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Case Report

Sequential Development of Putaminal Hemorrhage and Corona Radiata Infarction in the Same Lenticulostriate Arterial Territory

Yuki Inomata[§], Yoshiki Hanaoka[§*], Yu Fujii, Toshihiro Ogiwara, and Tetsuyoshi Horiuchi

Department of Neurosurgery, Shinshu University School of Medicine, Matsumoto, Nagano 390-8621, Japan

Putaminal hemorrhage is a common type of spontaneous cerebral hemorrhage. However, to our knowledge, there have been no reports of sequential cerebral hemorrhage and infarction in the same perforating arterial territory. Herein, we present the first reported case of the sequential development of putaminal hemorrhage and corona radiata infarction in the same lenticulostriate arterial (LSA) territory. Early intensive blood pressure lowering treatment may have aggravated ischemic damage. If a patient presents with motor weakness that cannot be explained by putaminal hemorrhage, the sequential development of corona radiata infarction in the same LSA territory should be ruled out.

Key words: arterial dissection, cerebral hemorrhage, cerebral infarction, lenticulostriate artery, perforating artery

Putaminal hemorrhage is the most frequent type of spontaneous cerebral hemorrhage [1]. Dissection of the lenticulostriate artery (LSA) has been identified as a main pathological cause of putaminal hemorrhage [2]. Cerebral arterial dissections in major cerebral arteries, including the internal carotid artery, anterior cerebral artery, middle cerebral artery, and vertebral artery, can lead to simultaneous hemorrhagic and ischemic stroke [3-5]. Nevertheless, to the best of our knowledge, no cases of the sequential development of cerebral hemorrhage and infarction in the same perforating arterial territory have yet been reported in the literature. Herein, we present the first reported case of confirmed sequential putaminal hemorrhage and corona radiata infarction in the same LSA territory.

Case Report

An 82-year-old woman presented to our institution with sudden right hemiparesis. The patient had a history of hypertension but no history of smoking, alcohol consumption, diabetes mellitus, dyslipidemia, stroke, or coronary artery disease. On admission (Day 1), neurological examination revealed that the right hemiparesis was moderate in the upper extremity and mild in the lower extremity. Computed tomography (CT) revealed a small hemorrhage confined within the left putamen (hematoma volume, 3.7 mL), but the left posterior limb of the internal capsule and corona radiata were intact (Fig. 1A). Early intensive blood pressure lowering treatment (target systolic blood pressure, < 140 mmHg) using intravenous nicardipine was started. On Day 2, the right hemiparesis worsened to flaccid right hemiplegia. CT showed that the hematoma size was unchanged, but the left corona radiata adjacent to the hematoma had become a slightly low-density area, which seemed to be perifocal edema around the hematoma (Fig. 1B). Intravenous glycerol was administered, but did not relieve this symptom. On Day 6, magnetic resonance imaging (MRI) showed acute cerebral infarc-

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*Corresponding author. Phone: +81-263-37-2690; Fax: +81-263-37-0480
E-mail: hanaoka@shinshu-u.ac.jp (Y. Hanaoka)
[§]These authors contributed equally to this work.

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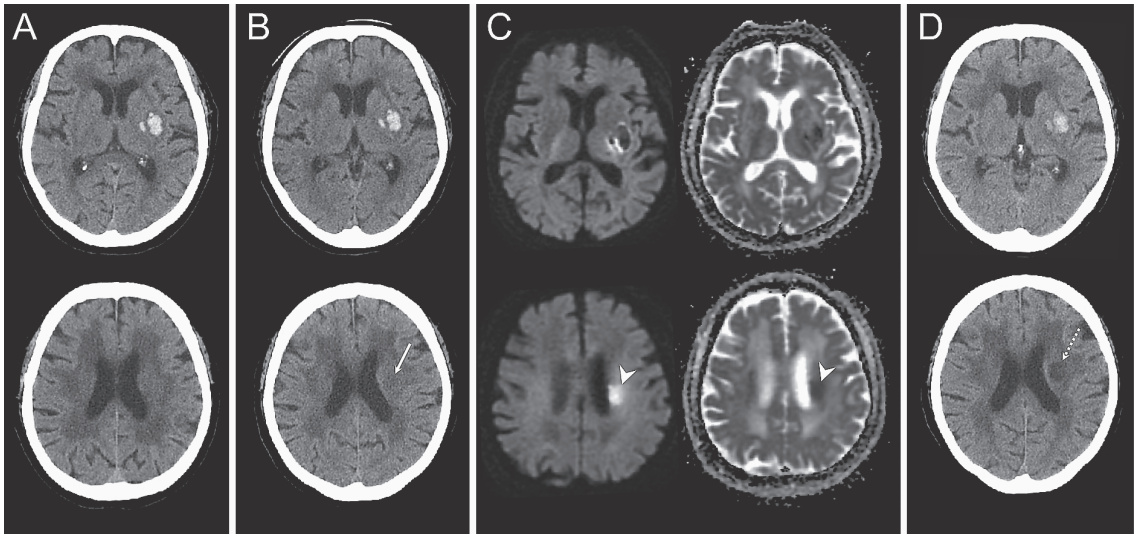

