

Carotid Free-Floating Thrombus in Association with Crohn's Disease

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Abstract

Backgrounds: Inflammatory bowel disease causes inflammatory changes in blood vessels, leading to vascular disease. It is associated with the risk of cardiovascular and cerebrovascular ischemic disease.

Case Presentation: The author has experienced a case of pseudo-occlusion of an internal carotid artery (ICA) in a patient with Crohn's disease who had no other risk factors for vascular disease. The patient presented with symptoms of ischemia in the ophthalmic artery. A free-floating thrombus in the ipsilateral ICA was observed. Revascularization was achieved by urgent carotid artery stenting.

Conclusion: The author reports a case of ischemic vasculopathy associated with inflammatory disease in a young patient. Literature review associated with Crohn's disease and carotid artery occlusion was also described.

Key words: Carotid Free-Floating Thrombus, Crohn's Disease, Carotid Artery Stenting

1. INTRODUCTION

Inflammatory bowel disease is associated with an inflammatory response and has been reported to cause vascular disease (1-6). In particular, ulcerative colitis is associated with the risk of cardiovascular and cerebrovascular disease (7-16). Complications such as internal carotid artery (ICA) occlusion have also been reported in Crohn's disease (17-19). It may be the cause of stroke in young patients or in patients without underlying hematologic diseases such as lifestyle-related diseases or hemo-clotting disorders. The author experienced a case of a free-floating thrombus in the ipsilateral ICA (19), which presented with symptoms of ocular arterial ischemia. Revascularization was obtained by urgent carotid artery stenting (CAS). Later close examination revealed a complication of Crohn's disease. The author reports previous cases, their pathophysiology, and treatment strategies, with a discussion of the literature.

2. CASE PRESENTATION

A 39-year-old female presented with photophobia of the left eye. At the time of initial

treatment, consciousness was clear and there was no motor paralysis in her limbs. She had no sensory disturbance, no ataxia, no limitation of eye movement, no pupillary irregularity, no abnormal light reflex, neither no lower cranial nerve palsy.

Blood pressure was 91/59 mmHg and heart beat rate were 78/min. She has a medical history of atopy. No history of alcohol consumption or smoking. A few days prior to the visit, she had fever, diarrhea, and body weight loss. A head magnetic resonance imaging (MRI) scan showed no acute ischemic lesions in the cranium, but there was decreased flow gain in the left ICA. This was accompanied by decreased blood flow in the left hemisphere (Figure 1). Carotid MRI showed high-signal lesions along the vessel wall at T1-weighted, T2-weighted, black-blood and time-of-flight images (Figure 1). Both axial images showed concentric signal changes in the vessel lumen. Ultra sound echo study against carotid artery showed a mobile plaque in left ICA. Echo brightness was low echoic. Blood peak flow velocity in the lesion was markedly decreased (22 cm/sec).

