more likely to have atrophy. This characteristic of young patients may make them more vulnerable, compared with older patients, to lenticulostriate artery injury after sudden brain movement. A wider subarachnoid space in older patients can function as an absorption space for impact energy. In Fig. 4, the lenticulostriate artery has proximal luminal irregularities that are absent in the distal portion. In this patient,

we were unable to identify a subarachnoid hemorrhage. These characteristics may indicate movement between the MCA main trunk in the subarachnoid space and the proximal lenticulostriate artery in the brain parenchyma. This may have resulted in tear and distortion of the lenticulostriate artery in the brain parenchyma. In addition, these vessels are functionally end arteries, so that ischemia is readily precipitated once they are injured. Sudden movement of the brain may mechanically disrupt the distal blood supply, or lead to intimal trauma and subsequent thrombosis. Therefore, hemorrhagic infarction may result from indirect injury of the lenticulostriate artery.

## Conclusion

In this case, a clustering of multifocal striatocapsular hemorrhagic infarctions following the patient's motorcycle accident may have resulted from multiple perforating artery damages from trauma-associated shearing injury. These were discovered using conventional imaging and VWI, the latter of which can be useful for identifying the mechanism of injury in brain trauma.

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## Conflicts of Interest Disclosure

The authors report no conflicts of interest concerning the materials and methods in, or results from, this study.

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