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Case Report

A Case of Internal Carotid Artery Occlusion Caused by En Bloc Distal Embolization of Carotid Free-Floating Thrombus Treated by Mechanical Thrombectomy

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Objective: We report a case of internal carotid artery (ICA) occlusion caused by en bloc distal embolization of carotid free-floating thrombus (FFT) treated by mechanical thrombectomy.
Case Presentation: A 57-year-old woman was brought to our hospital with dysarthria, right hemiparesis, and motor aphasia. MRI and MRA revealed acute infarction due to middle cerebral artery occlusion. Carotid ultrasonography demonstrated a pedunculated mobile plaque in the left ICA. We diagnosed embolic infarction due to the carotid FFT and started medical treatment. However, on the second hospital day, the carotid FFT detached from the arterial wall en bloc, resulting in left ICA occlusion. The occluded ICA was successfully recanalized by mechanical thrombectomy.
Conclusion: FFT is associated with a high risk of embolic ischemic stroke and the primary treatment strategy must be carefully considered.

Keywords carotid artery, free-floating thrombus, en bloc, embolic stroke, mechanical thrombectomy

Introduction

Carotid free-floating thrombus (FFT) is a rare etiology causing ischemic stroke. We report a patient with carotid FFT who was admitted to the hospital for middle cerebral artery (MCA) occlusion caused by carotid FFT, which subsequently caused internal carotid artery (ICA) occlusion due to its distal embolization en bloc on the day following admission, which was treated by mechanical thrombectomy.

Case Presentation

Patient: A 57-year-old woman.
Complaint: Motor aphasia and dysarthria.

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Medical history: None in particular.
Social history: No cigarette smoking.
Present illness: She was admitted to the emergency department 4 hours and 45 min after sudden onset of difficulty talking. On the visit, her blood pressure was 143/89 mmHg, heart rate was 82 beat/min, Glasgow Come Scale (GCS) was 14 (E4V4M6), motor aphasia, mild dysarthria, and muscle weakness of the right upper limb with a manual muscle test (MMT) score of 4, and the National Institutes of Health Stroke Scale (NIHSS) score was 5. On blood testing, dyslipidemia (low density lipoprotein cholesterol [LDL-C]: 191 mg/dL) and a mild increase in D-dimer (2.0 μg/dL) were detected. No brain natriuretic peptide (BNP) increase was noted (lower than 3.9 pg/mL). On chest radiography, no cardiac dilatation was noted and electrocardiography demonstrated sinus rhythm. On diffusion-weighted head MRI, a new infarct lesion with a diffusion-weighted imaging-Alberta Stroke Program Early CT Score (DWI-ASPECTS) of 9/11 was detected (**Fig. 1A** and **1B**). On T2*-weighted imaging, a susceptibility vessel sign was noted in the left MCA (**Fig. 1C**). On head MRA, the left distal MCA was occluded (**Fig. 1D**). On neck MRA, there was no abnormal finding (**Fig. 1E**). We performed DSA considering mechanical thrombectomy. The left carotid angiography revealed a contrast-defective lesion at the origin of the

