Cerebral Sinovenous Thrombosis in a Patient with Transient Eosinophilia

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Abstract

A 67-year-old man with a history of prostatic hypertrophy developed behavioral anomalies and a fever. At admission, diffusion-weighted brain magnetic resonance imaging (MRI) indicated a high-intensity signal lesion on both sides of the cerebral hemisphere. Immediately after hospitalization, he developed paralysis of the left side of the body. Diffusion-weighted MRI indicated hemorrhagic changes in the right and left parietal lobes and right occipital lobe. Magnetic resonance venography indicated superior sagittal sinus occlusion. The blood test results indicated transient eosinophilia. Cases of thrombosis with idiopathic eosinophilia have been reported, but this is the first known case of cerebral sinus thrombosis with transient eosinophilic leukocytosis.

Key words: cerebral sinovenous thrombosis, thromboembolism, transient eosinophilia

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Introduction

Eosinophilia is often complicated by thromboembolisms, such as a mural thrombus, cerebral infarction or retinal vessel microthrombus (1, 2). In addition, cases of thrombosis in patients with transient eosinophilia have been reported (3-9). In the present report, we describe a case of cerebral sinovenous thrombosis associated with transient eosinophilia; to our knowledge, no such case has previously been reported in the literature.

Case Report

A 67-year-old man with a history of prostatic hypertrophy presented to a hospital with a slight fever. The patient's white blood cell count was elevated [16,000/ μ L (neutrophils 89%; eosinophils, 3.8%)] and the C-reactive protein (CRP) level was 17.49 mg/dL. The findings of thoracoabdominal computed tomography (CT) and upper endoscopy were normal.

However, after three days, he was unable to change his clothes and perform calculations. He visited a hospital, and underwent diffusion-weighted brain magnetic resonance im-

aging (MRI), which indicated the presence of a high-intensity signal lesion in the left temporal lobe, right temporal lobe and right occipital lobe (Fig. 1).

He was transferred to our department, and exhibited a body temperature of 36.4°C, blood pressure of 185/76 mmHg, pulse rate of 93 beats/min and significant limb edema on physical examination. His Glasgow Coma Scale score was E4V4M6, and he exhibited a slow verbal response. Moreover, he showed dressing apraxia and acalculia.

The blood test results indicated a red blood cell count of 406×10^4 /L, a hemoglobin level of 15.8 g/dL, a hematocrit value of 44.6%, a white blood cell count of 14,030/ μ L, a platelet count of 180×10^3 /L and a percentage of eosinophils of 26.4%. In addition, the levels of CRP, D-dimer and fibrin degradation product-P were 4.69 mg/dL, 6.08 μ g/mL and 11.9 g/mL, respectively. During the cerebrospinal fluid examination, we noted that the pressure was 380 mmH₂O, the cell count was 1/mm³ and the protein level was 18 mg/dL. Furthermore, the levels of PR3-ANCA, MPO-ANCA, protein C, protein S, antithrombin III and anticardiolipin antibody were normal. The findings of echocardiography were also normal.

However, immediately after hospitalization, the patient exhibited paralysis of the left side of the body, as well as

Figure 1. A fluid-attenuated inversion recovery magnetic resonance image of the brain on the admission. Hyperintense areas can be noted in the left temporal lobe and right parietal and occipital lobes.

Figure 2. Magnetic resonance venography on admission to our hospital indicated the occlusion of the superior sagittal sinus. A fluid-attenuated inversion recovery magnetic resonance image indicating the presence of hyperintense areas in the right and left parietal lobes and right occipital lobe.

headache and disturbance of consciousness. Fluid-attenuated inversion recovery (FLAIR) MRI of the brain indicated the presence of a hyperintense area in the right and left parietal lobe and right occipital lobe. Magnetic resonance venography indicated occlusion of the superior sagittal sinus (Fig. 2). We diagnosed the patient with hemorrhagic changes, along with thrombosis of the venous sinuses in the brain. On the same day, we performed hematoma removal and cerebral decompression for a right brain hemorrhage. The eosinophil count reached a peak level (5,130/ μL) on the fourth day and normalized (1,018/ μL) on the fifth day. Treatment with heparin and warfarin was initiated on the fourth day. On the 14th day, magnetic resonance venography still indicated the presence of obstruction at the left transverse sinus and upper sagittal sinus (Fig. 3). However, the D-dimer levels gradually decreased, and showed a reduced level of 0.6 $\mu\text{g/mL}$ on the 33rd day. Although the disturbance of consciousness was also improved, left hemiplegia

was still observed and the patient was transferred to another hospital on the 38th day for rehabilitation.

