

# World Journal of *Clinical Cases*

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Editorial Board Member of *World Journal of Clinical Cases*, Domenico De Berardis, MD, PhD, Adjunct Professor, Chief Doctor, NHS, Department of Mental Health, Teramo 64100, Italy. domenico.deberardis@aslteramo.it

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# Hypereosinophilia with cerebral venous sinus thrombosis and intracerebral hemorrhage: A case report and review of the literature

Xiu-Hua Song, Tian Xu, Guo-Hua Zhao

**ORCID number:** Xiu-Hua Song 0000-0002-9514-8184; Tian Xu 0000-0002-4393-4829; Guo-Hua Zhao 0000-0001-7943-209X.

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**Xiu-Hua Song, Tian Xu, Guo-Hua Zhao**, Department of Neurology, The Fourth Affiliated Hospital, Zhejiang University School of Medicine, Yiwu 322000, Zhejiang Province, China

**Corresponding author:** Guo-Hua Zhao, MD, Director, Doctor, Department of Neurology, The Fourth Affiliated Hospital, Zhejiang University School of Medicine, No. 1 Shangcheng Avenue, Yiwu 322000, Zhejiang Province, China. [gzhao@zju.edu.cn](mailto:gzhao@zju.edu.cn)

## Abstract

### BACKGROUND

Hypereosinophilia (HE) is defined as a peripheral blood eosinophil count of  $> 1.5 \times 10^9/L$  and may be associated with tissue damage. The clinical presentations of HE vary; however, myocardial fibrosis and thrombosis can threaten the lives of patients with sustained eosinophilia. Cerebral venous sinus thrombosis (CVST) in the setting of eosinophil-related diseases has seldom been reported. Here, we review the literature on HE with CVST to increase knowledge and encourage early diagnosis.

### CASE SUMMARY

A previously healthy 41-year-old man was admitted to hospital with diarrhea and abdominal pain. He was treated with antibiotics for suspected acute colitis. Three days later, he experienced headache and vomiting. Brain computed tomography (CT) revealed thrombosis of the left jugular vein to the left transverse sinus vein. Platelet (PLT) count decreased to  $60 \times 10^{12}/L$ , and absolute eosinophil count (AEC) increased to  $2.41 \times 10^9/L$ . He was treated with low-molecular-weight heparin. PLT count progressively decreased to  $14 \times 10^9/L$ , and we terminated anticoagulation and performed PLT transfusion. Six days after admission, he complained of a worsening headache. Brain CT revealed right temporal lobe and left centrum semiovale intracerebral hemorrhage, and AEC increased to  $7.65 \times 10^9/L$ . We used prednisolone for HE. The level of consciousness decreased, so emergency hematoma removal and decompressive craniectomy for right cerebral hemorrhage were performed. The patient was alert 2 d after surgery. He was treated with anticoagulation again 2 wk after surgery. Corticosteroids were gradually tapered without any symptomatic recurrence or abnormal laboratory findings.

### CONCLUSION

HE can induce CVST, and we need to focus on eosinophil counts in patients with CVST.

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**Core Tip:** Thromboembolism is a rare but serious complication of hypereosinophilia (HE). We report a 41-year-old man who presented with colitis, cerebral venous sinus thrombosis (CVST), and intracerebral hemorrhage caused by HE. Blood eosinophil count decreased quickly after corticosteroid therapy, and CVST caused headache, which improved after anticoagulation therapy. Good clinical outcomes were observed during a 6-mo follow-up period. We conclude that HE can induce CVST, and we need to focus on eosinophil counts in patients with CVST. Corticosteroids are a useful first-line therapy for platelet-derived growth factor receptor A/B-negative HE.

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## INTRODUCTION

Cerebral venous sinus thrombosis (CVST) can occur in the early stages of hypereosinophilia (HE) and can be life-threatening if not identified early. We searched the Web of Science, Scopus, Embase, and PubMed up to December 2020 using medical subject headings of "eosinophilia/HE" and "cerebral venous thrombosis/sinus thrombosis" (limits: Full text available, clinical trials, human studies, and studies in English), and identified eight publications (Table 1) related to HE with CVST in eight cases[1-8]. Herein, we report a 41-year-old man who presented with HE-associated CVST and intracerebral hemorrhage (ICH). Blood eosinophil count decreased quickly after corticosteroid therapy, and CVST caused headache, which improved after anticoagulation therapy.