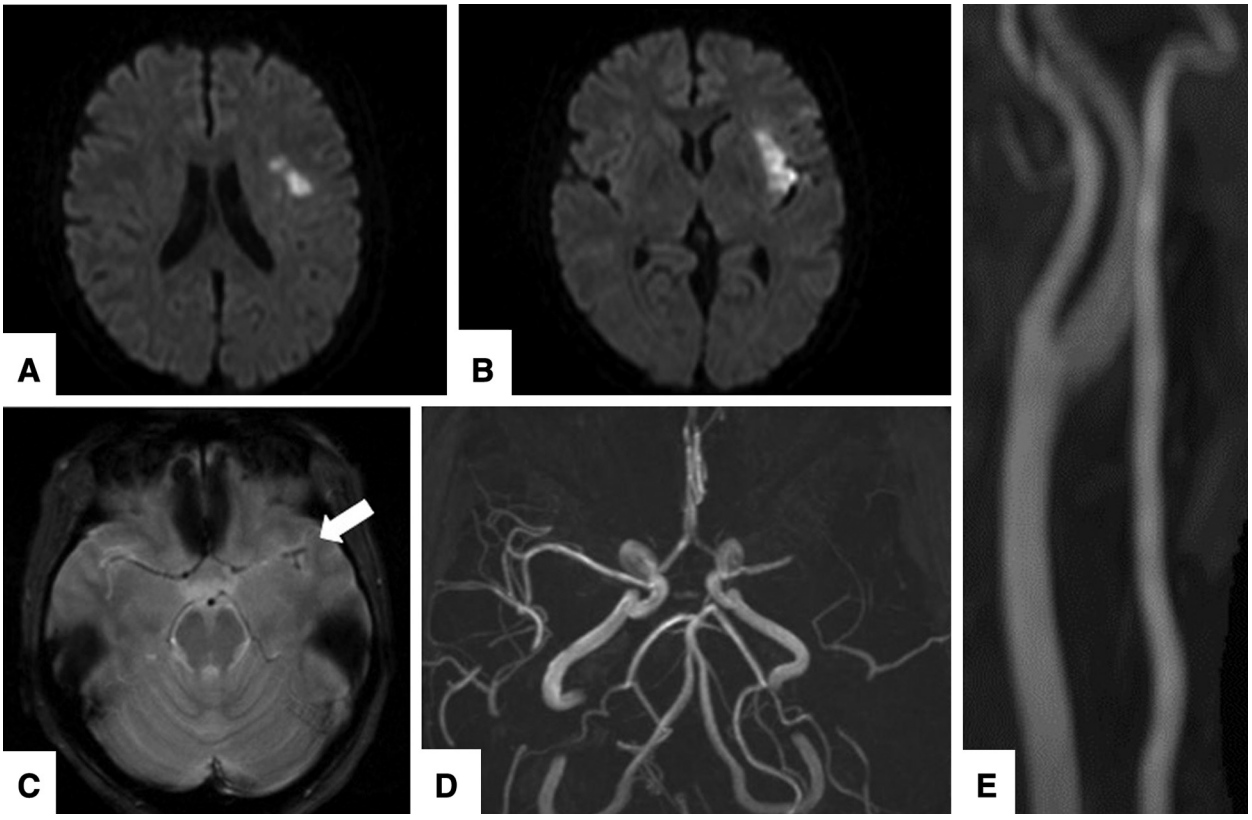

Fig. 1 On admission. (**A** and **B**) Diffusion-weighted head MRI. A new infarct lesion with DWI-ASPECTS of 9/11 was noted. (**C**) T2*-weighted imaging. A susceptibility vessel sign was noted (arrow). (**D**) Head MRA. The left distal MCA was occluded. (**E**) Neck MRA. No abnormal findings. DWI-ASPECTS: diffusion-weighted imaging-Alberta Stroke Program Early CT Score; MCA: middle cerebral artery

cervical ICA and distal MCA occlusion as visualized by head MRA (**Fig. 2A** and **2B**). On cone-beam CT, a lesion originating from the ICA wall and protruding into the vascular lumen was present (**Fig. 2C**). Carotid ultrasonography was performed with the sheath remained in the right femoral artery. Isoechoic FFT with a maximum diameter of 6.6 mm adhering to the vascular wall and moving with the heartbeat was present at the origin of the left ICA. There was no evidence of arterial dissection (**Fig. 2D** and **2E**). Endovascular treatment of left MCA occlusion and carotid FFT was considered, but medicinal treatment was first selected because collateral circulation was favorable, neurologic symptoms were relatively mild, and it was possible for the procedure to induce new distal embolization. Argatroban drip infusion was initiated after admission. However, aphasia aggravated to NIHSS score 7 on the day following admission (second hospital day), mild expansion of the infarct lesion (DWI-ASPECTS 8/11) was noted on diffusion-weighted head MRI, and new left ICA occlusion was noted on head and neck MRA (**Fig. 3A** and **3B**). On carotid ultrasonography, FFT observed on the previous day disappeared (**Fig. 3C** and **3D**), but a non-mobile isoechoic plaque with a maximum intima media thickness (IMT) of 3.2 mm remained (**Fig. 3E**). As the FFT was considered to have caused distal embolization en bloc, mechanical thrombectomy was performed.

