



Common carotid arterial thrombosis associated with ulcerative colitis

Hitoshi Nogami, Tsuneo Iiai, Satoshi Maruyama, Tatsuo Tani, Katsuyoshi Hatakeyama

Hitoshi Nogami, Tsuneo Iiai, Satoshi Maruyama, Tatsuo Tani, Katsuyoshi Hatakeyama, Division of Digestive and General Surgery, Department of Surgery, Niigata University, Niigata, Japan
Correspondence to: Hitoshi Nogami, MD, Division of Digestive and General Surgery, Department of Surgery, Niigata University, School of Medicine, Asahimachidouri1-757 Niigata, 951-8510, Japan. h-nogami@med.niigata-u.ac.jp
Telephone: +81-25-2272228 Fax: +81-25-2270779
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Abstract

A 26-year-old woman with ulcerative colitis was transferred to our hospital with left hemiparesis due to cerebral infarction. Cervical ultrasonography and magnetic resonance imaging angiography revealed thrombosis at the right common carotid artery and the right internal carotid artery. Antithrombotic and anticoagulant therapies were commenced. After about 2 wk of the treatment, the frequency of her diarrhea increased. She underwent emergency subtotal colectomy, but 10 d later an abundant hemorrhage from the remnant rectum occurred, so the remnant rectum was resected and an ileal pouch anal anastomosis was performed. Antithrombotic and anticoagulant therapies were continued, but neither her neurological status nor magnetic resonance imaging angiography findings showed subsequent changes. She was discharged 3 mon after operation. This is a rare case of common carotid arterial thrombosis occurring as a complication of ulcerative colitis, in which antithrombotic and anticoagulant therapies are considered to provoke a deterioration of the patient's bowel disease.

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Key words: Thrombus at the common carotid artery; Antithrombotic therapy; Anticoagulant therapy; Bleeding from the remnant rectum; Ulcerative colitis

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INTRODUCTION

Ulcerative colitis (UC) is often complicated with various extra-intestinal manifestations during the course of the disease. Of the secondary conditions, thromboembolic events are rare but well-recognized^[1-4]. Furthermore, cerebral infarction due to arterial thrombus occurs as a very rare complication. However, the treatment of thrombosis associated with UC is controversial, and the indications for surgery have not been established. Here, we report a case of UC in a young woman who suffered from cerebral infarction due to thrombosis of the common carotid artery.

CASE REPORT

A 26-year-old woman was admitted to another hospital on January 23, 2004 for frequent bloody diarrhea. A diagnosis of UC relapse was made and she was administered soluble prednisolone. Three days after admission, she was found lying on the floor. She was conscious and alert, but physical examination revealed left hemiparesis. Urgent computed tomography showed an area of low density in the right temporal lobe (Figure 1), and a diagnosis of cerebral infarction was made. Because of concerns that bleeding from the large intestine might increase, she did not undergo aggressive anticoagulant therapy, but only received osmotic diuretics. After 1 wk of the therapy, no improvement was found in her bloody diarrhea and hemiparesis, so she was transferred to our hospital on February 3, 2004.

On admission, she was moderately emaciated (48 kg, 162 cm), and her temperature was 37.8°C. She was conscious, with left hemiparesis but no pathological reflexes. The conjunctivae were anemic but not icteric. A total dose of steroids of 8350 mg was administered for about 4 years. Her electrocardiogram was normal but biochemical tests revealed severe anemia (hemoglobin, 6.7 g/dL, hematocrit, 21.5%), hypoproteinemia (total protein, 4.6 g/dL, albumin, 2.3 g/dL) and an elevation of her C-reactive protein levels and erythrocyte sedimentation rate. A coagulation test revealed no signs of hypercoagulability such as thrombocytosis. Immunological examination revealed no autoimmunoantibody, and Lupus anti-coagulant was also negative. Her protein C level (105%), APL (111%) and PLG (93%) were within normal range.

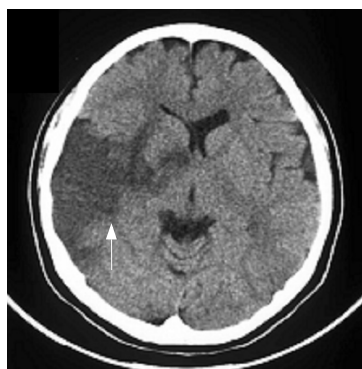


Figure 1 Plain computed tomography of brain indicates a low-density area in the right temporal lobe.

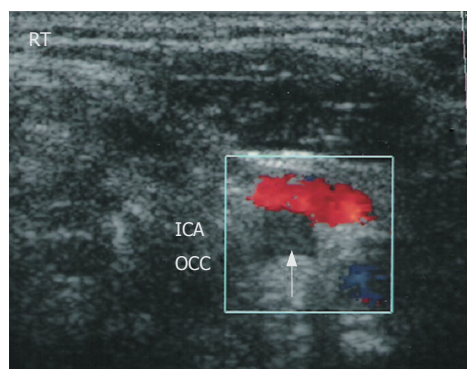


Figure 2 Cervical ultrasonography reveals a complete occlusion at the right internal carotid artery (arrow).

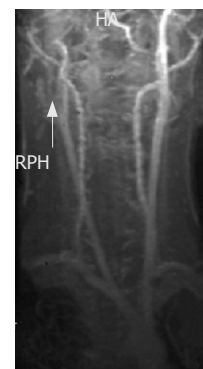


Figure 3 MRA shows occlusion at the right common carotid artery (arrow).