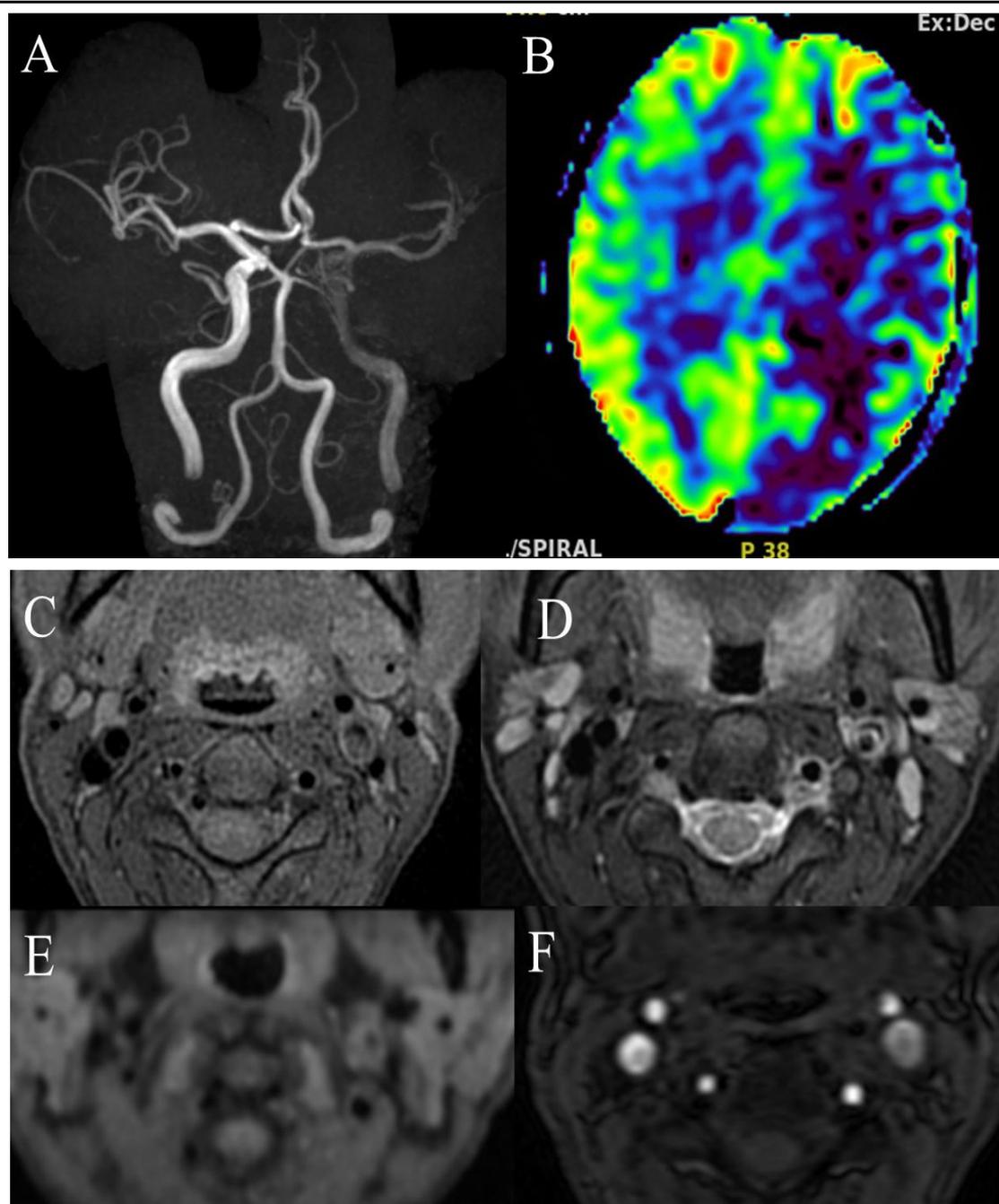


Figure 1. Magnetic resonance imaging (MRI) analysis at the time of admission. (A) shows the time-of-flight (TOF) head MRI. (B) shows the head MRI of the arterial spin labelling-perfusion weighted image. (C) shows the neck MRI in T1-weighted image. (D) shows the neck MRI scan in T2-weighted image. (E) shows the black-blood image of the neck MRI. (F) shows the TOF neck MRI.

Digital subtraction angiography showed severe stenosis of the left ICA. The stenotic lesion was confined to the proximal cervical carotid artery, not including the ICA bifurcation. Angiographical study appeared to be a free-floating thrombus (FFT) (18-23).

The intracranial left ICA and middle cerebral artery (MCA) had slow flow. Decreased flow was supplied from vertebral artery (VA) to left ICA via posterior communicating artery and from right ICA to left MCA via anterior

communicating artery. Emergency carotid artery stenting (CAS) was performed. Three pieces of open cell stent ($\phi 6\text{mm} \times 30\text{mm}$, $\phi 8\text{mm} \times 40\text{mm}$, $\phi 10\text{mm} \times 40\text{mm}$, Precise Carotid Stent; Cordis, Miami lake, FL, USA) were implanted. During the endovascular intervention, balloon wires were placed as protection devise (PercuSurge GuardWire System; Medtronic, Dublin, Ireland). ICA stenosis was well dilated, without any post dilating balloon assist (Figure 2). She had no complication of distal embolic stroke, restenosis of carotid artery nor other neurological deficits.



Figure 2. An angiography image of the left common carotid artery in the neck, taken from a lateral view, is presented. (A) shows the angiography taken prior to carotid artery stent deployment. (B) shows the angiography after the deployment of the carotid artery stent.

After surgery, the patient was treated with dual antiplatelet treatment including aspirin and clopidogrel orally. Acute periods of intervention, argatroban was dripped intravenously. No ischemic symptoms were observed. Photophobia in the left eye improved, but intermittent symptoms like flying mosquitoes were observed. There were no complications such as intracranial hemorrhage or hyper perfusion impairment in the post operative period. There was no thrombus formation in the stent. The patient has maintained good blood flow in cerebral artery, for 4 years after surgery. She had high inflammatory response such as high C-reactive protein (CRP) level and prolonged gastrointestinal symptoms such as diarrhea. The diagnosis of digestive system disorder was made by gastrointestinal fiber and colon fiber. Pathological features of the colon lesion and clinical symptoms supported the diagnosis of Crohn's disease. Administration of steroid and immunosuppressant were continued, according to the intestinal symptom.

