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Lacunar Infarction Caused by Chronic Subdural Hematoma

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Abstract

In chronic subdural hematoma (CSDH) patients, motor functions usually recover quickly after burr-hole surgery; however, in a rare case, the hemiparesis showed poor improvement after surgery. In that case, investigation of cerebral infarctions is important. Among the 284 CSDH patients with motor weakness, magnetic resonance image (MRI) and MR angiography (MRA) were acquired in 82 patients before surgery when the hemiparesis progressed rapidly. Small lacunar infarction was identified on the hematoma side in five cases; all were older than 80 years with hypertension, and diabetes mellitus had been diagnosed in two. In all the five patients (100%), MRA demonstrated a downward or upward shift of the M1 portion of the middle cerebral artery on the hematoma side, where the perforating arteries originate. Conversely, only 4 CSDH patients (5.2%) without lacunar infarction demonstrated M1 downward shift. The risk factors of lacunar infarction were high in the five detected cases; however, distortion, twisting, or elongation of the lenticulostriate arteries might be a cause of the lacunar infarctions, rather than the formation of lipohyalinosis or microatheroma in the arteries. Therefore, anti-platelet treatment might not be necessary for CSDH-inducing lacunar infarction. The lacunar infarctions caused by CSDH were small, the patients' hemiparesis was mild, a prognosis of all the patients was good, and they recovered well from the motor weakness after physical rehabilitation. MR examinations before surgery are recommended for CSDH patients especially when a patient complains of sudden onset or rapid deterioration of motor weakness.

Keywords: chronic subdural hematoma, lacunar infarction, M1 portion, middle cerebral artery, lenticulostriate artery

Introduction

Lacunar infarction is the most common type of cerebral stroke occurring in the subcortical regions of the brain.[1)] The usual mechanism of lacunar infarction is considered to be the occlusion of small deep penetrating arteries by the formation of lipohyalinosis or microatheroma,[2,3)] and hypertension, diabetes mellitus (DM), and aging are the known risk factors.[4-7)] Another causes of lacunar infarction are intracerebral hemorrhage, multiple sclerosis, an unruptured basilar aneurysm, CSDH, brain abscess, glioblastoma, or Chiari-malformation type I.[8,9)] Anzalone et al. reported that, among 97 patients with lacunar infarction, one patient had CSDH,[8)] and Lazzarino et al. mentioned that lacunar infarction was caused by CSDH in one patient among 137 consecutive lacunar infarction patients.[9)] But neither of them clarify the mechanism of lacunar infarction caused by CSDH. In this study, the authors demonstrate five cases with lacunar infarction caused by CSDH, and consider mechanism for lacunar infarction based on MRI and MRA findings.

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Materials and Methods

Ethic approval

Teikyo University Ethics Committee approved this study (Teirin 13-251), and Teikyo University Conflicts of Interest Management Committee concluded there were no conflicts of interest in this study (TU-COI 13-1606).

Patients

From April 2009 to March 2018, burr-hole surgery was performed in 358 CSDH patients, and motor weakness was identified in 284 patients (79.3%) before surgery. The diagnosis of CSDH was achieved by computed tomography (CT) scan. When the hemiparesis occurred suddenly or progressed rapidly within 3 days (72 hours), MRI and MRA were performed to identify ischemic lesions before surgery.

Evaluation

The patients' age, previous history of lacunar infarction, hypertension, diabetes mellitus, thickness of subdural hematoma, existence of fresh cerebral infarctions on diffusion-weighted image (DWI), and shift of M1 portion of MCA in the hematoma side were analyzed. And relation of lacunar infarction to M1 shift was evaluated. The article defining M1 shift is not found. As shown in Fig. 1A, when distance between petrosal portion of internal carotic artery (ICA) and M1M2 segment of MCA was equal in the left and the right sides, no M1 shift was identified. When the distance was shorter in hamatoma side, M1 downward shift was defined, which is correlated with hammock MCA mentioned by Hirono S et al.[10] When the distance was longer in hamatoma side, upward shift was defined, which is associated with lift of M1M2 porition by Hirono S et al.[10]

Statistical analysis

Excel 2010 software (Microsoft, Redmond, WA, USA) was used for statistical analysis. Student *t*-test was applied for comparisons of mean values between two groups and values of $P < 0.05$ were considered to indicate statistical significance. For χ^2 test, Chi-squared test with or without Yate's continuity correction and Cochran-Mantel-Haenstzel test were used.