Endovascular treatment: A 9-F sheath was inserted into the right femoral artery. After heparinization, an Optimo (EPD 9F 90 cm; Tokai Medical Products, Aichi, Japan) was placed in the left CCA. On left common carotid angiography, the FFT was lost, as observed on ultrasonography, and mild plaque remained at the origin of the ICA (**Fig. 4A**). The Optimo was carefully advanced to the left ICA while paying attention to the residual plaque. Left internal carotid angiography revealed ICA occlusion beyond ophthalmic artery (**Fig. 4B**). While the ICA was blocked by inflating the balloon of the Optimo, a 5 MAX JET 7 (Penumbra, Alameda, CA, USA) was placed just proximal to FFT using a microguidewire and microcatheter in order to apply a direct aspiration first pass technique (ADAPT)[1] (**Fig. 4C**). A JET 7 was connected to the Penumbra aspiration pump and aspiration was initiated, followed by waiting for 90 s. Back flow was noted during retrieval of the JET 7 and the thrombus was unable to be

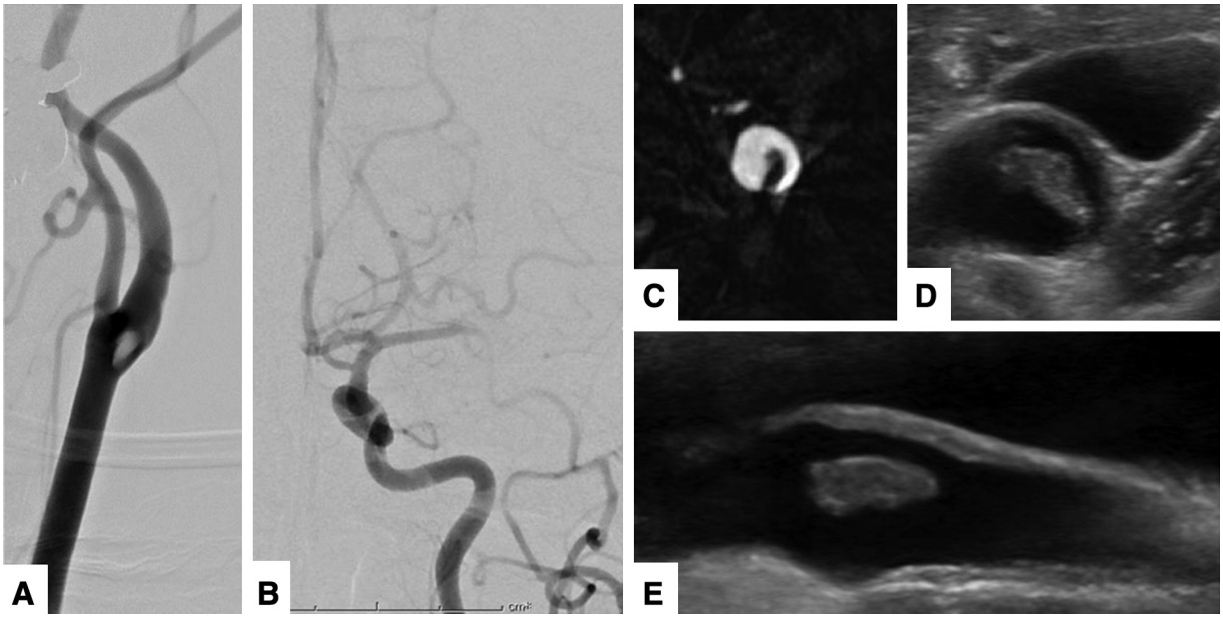

Fig. 2 On admission. (**A**) Left common carotid angiography. A contrast-defective lesion was present at the origin of the ICA. (**B**) Left internal carotid angiography. The left distal MCA was occluded. (**C**) Axial view on cone-beam CT (contrast-enhanced). A structure originating from the ICA wall and protruding into the vascular lumen was present. (**D** and **E**) Carotid ultrasonography (**D**: short axis view, **E**: long axis view). An isoechoic pedunculated mobile lesion with a maximum diameter of 6.6 mm adhering to the vascular wall and moving with the heartbeat was present at the origin of the left ICA, and diagnosed as carotid FFT. FFT: free-floating thrombus; ICA: internal carotid artery; MCA: middle cerebral artery

