

Case Studies

A Recurrent Case of Ischemic Stroke Caused by Vasospasm due to Giant Cell Arteritis

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A 73-year-old man was admitted with sudden right upper-limb weakness. He had a temporal headache on the left side and had a 4-month history of fever. Meandering of the left temporal artery (TA) with induration and high inflammatory responses (white blood cell count 22,500 per microliter, C-reactive protein 35.0 mg/dL, and elevated sedimentation rate [ESR] 80 mm/h) were observed. Glycometabolism and lipid metabolism were normal, and autoimmune antibodies were negative. Cultivation tests revealed no bacteria in either blood culture or cerebrospinal fluid. Brain magnetic resonance imaging (MRI) showed ischemic lesion in the left frontal lobe, while magnetic resonance angiography (MRA) and carotid ultrasonography showed unstable plaque lesions in the left extracranial internal carotid artery (ICA). According to reported criteria (age > 50 years, new onset of headache, abnormality of the TA, and raised ESR), we diagnosed giant cell arteritis (GCA) with acute ischemic stroke (IS) and gave the patient antithrombotic therapy (aspirin 100 mg, cilostazol 200 mg). After admission, hemiparesis progressed but fluctuated. Subsequent MRI showed new lesions in the left watershed area. MRA also showed vasospasm in the middle cerebral artery and C5 portion of the ICA. Considering the correlation with GCA pathophysiology, oral prednisolone therapy was administered. Steroid therapy has prevented stroke recurrence and improved the symptoms and vasospasm. We wish to emphasize that GCA can induce IS via vasospasm, and steroid therapy is recommended. **Key Words:** Giant cell arteritis—unstable carotid plaque—carotid artery stenosis—vasospasm—stroke recurrence.

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A 73-year-old man was admitted with sudden onset of right upper-limb weakness. He reported temporal headache on the left side and had a 4-month history of fever. Orbital pain and hearing loss were also gradually progressing. Apart from hypertension, he had no noteworthy medical, family, preference, or drug history. Meandering of the left temporal artery (TA) with induration and high inflammatory responses (white blood cell count 22,500 per microliter, C-reactive protein 35.0 mg/dL, and elevated sedimentation rate [ESR] 80 mm/h) were observed. There was no abnormality in glycometabolism/lipid