Results

Age and lacunar infarction

The average age of the 284 CSDH patients was 73.4 ± 12.8 years, and MRI was performed in 82 patients (average age 75.7 ± 13.6 years). Lacunar infarction was identified in 5 (average age 83.4 ± 2.6 years), and cortical infarction was recognized in 2 patients, an 87-year-old man and a 72-year-old-woman. These two cortical infarction cases were not included in lacunar infarction group. The average age of CSDH patients with lacunar infarction was significantly higher than those without lacunar infarction ($P < 0.05$).

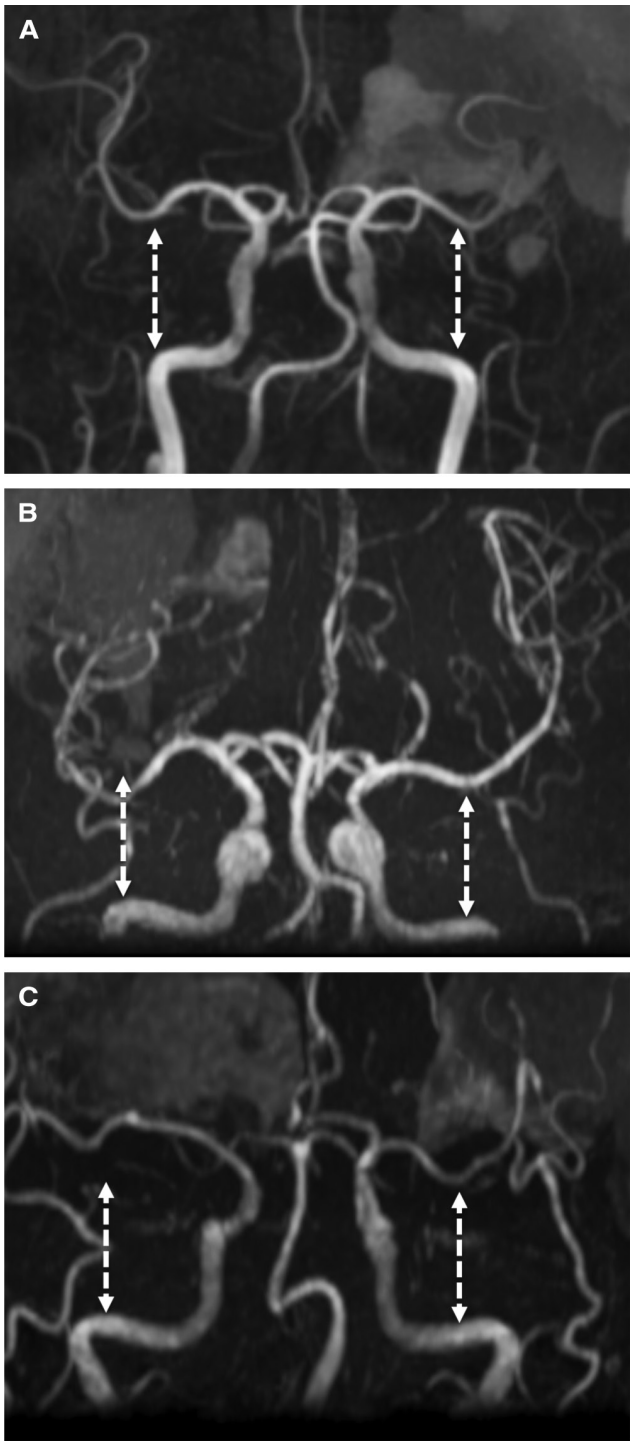

Fig. 1 In this figure, the length of white dotted arrows are same in the left and the right sides. A. No M1 shift: The distance between petrous portion of intercarotid artery (ICA) and M1M1 portion (white dotted arrows) is same. B. M1 downward shift: White dotted arrows are with same length. The distance between petrous portion of ICA and M1M1 portion is shorter in the hematoma side. C. M1 upward shift: The distance is longer in the hematoma side. White dotted arrows are with same length. ICA: internal carotid artery

Table 1 Details of five CSDH patients with lacunar infarction

Case	Age	Sex	Complications	Side of Hx	Side of lacunar infarction	MMT		M1 shift	Duration of admission
						upper	lower		
1	87	M	HT, DM, Old CI	left	left	4+/5	4–/5	upward	30 days
2	84	M	HT, DM	right	right	5–/5	4+/5	downward	14 days
3	80	M	HT	right	right	5–/5	4+/5	upward	20 days
4	84	M	HT	right	right	4–/5	4–/5	downward	29 days
5	82	F	HT	right	right	5–/5	4+/5	downward	10 days