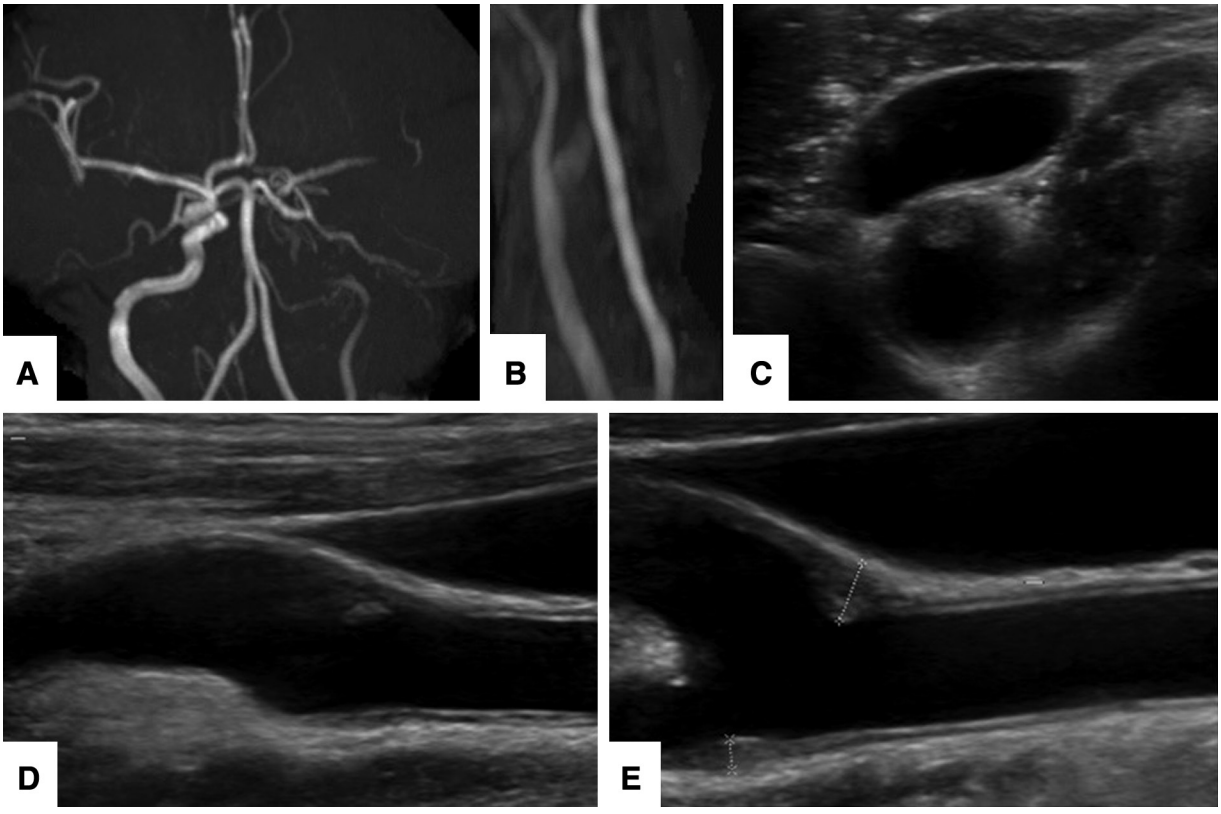

Fig. 3 On the day following admission (second hospital day). (**A** and **B**) MRA (**A**: head, **B**: neck). Left ICA occlusion was newly observed. (**C** and **D**) Carotid ultrasonography (**C**: short axis view, **D**: long axis view). The FFT observed on the previous day disappeared. (**E**) Long axis view on carotid ultrasonography. Non-mobile isoechoic plaque with a maximum IMT of 3.2 mm remained. FFT: free-floating thrombus; ICA: internal carotid artery