3. DISCUSSION

There have been case reports of carotid artery stenosis or obstruction associated with Crohn's disease (24-26). Inflammation of the vascular endothelium, narrowing of the vascular lumen due to reactive changes, and decreased blood flow due to atheromatic changes are thought to be the underlying causes.

Unlike atherosclerotic exchange, thrombosis is the main cause of blood flow disorders. Therefore, not only major arteries but also sinus thrombosis have been reported (7,8). Carotid artery imaging with MRI also reveals localized areas of large plaques.

However, in many cases, the bifurcation of the internal carotid artery, which is a common site of atheromatous change, is not involved. In addition, the surface of the stenotic lesion is smooth, and the plaque may be mobile along the carotid blood flow. In the present case, there was a localized lesion distal to the bifurcation, with a smooth surface and the appearance of a free-floating thrombus with low echoic feature. Furthermore, the blood flow velocity at the stenosis of the ICA was markedly reduced due to severe distal stenosis.

Various treatment options for free-floating thrombus have been reported, including conservative treatment with antithrombotic drugs and revascularization with carotid endarterectomy (CEA) or CAS (27-31). Because of the small number of clinical cases and the wide variety of symptoms, there is no conclusion that any of these treatment methods is the best (29, 30). In the present case, the patient had only visual field symptoms but had progressed without improvement. The blood flow velocity in the most stenotic area was markedly reduced. Considering the severity of ischemic symptoms

in the event of complete occlusion, CAS was performed urgently.

Inflammation causes vascular injury (32-37). The constant hypercoagulability of the coagulation system against a background of chronic inflammatory disease leads to thrombophilia. Inflammatory cytokines damage the vascular endothelium, and localized hypercoagulation leads to localized plaque formation. Furthermore, IBD is predominantly an intestinal inflammatory condition, which can easily lead to intravascular dehydration and promote thrombus formation. Some reports of cerebral ischemia in cases of Crohn's disease with Takayasu's disease have a background of cerebral vascular stenosis and not thrombus formation (38-40).

Takayasu disease, a type of vasculitis, causes permanent stenosis of major vessels in the head and neck region. There have also been reports of coexistence with Crohn's disease. Takayasu's disease is an inflammatory disease similar to Crohn's disease and tumor necrotic factor alpha (TNF- α) and interleukin (IL) -6 have been considered responsible molecules and therapeutic targets. Recently, it has been reported that component p40 of IL-23, which is an inflammatory cytokine categorized in the IL-12 families, is strongly related to the inflammatory disease. Molecule p40 induces granulomatous reaction and mononuclear inflammation, which are the pathophysiology of inflammation in Takayasu's disease and Crohn's disease (41-47). IL-23 is hetero dimeric molecule composed with p19 subunit and p40 subunit, that is common molecule with IL-12 subunit. IL-23 activates the Janus kinase/signal transducer and activator of transcription (JAK/STAT) pathway via IL-23 receptor. Activated helper T 17 cell (Th17) produces IL-17A, IL-17F, IL-21 and IL-22, those contribute to inflammatory reaction (42-46, 48, 49). Further pathophysiological studies are still underway. There are also reports that significant thickening of IMT does not necessarily correlate with Crohn's disease (50). It seems that there are some factors that cannot be unambiguously established as the cause of carotid stenosis and occlusion.

In the case of a young stroke with focal major vessels lesions, it seemed necessary to evaluate factors other than systemic vasculitis and coagulation abnormalities. Based on this case and previous reports, we also considered it necessary to evaluate for IBD complications. In this case, blood cytokine and inflammatory parameters were not evaluated. Further close examination would have been necessary.

4. CONCLUSION

Middle age woman with Crohn's disease had a carotid free-floating thrombus accompanied with ischemic attack. Emergency CAS was performed without any ischemic disorders. Inflammatory arteritis might have been based on the focal thrombosis. Chronic inflammatory disease including IBD should be considered as a cause of ischemic stroke, without underlying lifestyle-related diseases or hemo-clotting disorders.

5. CONFLICT OF INTEREST

The author declares no conflict of interest.

6. ACKNOWLEDGEMENTS

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