CSDH: chronic subdural hematoma

Table 2 Lacunar infarction and M1 shift

		Lacunar infarction		Total
		+	–	
M1 shift	+	5	4	9
	–	0	73	73
Total		5	77	82

Table 3 Statistical analyses

Type of Test	χ^2	Degrees of freedom	*P* value
Chi-squared test	43.189	1	0.000
Yate's continuity correction	34.031	1	0.000
Cochran-Mantel-Haenstzel test	42.662	1	0.000

Hematoma thickness

The mean of hematoma thickness was 20.8 ± 5.5 mm in the five CSDH patients with lacunar infarction, and 19.2 ± 5.0 mm in CSDH patients without lacunar infarction. There was no significant difference in hematoma thickness between the two groups ($P > 0.05$).

Five CSDH patients with lacunar infarction

Details of the five CSDH patients with lacunar infarction were described in Table 1, and MRI and MRA were shown in Fig. 2. All the five patients have been taking anti-hypertensive medication, two had diabetes mellitus, and one had history of lacunar infarction. Among 82 CSDH patients, MRA showed M1 shift in 9 CSDH patients. As shown in Table 2, all the five patients with lacunar infarction demonstrated M1 shift (100%); downward shift was identified in three and upward in two. On the other hand, M1 downword shift was recognized in only 4 cases among 77 CSDH patients without lacular infarction (5.2%). As shown in Table 3, Chi-squared test with or without Yate's continuity correction and Cochran-Mantel-Haenstzel test demonstrated significantly higher incidence of lacunar infarction in the patients with M1 shift than in those without M1 shift ($P < 0.0001$).

Anti-platelet medicine was restarted from third-day after the burr-hole surgery only in Case 1, because the patient had been taking the medicine for an old lacunar infarction before admission; however, anti-platelet treatment was not applied in the other four cases. The five CSDH patients required physical rehabilitation, and all of them recoverd smoothly and were discharged to their home within one month.

Discussion

It is possible that CSDH becomes a cause of lacunar infarction but the mechanism has not been clarified yet.[8,9] In our study, all five CSDH patients with lacunar infarction were older than 80 years and had hypertension, and two of them had DM (Table 1). The risk of lacunar infarction was therefore high in all the cases.[4-7] The authors consider that distortion, twisting, or elongation of the lentriculostriate arteries (LSA) must have been an additional factor of the lacunar infarction in these CSDH patients. A shift in the M1 to M1M2 portion of the MCA on the hematoma side was identified on MRA in all five cases with lacunar infarction, while such a shift was recognized in only four patients among 77 patients without lacunar infarction. And without M1 shift, lacunar infarction does not occur ($P < 0.0001$) (Tables 2 and 3). Hirono et al.[10] reported that LSAs are stretched when MCA pushed downward (hammock MCA) and ischemia can be caused in LSA territory. However, the authors consider that only M1 shift is not always the cause of lacunar infarction, and as recognized in acute SDH patients with brain herniation, the incidence of lacunar infarction is not high although the incidence of infarction in