Fig. 1 Chronological findings on neuroimaging. **A**, Computed tomography (CT) on admission (Day 1) showing a small left hemorrhage confined within the putamen; **B**, Follow-up CT on Day 2. The hematoma had not changed in size, but the density of the left corona radiata adjacent to the hematoma had lowered slightly (*arrow*); **C**, Diffusion-weighted imaging (*left*) and apparent diffusion coefficient maps (*right*) on Day 6 revealing acute cerebral infarction in the left corona radiata (*arrowheads*); **D**, Follow-up CT on Day 9 showing a clear low-density area (*dotted arrow*) corresponding to the corona radiata infarction on magnetic resonance imaging.

tion in the left corona radiata corresponding to the low-density lesion on CT (Fig. 1C). High-resolution magnetic resonance angiography and three-dimensional T1 black-blood imaging detected no evidence of vascular abnormalities in the left LSA. We concluded that the right hemiplegia was caused primarily by the corona radiata infarction, not the putaminal hemorrhage. Early intensive blood pressure lowering treatment was abandoned. On Day 9, follow-up CT demonstrated a clear low-density area in the left corona radiata (Fig. 1D). On Day 17, no additional lesions were identified on follow-up MRI. On Day 25, the patient was discharged for rehabilitation for severe and persistent hemiparesis.

Discussion

To the best of our knowledge, no cases in which cerebral infarction developed sequentially with intraparenchymal hemorrhage in the same perforating arterial region have yet been reported in the literature. In the present case, the patient was admitted with moderate right hemiparesis that could not be sufficiently explained neuroradiologically by a small localized left putaminal hemorrhage. MRI on Day 6 revealed corona radiata ischemia adjacent to the small putaminal hemorrhage.

The corona radiata adjacent to the putamen is supplied by the LSAs, long insular arteries (LIAs), and medullary arteries originating from the cortical branches of the middle cerebral arteries (Fig. 2A) [6]. In coronal images at the level of the basal ganglia, LSA infarction extends vertically from the periventricular white matter to the putamen, whereas LIA infarction is horizontally located from the periventricular white matter to the top of the insular cortex or extreme capsule (Fig. 2A) [7]. Medullary artery infarction occurs cranial to the LIA vascular territory (Fig. 2A). In the present case, a coronal image reconstructed from diffusion-weighted imaging is consistent with LSA infarction (Fig. 2B). Due to the anatomical relationship between putaminal hemorrhage and corona radiata infarction, the patient developed putaminal hemorrhage and corona radiata infarction sequentially in the same LSA territory.

Mizutani *et al.* performed a pathological analysis of surgical specimens of LSAs that were compatible with the origin of spontaneous putaminal hemorrhage based on microsurgical findings [2]. Nineteen LSAs were collected from 12 patients. Of these, 15 LSAs were identified as the pathological cause of hemorrhage. The 15 LSAs included 6 arterial dissections, 6 arterial ruptures