Discussion

To our knowledge, the present case is the first reported case of cerebral sinovenous thrombosis caused by transient eosinophilia. The causes of cerebral sinovenous thrombosis include drugs, such as oral contraceptives, androgens and danazol; prothrombotic conditions, such as antithrombin III deficiency, protein C deficiency and protein S deficiency; infection; cancer-related factors; antiphospholipid and anticardiolipin antibodies and mechanical precipitants (10). None of these causes were observed in the present case. According to Chusid et al, the criteria for hypereosinophilic syndrome include: (1) eosinophilia (1,500 eosinophils/ mm^3) persisting for more than six months, or death within six months that is associated with the signs and symptoms of hypereosino-

Figure 3. Magnetic resonance venography 14 days after admission indicated that there was occlusion of the transverse sinus and superior sagittal sinus.

Table. Reported Cases of Thromboembolic Events in Patients with Transient Eosinophilia

Reference	Age (years) /Sex	WBC count (/μL)	Eosinophil count (/μL)	% of Eosinophils	Duration of eosinophilia (days)	Medical history	Organ involvement
3	30/M	20,000	13,300	60	NR	None	Hepatic vein thrombosis
4	26/M	14,600	6,000	42	NR	Ulcerative colitis	Biventricular thrombus, right popliteal artery embolus
5	29/F	10,400	3,120	30	11	None	Pulmonary embolism, DVT
6	24/F	22,800	9,350	41	21	None	Pulmonary embolism, DVT, hepatic vein thrombosis
7	19/M	18,500	8,000	40	NR	Asthma	Pulmonary embolism, DVT
8	40/F	12,600	6,930	55	NR	Psoriasis, atopic dermatitis	Splenic, portal, and superior mesenteric vein thrombosis
9	21/F	NR	5,900	NR	NR	None	Portal and splenic vein thrombosis
Present case	67/M	18,320	5,130	28	3	Prostatic hypertrophy	Cerebral sinovenous thrombosis

NR: not reported, WBC: white blood cells, DVT: deep vein thrombosis

philic disease; (2) a lack of evidence of parasitic, allergic or other known causes of eosinophilia and (3) presumptive signs and symptoms of organ involvement, including hepatosplenomegaly, organic heart murmur, congestive heart failure, diffuse or focal central nervous system abnormalities, pulmonary fibrosis, fever, weight loss and anemia (11). In the present case, the eosinophilia did not persist for more than six months. Therefore, we could not diagnose the patient with hypereosinophilic syndrome.

Idiopathic hypereosinophilic syndrome has been reported to cause thrombosis in the cardiac chamber (12), coronary artery (13), mesenteric artery (14), brachial artery (15), limb vessels (16), hepatic vein (17) and central sinovenous area (18-20). Moreover, seven cases in which thrombosis was caused by transient eosinophilia have been reported (Table). These reports indicate that these thromboses occurred at a relatively young age (range, 19-40 years) and primarily occurred in the hepatic vein, lung vessels, splenic vein, portal vein and popliteal artery.

Thrombomodulin (TM) is known to play a critical role in the anticoagulation system (21). The functional impairment of TM may potentially induce a hypercoagulable state. Mukai et al. reported that mature major basic protein (MBP), which is elevated in the sera of patients with eosinophilia, inhibits the activity of the TM cofactor, thus indicating the potential contribution of eosinophil granule proteins to thrombosis in patients with eosinophilia (22). Thus, we believe that increased levels of eosinophil granule proteins, including MBP, contribute to thrombotic diathesis in patients with eosinophilia. It should be noted that the thrombosis in the present case appear to have been caused by the eosinophilia, even though it only occurred transiently.

The authors state that they have no Conflict of Interest (COI).

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