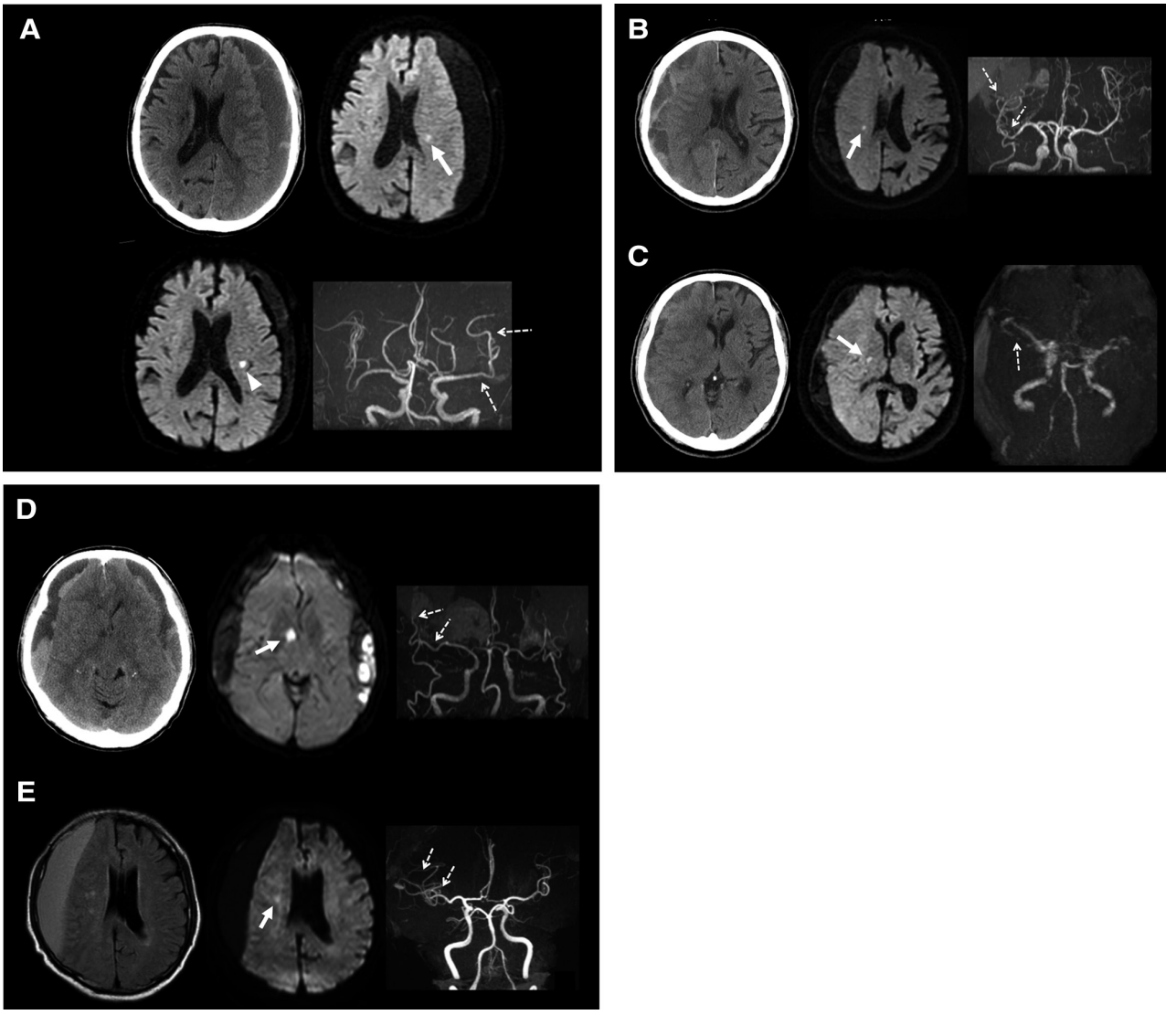

Fig. 2 A. CT demonstrates left CSDH, and a high-signal spot is identified in the left corona radiata on DWI (white arrow), which becomes clearer (white arrowhead) on follow-up MRI. MRA, which was performed next day after surgery, shows a medial shift of the M3 and M4 portions, and mild elevation of the M1 portion of the left MCA (dotted white arrows). B. CT shows right CSDH and DWI of MRI demonstrates a spotty ischemic lesion in the right corona radiata (white arrow). A downward shift of the M1 and M2 portions of the right MCA is recognized on MRA (dotted white arrows). C. Right CSDH is identified on CT, and a spotty high signal lesion is recognized in the right putamen on DWI (white arrow). MRA shows a remarkable downward shift of the M1 and M2 portions of the right MCA (dotted white arrow). D. Bilateral CSDH is demonstrated on CT, and a clear high-signal lesion is identifiable in the right posterior limb of the internal capsule on DWI (white arrow). MRA shows downward compression from the M2 to M4 portions of the right MCA, causing elongation of the M1 portion (dotted white arrows). E. Thick CSDH and a spotty high-signal lesion (white arrow) are identified in the right corona radiata on MRI. On MRA, the M2 to M4 portions of the right MCA are compressed downwards and the M1 portion is elongated (dotted white arrows). CT: computed tomography, CSDH: chronic subdural hematoma, DWI: diffusion-weighted image, MCA: middle carotid artery, MRA: magnetic resonance angiography, MRI: magnetic resonance imaging

posterior cerebral artery (PCA) is high. [11-13] On the other hand, the infarction of the PCA area is rarely identified in CSDH patients, even though the hematoma is extremely thick with a remarkable midline-shift.[14,15]

Therefore, the authors concluded that CSDH can cause lacunar infarction by distortion, twisting, or elongation of the LSA in the elderly with hypertension and DM, those who are potentially in a high risk of the formation of lipohyalinosis or

microatheroma in the arteries. Inopportunely, the postoperative MRI and MRA were not performed in the five cases with lacunar infarction when CSDH completely disappeared, and it is impossible to prove that an arteriosclerosis-related change progressed in the patients. The authors intend to investigate this point in a future study.