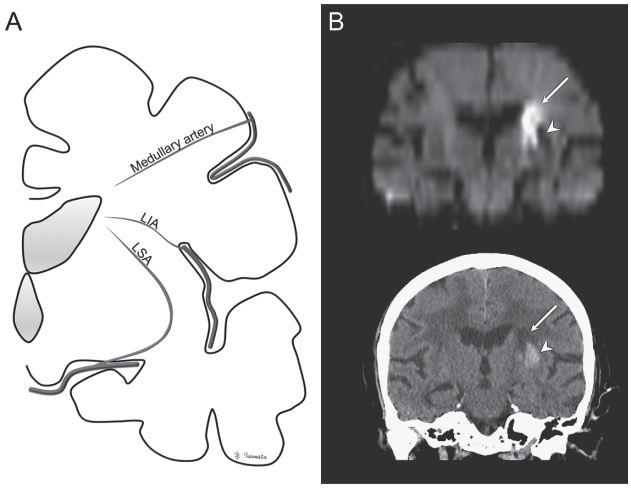

Fig. 2 Schematic diagram showing the vascular territories of the periventricular white matter on a coronal image at the level of the basal ganglia (**A**) and reconstructed coronal images of the present case (**B**). **A**, Lateral lenticulostriate artery (LSA) infarction occurs vertically from the periventricular white matter to the putamen. Long insular artery (LIA) infarction occurs horizontally from the periventricular white matter to the top of the insular cortex or extreme capsule. Medullary artery infarction occurs cranial to the LIA vascular territory; **B**, Coronal images reconstructed from diffusion-weighted imaging (*upper*) on Day 6 and computed tomography (*lower*) on Day 9. The corona radiata infarction (*arrows*) is consistent with the lateral lenticulostriate arterial territory. Considering the anatomical relationship between the putaminal hemorrhage (*arrowheads*) and the corona radiata infarction (*arrows*), we believe that our patient developed hemorrhage and infarction sequentially in the same lateral lenticulostriate arterial territory.

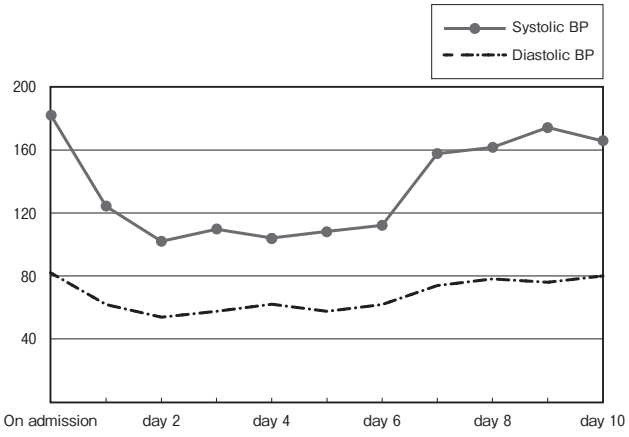

Fig. 3 Line graph showing the course of the patient's systolic/diastolic blood pressure (BP), including her systolic/diastolic BP on admission and average systolic/diastolic BP from Day 1 to Day 9. Early intensive BP lowering treatment was performed from Day 1 to Day 6. Right hemiparesis was moderate in the upper extremity and mild in the lower extremity on admission, but progressed to flaccid right hemiplegia on Day 2.

with substantial degenerative changes, and 3 arterial ruptures with few degenerative changes. The pathological findings in the LSA dissections were similar to those of typical cerebral arterial dissections in major cerebral arteries, which can occasionally cause sequential hemorrhage and/or cerebral infarction [3-5]. Although in our case there was no radiological evidence of any vascular abnormality, LSA dissection might have caused the sequential putaminal hemorrhage and corona radiata infarction. Figure 3 shows the course of blood pressure in the current case. Early intensive blood pressure lowering treatment and decreased local cerebral perfusion pressure due to hematoma may have exacerbated ischemic damage [8]. Considering that LSA dissection is a common cause of spontaneous putaminal hemorrhage, the sequential development of putaminal hemorrhage and corona radiata infarction in the same LSA territory may have been overlooked. In patients with a localized putaminal hemorrhage (*i.e.*, an "intact" pyramidal tract) on CT, the sequential development of corona radiata infarction in the same LSA territory should be ruled out if the patient presents with serious and/or progressive motor weakness. Neurologists and neurosurgeons should be aware that putaminal hemorrhage and corona radiata infarction can occur sequentially in the same LSA territory.

Conclusions

We present the first reported case of the sequential development of putaminal hemorrhage and corona radiata infarction in the same LSA territory. Early intensive blood pressure lowering treatment may have aggravated ischemic damage. If a patient with a localized putaminal hemorrhage develops serious and/or progressive motor weakness, the sequential occurrence of putaminal hemorrhage and corona radiata infarction in the same LSA territory should be ruled out.

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