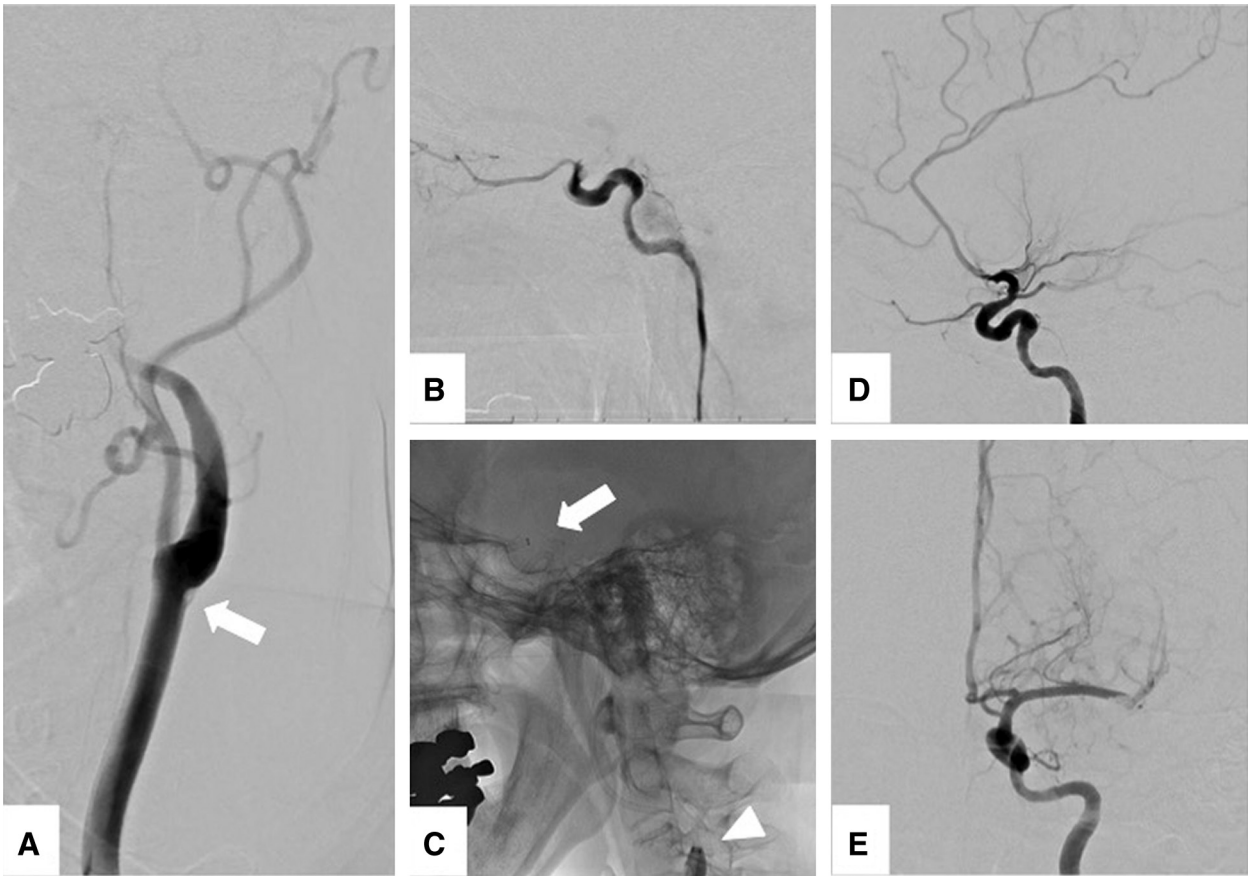

Fig. 4 Endovascular treatment. (**A**) Left common carotid angiography. Similar to ultrasonography, the FFT was lost and mild residual plaque was present at the origin of the ICA (arrow). (**B**) Left internal carotid angiography (before treatment). ICA occlusion beyond ophthalmic artery was confirmed. (**C**) While the ICA was blocked with the inflated balloon of the Optimo (arrowhead), a 5 MAX JET 7 was placed just proximal to the FFT (arrow) and ADAPT was performed. (**D**) Lateral view on left internal carotid angiography (after treatment). The ICA was recanalized. (**E**) Frontal view on left internal carotid angiography (after treatment). The left MCA occlusion was recanalized compared with that on the previous day. ADAPT: a direct aspiration first pass technique; FFT: free-floating thrombus; ICA: internal carotid artery; MCA: middle cerebral artery

confirmed, but when it was manually aspirated through the Optimo, the thrombus was removed. On left internal carotid angiography, recanalization of the left ICA was confirmed (**Fig. 4D** and **4E**). As only non-mobile mild plaque remained in the cervical ICA, course observation was selected without carotid artery stenting (CAS). The left MCA occlusion was recanalized compared with that on the previous day (**Fig. 4E**) and the procedure was completed at this point.