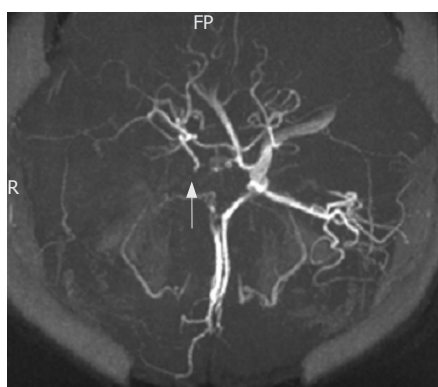


Figure 4 The right middle cerebral artery was not detectable (arrow).

Cervical ultrasonography found a complete occlusion of the right internal carotid artery (Figure 2). In magnetic resonance imaging angiography (MRA), the right common carotid artery was occluded (Figure 3) and the right middle cerebral artery was not detectable (Figure 4). Ultrasonic cardiography did not reveal any thrombus in the left atrium. It was suspected that the cerebral infarction was a complication of her UC. Heparin (15 000 units/d) was administered for the cerebral infarction, and conservative treatment for UC (soluble prednisolone) was continued. Fourteen days after admission, remarkable bloody diarrhea occurred (53 episodes/d), and we decided to perform an emergency subtotal colectomy. As thrombosis was still an active disease, we decided to continue the anticoagulant therapy. Ten days after the first operation, massive bleeding from the remnant rectum occurred, so we resected the remnant rectum and colon, and performed an ileal pouch anal anastomosis, with a diverting ileostomy. Antithrombotic therapy was continued. No subsequent changes were observed in the patient's neurologic status, including her left hemiparesis. Three months after operation, she was transferred to another hospital for rehabilitation.

DISCUSSION

Thromboembolic complications of UC are uncommon with a reported incidence of 1.2%-7.5%, but well

recognized because of their high mortality^[1-4]. Cerebral infarction associated with UC is particularly rare, but is clinically important because of its high morbidity and mortality^[5-7]. Prior to this case, there had been only 2 reported instances of cerebral infarction caused by carotid arterial thrombosis in association with UC^[8,9].

The pathogenesis of thrombosis in UC remains unclear^[7,10-15]. Many reports have suggested that hypercoagulability in UC may predispose patients to arterial and venous thromboses^[11-15]. Previous reports also indicated that prothrombotic events occur more frequently during the active phase of UC than during remission^[8,9,11,15]. However, there have been some case reports of thromboembolic events occurring during the remission phase of UC. In the present case, the cerebral infarction occurred during a rapid recurrence of UC, and the relapse of UC may have triggered the thrombosis.

Antithrombotic therapies for thromboembolism associated with inflammatory bowel disease (IBD) are controversial^[16,17]. Anticoagulant therapy is considered by some authors to be unnecessary and potentially dangerous, because of the possibility that it may promote mucosal bleeding from the UC lesion. In contrast, others have suggested that aggressive anticoagulant therapy may be useful for thrombosis that arises as a complication of IBD^[18,19]. In the present case, only an osmotic diuretic was used in the early stage of the cerebral infarction. We should mention that it was late to use heparin for thrombosis. Although the hemorrhage might deteriorate, we used aggressive anticoagulant therapy because thrombosis of the common carotid artery was detected by cerebral ultrasonography and MRA. Unfortunately, her bloody stool increased markedly, and she twice required emergency surgery. After ileal pouch anal anastomosis, her general condition improved, and anticoagulant therapy was continued safely. However, her left hemiparesis did not improve and thrombus was still detected at the right common carotid artery by cervical ultrasonography and MRA. Although the thrombus unlikely disappear, anticoagulant therapy has been continued to prevent extension of the thrombus. Ongoing rehabilitation will be needed to improve her mobility.

Several factors may explain thromboembolism in IBD. Platelets and coagulation cascade abnormalities are

likely to play a key role in clot formation in IBD^[11,12,20]. Thrombophilias are found in 33%-50% of patients in the general population with venous thromboembolism^[21,22], and factor V Leiden mutation or activated protein C (APC) resistance is the most frequent genetic abnormality^[15]. In IBD patients, the incidence of APC resistance or factor V Leiden mutation might not be increased^[13,23,24]. While APC resistance itself is not possibly associated with IBD, it does appear to increase the risk of thromboembolic events when present^[14]. In this case, the platelet count was within the normal range, but neither platelet aggregation nor coagulation cascade activation was examined, and no thrombophilia evaluation was performed.

Craig *et al.*^[15] suggested that due to the relatively high frequency of identifiable thrombophilia, all IBD patients with venous thromboembolism should undergo a comprehensive thrombophilia evaluation. They also suggested that the presence of a hypercoagulable condition might be a relative indication for lifelong anticoagulation with Warfarin^[18].

Some authors have reported that medically treated patients develop thrombotic complications more often than surgically treated patients^[2,3]. However, a rare instance of cerebral venous thrombus 10 years after panproctocolectomy has been recorded^[25], and Craig *et al.*^[15] reported that proctocolectomy is not protective for recurrent thromboembolism. Our patient needed proctocolectomy for abundant hemorrhage from the large intestine in poor general condition. It is unlikely that marked bloody diarrhea is associated with conditions that promote the development of thrombus. If evaluations for thrombophilias were performed and at least one of them was identified, bloody diarrhea might be a relative indication for proctocolectomy. We consider that early proctocolectomy should have protected our patient from thromboembolism.

We have described here our experience with one case of common carotid arterial embolism associated with UC. Cervical ultrasonography and MRA were useful for identification of the thrombus. Further work is required to clarify the correlation between IBD and the thrombophilias, and to evaluate the role of anticoagulant therapy and proctocolectomy in the management of these patients.

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