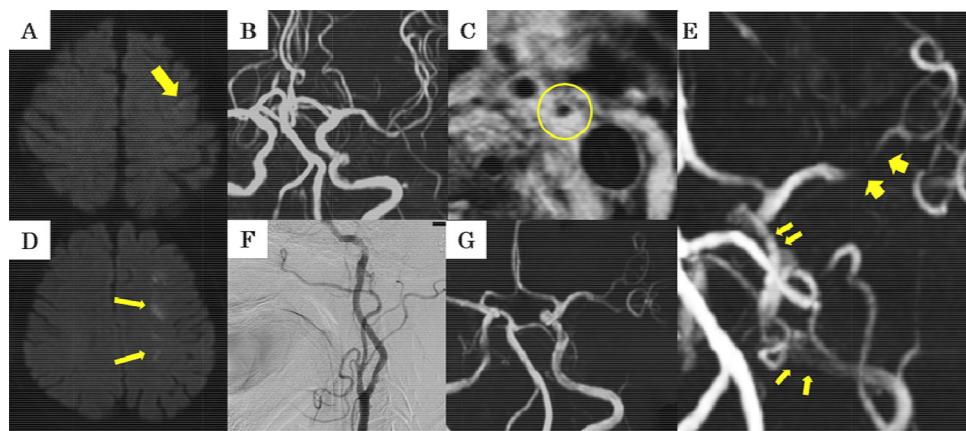


Figure 1. (A) Diffusion-weighted image showed high-signal lesion in left frontal lobe (→) (on admission). (B) Magnetic resonance angiography showed normal findings in intracranial vessels (on admission). (C) Magnetic resonance plaque imaging (by T2 black-blood method) showing unstable plaque lesion in the left internal carotid artery (○). (D and E) Two days after admission, magnetic resonance imaging and magnetic resonance angiography showed new ischemic lesions in the left watershed area (→) and vasospasm in the left middle cerebral artery and the C5 portion of the internal carotid artery (→). (F) Internal carotid artery flow was improved in catheter angiography 2 weeks after starting steroid therapy. (G) Vasospasm in the left middle cerebral artery and the C5 portion of the internal carotid artery was improved on magnetic resonance angiography performed after 6 months.

metabolism, and autoimmune antibodies (including antinuclear antibody, anti-SS-A/B antibodies, and antineutrophil cytoplasmic antibody) were negative. Cerebrospinal fluid showed elevation of protein (97 mg/dL) and pleocytosis (76 per microliter). Cultivation tests revealed no bacteria. Brain magnetic resonance imaging showed an ischemic lesion in the left frontal lobe (Fig 1, A). Magnetic resonance angiography (Fig 1, B,C) and carotid ultrasonography showed an unstable plaque in the left extracranial internal carotid artery (ICA). Antithrombotic therapy (aspirin 100 mg, cilostazol 200 mg) was given for acute ischemic stroke (IS). Giant cell arteritis (GCA) was also suspected from the symptoms and examination results. After admission, hemiparesis progressed but fluctuated, and subsequent magnetic resonance imaging (Fig 1, D) showed new lesions in the left watershed area. Magnetic resonance angiography (Fig 1, E) showed vasospasm in the middle cerebral artery and the C5 portion of the ICA. Oral prednisolone therapy (from 40 mg to 10 mg as maintenance therapy) was immediately started after TA biopsy, considering the correlation with GCA pathophysiology. Fever, headache, orbital pain, hearing loss, TA abnormality, and inflammatory responses (white blood cell count 8300 per microliter, C-reactive protein .29 mg/dL, and ESR 10 mm/h) were improved after 2 weeks. Steroid therapy has also prevented stroke recurrence and improved vasospasm (Fig 1, FG). Although inflammatory infiltration was unclear from the biopsy, the other criteria for GCA diagnosis (age > 50 years, new onset of headache,

TA abnormality, and raised ESR) were all fulfilled, and finally he was diagnosed with GCA.¹

We considered that IS had been induced by an unstable plaque, but IS recurrence could have occurred through a hemodynamic mechanism via vasospasm due to GCA. Improvement of symptoms and vasospasm after steroid therapy also supported this diagnosis of IS with GCA. GCA-related vasculitis (vertebral–basilar artery or ICA) has been reported to induce IS.²⁻⁵ However, intracranial artery vasospasm (especially in the middle cerebral artery) is rare in IS with GCA.⁵ We wish to emphasize that GCA can induce IS via vasospasm, and steroid therapy is recommended.

References

1. Ness T, Bley TA, Schmidt WA, et al. The diagnosis and treatment of giant cell arteritis. *Dtsch Arztebl Int* 2013; 110:376-386.
2. Zarar A, Zafar TT, Khan AA, et al. Internal carotid artery stenosis associated with giant cell arteritis: case report and discussion. *J Vasc Interv Neurol* 2014;7:24-27.
3. Gonzalez-Gay MA, Vazquez-Rodriguez TR, Gomez-Acebo I, et al. Strokes at time of disease diagnosis in a series of 287 patients with biopsy-proven giant cell arteritis. *Medicine (Baltimore)* 2009;88:227-235.
4. Lariviere D, Sacre K, Klein I, et al. Extra- and intracranial cerebral vasculitis in giant cell arteritis: an observational study. *Medicine (Baltimore)* 2014;93:1-8.
5. Salvarani C, Giannini C, Miller DV, et al. Giant cell arteritis: involvement of intracranial arteries. *Arthritis Rheum* 2006;55:985-989.