Postoperative course: On the day following treatment (third hospital day), DWI-ASPECTS was 7/11 on head MRI, suggesting mild aggravation, but no intracranial hemorrhage was noted (**Fig. 5A** and **5B**). On head MRA, visualization of the left MCA improved (**Fig. 5C**). Argatroban drip infusion and oral administration of cilostazol at 200 mg/day and atorvastatin at 10 mg/day were initiated. Paralysis of the right upper limb was temporarily exacerbated but recovered to MMT 4 on the seventh hospital day and the NIHSS score was 8. On the eighth hospital day, the left MCA was completely recanalized on head MRA (**Fig. 5D**). On the 15th hospital day, the residual plaque with a maximum IMT of 3.2 mm withdrew to 1.3 mm on carotid artery ultrasonography (**Fig. 5E**). Rehabilitation was continued thereafter. On the 68th hospital day, the residual plaque withdrew to a maximum IMT of 1.1 on carotid ultrasonography, being almost normalized (**Fig. 5F**). On the 69th hospital day, the patient was discharged to home with a modified Rankin Scale score of 2, residual mild right hemiplegia, and mild aphasia (NIHSS score 3).

Discussion

Carotid FFT is a rare cause of ischemic stroke and its incidence was reported to be 1.53%.[2] Bhatti et al. defined FFT as an elongated thrombus attached to the arterial wall with cyclical motion related to cardiac cycles and reviewed 145

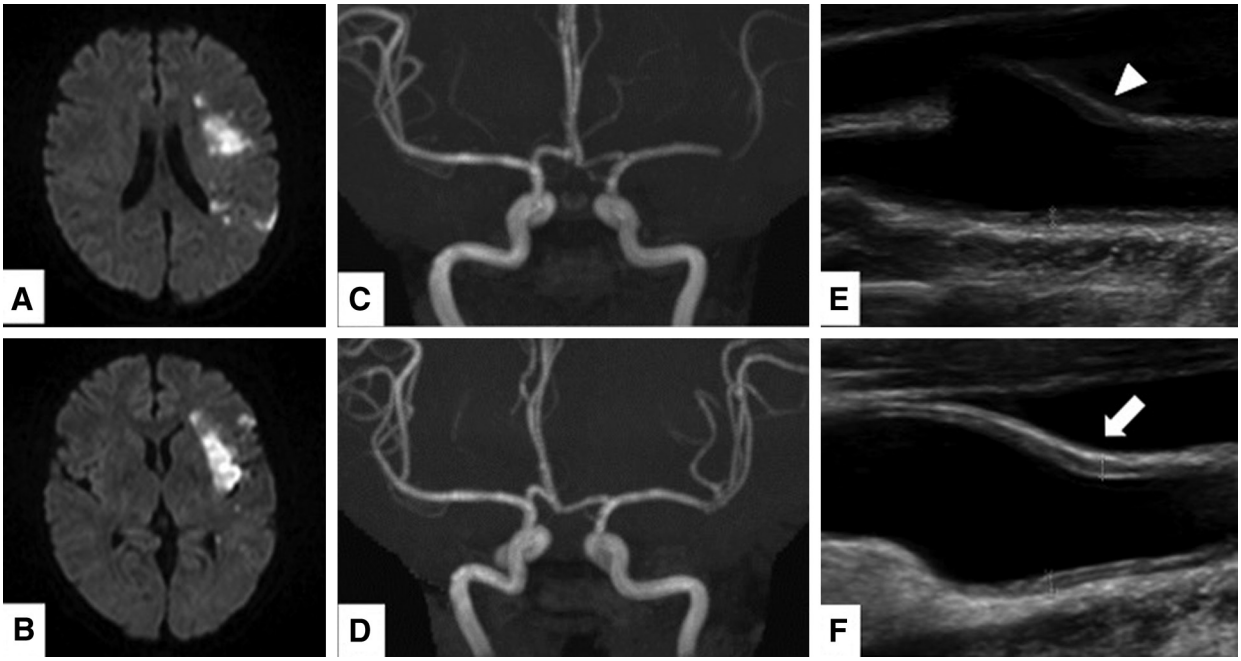

Fig. 5 (**A** and **B**) Diffusion-weighted head MRI on the third hospital day. DWI-ASPECTS was 7/11, showing mild aggravation. (**C**) Head MRA on the third hospital day. Patency of the left ICA and improvement of visualization of the left MCA periphery were confirmed. (**D**) Head MRA on the eighth hospital day. The left MCA was completely recanalized. (**E**) Carotid ultrasonography on the 15th hospital day. The residual plaque (arrowhead) withdrew to a maximum IMT of 1.3 mm. (**F**) Carotid ultrasonography on the 68th hospital day. The residual plaque (arrow) withdrew to a maximum IMT of 1.1 mm, being mostly normalized. DWI-ASPECTS: diffusion-weighted imaging—Alberta Stroke Program Early CT Score; ICA: internal carotid artery; IMT: intima media thickness; MCA: middle cerebral artery