The incidence of lacunar infarction in CSDH patients with rapidly worsened motor weakness was 6.1% (5/82) in our series; however, the true incidence was unclear because MR examination was not applied to all CSDH patients. In our study, MRI was performed in only the CSDH patients with motor weakness and asymptomatic lacunar infarctions might be overlooked. MRI and MRA examinations before surgery are recommended for CSDH patients, especially when the patient complains of sudden onset or rapid deterioration of motor weakness. In our cases, the lacunar infarctions were small and did not cause severe sequelae; however, the patients and their family might claim that the surgery could cause the infarction if a small lacunar infarction is identified after surgery. Anti-platelet therapy might not always be necessary for CSDH induced lacunar infarction, because the mechanism of the infarction is different from that of lipohyalinosis or microatheroma formation, and the use of anti-platelet medicine after surgery is associated with a high recurrence rate of CSDH.[14] Without rehabilitation, the motor weakness might be prolonged after surgery; therefore, early detection of infarction and early initiation of rehabilitation should be achieved for CSDH patients with lacunar infarction.

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Conflicts of Ineterest Disclosure

On behalf of all authors, the corresponding author states that there is no conflict of interest.

References

1) Mohr JP. Lacunes. *Stroke* 13: 3–11, 1982
2) Horowitz DR, Tuhrim S, Weinberger JM, Rudolph SH. Mechanisms in lacunar infarction. *Stroke* 23: 325–327, 1992
3) Caplan LR: Lacunar infarction and small vessel disease: pathology and pathophysiology. *J Stroke* 17: 2–6, 2015
4) Weisberg LA: Diagnostic classification of stroke, especially lacunes. *Stroke* 19: 1071–1073, 1988
5) You R, McNeil JJ, O'Malley HM, Davis SM, Donnan GA: Risk factors for lacunar infarction syndromes. *Neurology* 1995; 45: 1483–1487.
6) Arboix A, Martí-Vilalta JL: Lacunar stroke. *Expert Rev Neurother* 9: 179–196, 2009
7) Kaufman JL, Karceski S: Risk factors and prevention of lacunar infarcts in 60- to 64-year-olds. *Neurology* 73: e17–e19, 2009
8) Anzalone N, Landi G: Non ischaemic causes of lacunar syndromes: prevalence and clinical findings. *J Neurol Neurosurg Psychiatry* 52: 1188–1190, 1989
9) Lazzarino LG, Martinelli A, Nicolai A: Lacunar syndromes due to non ischaemic causes: prevalence and clinical findings (study of 19 patients). *Acta Neurol (Napoli)* 15: 401–408, 1993
10) Hirono S, Ozaki K, Ito D, Matsutani T, Iwadate Y: Hammock middle cerebral artery and delayed infarction in lenticulostriate artery after staged resection of giant insular glioma. *World Neurosurg* 117: 80–83, 2018
11) Meyer A: Herniation of the brain. *Arch Neurol Psychiatry* 4: 387–400, 1920
12) Namura S, Kang Y, Matsuda I, Kamijyo Y: Magnetic resonance imaging of sequelae of temporal lobe herniation secondary to traumatic acute subdural hematoma: Kernohan's notch and posterior cerebral artery territory infarctions contralateral to the supratentorial lesion—case report. *Neurol Med Chir (Tokyo)* 37: 32–35, 1997
13) Server A, Dullerud R, Haakonsen M, Nakstad PH, Johnsen UL, Magnaes B: Post-traumatic cerebral infarction. Neuroimaging findings, etiology and outcome. *Acta Radiol* 42: 254–260, 2001
14) Yamada SM, Tomita Y, Murakami H, et al.: Headache in patients with chronic subdural hematoma: analysis in 1080 patients. *Neurosurg Rev* 41: 549–556, 2018
15) Tomita Y, Yamada SM, Yamada S, Matsuno A: Subdural tension on the brain in patients with chronic subdural hematoma is related to hemiparesis but not to heacache or recurrence. *World Neurosurg* 119: e518–e526, 2018

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