cases of carotid FFT, excluding embolism cases such as cardiogenic embolism cases and occlusion cases.[3] They stated that many patients had a hypercoagulability and were younger than those with standard carotid artery stenosis as a characteristic of carotid FFT. ICA lesions were the most frequent, accounting for 75% and neurological symptoms were noted in as many as 92%. Underlying lesions of FFT include carotid aneurysm and small dissection, in addition to atherosclerosis, but the cause of the disease is unclear in many cases.[3] Vassileva et al. reported that carotid FFT was observed in 0.18% of ischemic stroke cases without ICA stenosis,[4] and thus it should be considered as a cause of juvenile ischemic stroke with a low risk of arteriosclerotic disease. In the present patient, no abnormality was observed on transthoracic echocardiography or 24-hour Holter electrocardiography, and the lesion with a maximum diameter of 6.6 mm was pedunculated, adhering to the ICA wall, and moved with the heartbeat on carotid ultrasonography, based on which it was diagnosed as carotid FFT-induced embolic cerebral infarction (**Fig. 2D** and **2E**). Regarding the pathology of FFT removed by mechanical thrombectomy, no bacterial body, cholesterin cleft, or fibrous tissue suggesting carotid web was noted, and it was a fibrin thrombus composed of fresh red blood cells and fibrin (**Fig. 6**). As dyslipidemia was observed and residual plaque was present after distal embolization with FFT (**Fig. 3C**, **3D**, and **3E**), collapse of athromatous plaque may be an etiology of FFT formation. However, the residual plaque markedly withdrew only within 2 weeks (**Fig. 5E**) and this is atypical as athromatous plaque. The etiology of the present case was unable to be clarified because the pathology of the plaque itself was unable to be investigated, but it is also possible that FFT was formed by small dissection. Treatment methods for carotid FFT include medicinal treatment (anticoagulant therapy, antiplatelet therapy, and combination of these), surgical treatment (ex. carotid endarterectomy), and endovascular treatment. The efficacy of each treatment has been reported, but none were superior.[2,3] Bhatti et al. reported that neurological manifestations did not aggravate and FFT disappeared in 86% of patients treated by medicinal treatment,[3] suggesting that medicinal treatment should be considered first, but the outcome of intravenous thrombolysis (IVT) is not favorable. Vanacker et al. reported recurrence or rapid progressive worsening in 4 of 11 patients after IVT for FFT. They described that IVT may have been related to distal mobilization of the thrombus.[5] Fridman et al. reported that the recurrence of ischemic stroke within

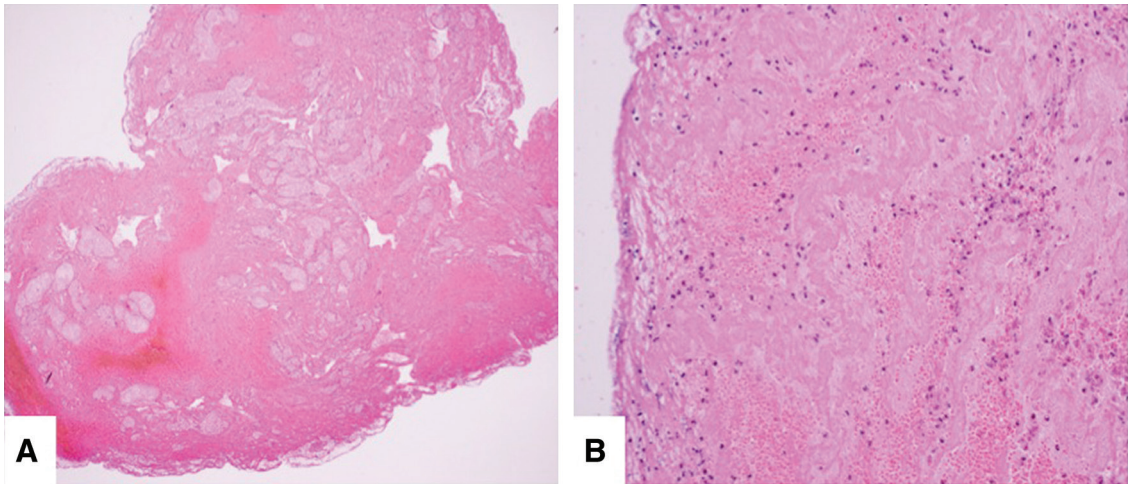

Fig. 6 Pathology of the FFT (**A**: 40× magnification, **B**: 200× magnification). FFT was composed of fresh red blood cells and fibrin accompanied by a small number of neutrophils. No organization was noted, nor was there fibrous tissue suggesting bacterial body, cholesterin cleft, or carotid web. It was a fibrin thrombus. FFT: free-floating thrombus

24 hours significantly increased in the IVT group compared with that in the non-IVT group.[2] Endovascular treatment of carotid FFT is advantageous in the following points compared with surgical treatment: General anesthesia is not essential, less time is required for the procedure and vascular operation, the risk of embolic complications can be minimized, perioperative anticoagulant therapy can be continued if necessary, and intracranial endovascular treatment can be attempted immediately if a thrombus is carried into the intracranial circulation.[6] Bhogal et al. reported seven cases treated by CAS for carotid FFT. Using an embolic protection device (EPD) in all cases, they did not apply predilation in consideration of the risk of distal migration of the thrombus.[7] They performed treatment using a closed-cell type or micromesh-type stent and acquired favorable outcomes. They mentioned that selection of a longer size is necessary to ensure complete coverage of the FFT, and a micromesh stent is desirable to prevent thrombus fragmentation and distal migration. Following the recent progression of endovascular treatment, carotid FFT treated by mechanical thrombectomy (stent retriever and aspiration catheter) has been increasingly reported. Fitzpatrick et al.[6] and Giragani et al.[8] performed treatment using a stent retriever by combining proximal balloon protection or distal filter protection, and they were able to remove the FFT without complications. Otawa et al.[9] and Kubo et al.[10] reported cases in which the FFT was removed by aspiration using an aspiration catheter followed by CAS applied to the residual lesion. Carr et al. reported a case in which ADAPT was performed for the first pass, but it was only partially removed; therefore, they used the technique combining a stent retriever and an aspiration catheter for the second pass, and the FFT was able to be completely removed without complications.[11] On the other hand, some cases of endovascular treatment required postoperative additional treatment or intraoperative alteration of the treatment strategy. Bhogal et al. reported a case in which stents were telescoped twice for the in-stent thrombosis after CAS for carotid FFT[7] and Park et al. reported a case in which they tried to remove the FFT by aspiration, but abandoned it because of a large thrombus volume and changed the procedure to internal trapping,[12] suggesting the importance of sufficient preoperative consideration and postoperative follow-up. Thus, attention should be paid in some cases, but the EPD method and mechanical thrombectomy techniques used in CAS and other endovascular treatment techniques can be combined to minimize complications corresponding to the situation, being an advantage of endovascular treatment.

No timing of surgical or endovascular recanalization treatment has been standardized, as no standard has been established for the treatment method.[2] In acute-phase treatment, the risk of embolic complication increases due to high instability of lesions, whereas elective treatment falls behind, i.e., the brain, which was salvable, may be sacrificed due to recurrence of an ischemic event.[13] In the present patient, medicinal treatment was first selected, but FFT caused en bloc distal embolization and induced ICA occlusion on the day following admission. To our knowledge, this is the first case of FFT causing en bloc distal embolization and occlusion of the major artery. Due to mechanical thrombectomy, only mild sequela remained because

collateral circulation developed. As the FFT had a small adhering region, high mobility, and MCA occlusion, that is, major artery occlusion already occurred, endovascular treatment should have been urgently performed because the risk of recurrence of ischemic stroke is presumed to be high. No standard treatment for carotid FFT has been established and further studies are needed, but for initial treatment, low-invasive, and low-risk medicinal treatment may be considered first in many cases. However, serious ischemic stroke is likely to occur in the future in cases in which the FFT has a small adhering region with high mobility and a large thrombus volume, as observed in the present case. The efficacy and safety of endovascular treatment for carotid FFT have recently been frequently reported as described above, suggesting that endovascular treatment may be considered from the initial treatment in such cases.

Conclusion

A patient in whom carotid FFT caused en bloc distal embolization on the day following admission was treated by mechanical thrombectomy. This case highlighted the risk of FFT-induced ischemic stroke and the importance of the initial treatment.

Disclosure Statement

The authors declare no conflicts of interest.

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