## CASE PRESENTATION

### Chief complaints

A 41-year-old man was admitted to the Department of Neurology of our hospital after experiencing headache and vomiting for 1 d on September 9, 2020.

### History of present illness

Six days before admission, the patient presented with diarrhea (2-3 times per day) and abdominal pain with a slight fever. Self-medication with trimebutine maleate and compound *Lactobacillus acidophilus* showed no improvement. Three days before admission, the patient visited our hospital on emergency and was treated with antibiotics (levofloxacin) for suspected colitis. The patient achieved remission from diarrhea, but still demonstrated abdominal distension. The patient did not experience chest tightness or heart palpitations. Six days after admission, he complained of a worsening headache and developed left hemiplegia. His consciousness rapidly deteriorated with signs of brain herniation. Thirteen days after admission (2 d after surgery), he had no symptoms, but he did demonstrate acne on his skin.

### History of past illness

The patient had a history of tonsillectomy, appendectomy, and kidney stones.

### Personal and family history

The patient denied a past history of drug or alcohol abuse, smoking, promiscuous sexual behavior, raw food consumption, and travel. There was no family history of

Table 1 Reported cases of cerebral venous sinus thrombosis with hypereosinophilia

| Ref.                            | Age/sex | Medical history                                               | Initial symptoms                                      | CVST                                           | ICH                                            | Thrombocytopenia     | Treatment                                                                   | Prognosis |
|---------------------------------|---------|---------------------------------------------------------------|-------------------------------------------------------|------------------------------------------------|------------------------------------------------|----------------------|-----------------------------------------------------------------------------|-----------|
| Schulman <i>et al</i> [1], 1999 | 11/M    | None                                                          | Bug bites, rash                                       | Straight sinus; superior sagittal sinus        | Brain stem                                     | No                   | Prednisone, heparin                                                         | Death     |
| Sakuta <i>et al</i> [2], 2007   | 7/M     | Seizures, taking valproate                                    | HE for 10 m                                           | Superior sagittal sinus                        | Right hemisphere                               | $50 \times 10^9/L$   | Heparin, warfarin                                                           | Cure      |
| Numagam <i>et al</i> [3], 2008  | 76/F    | None                                                          | Right mandible swelling                               | Left transverse sinus                          | Left temporal lobe                             | No                   | Prednisolone, evacuation of hematoma                                        | Death     |
| Ananth <i>et al</i> [4], 2016   | 17/M    | Asthma                                                        | Intermittent fever, dyspnea                           | Transverse sinus                               | None                                           | No                   | Prednisolone, warfarin                                                      | Death     |
| Teresa <i>et al</i> [5], 2006   | 40/F    | Rhinitis asthma, nasal polyps, taking contraceptives for 6 yr | Asthenia, myalgia, fever<br>Left arm/foot paresthesia | Superior sagittal sinus<br>Right lateral sinus | Bilateral parietal lobe                        | No                   | Heparin, warfarin                                                           | Cure      |
| Kanno <i>et al</i> [6], 2005    | 34/F    | None                                                          | Lump on left thigh                                    | Left transverse sinus                          | Left hemisphere                                | $10.4 \times 10^9/L$ | Antibiotics, corticosteroid, decompression surgery                          | Death     |
| Chan <i>et al</i> [7], 2004     | 49/F    | None                                                          | Headache, diplopia                                    | Cavernous sinus, transverse sigmoid sinuses    | None                                           | No                   | Bilateral endoscopic sphenoidectomy, antihistamines, steroids, itraconazole | Cure      |
| Sano <i>et al</i> [8], 2014     | 67/M    | Prostatic hypertrophy                                         | Slight fever                                          | Superior sagittal sinus                        | Bilateral parietal lobes, right occipital lobe | No                   | Evacuation of hematoma<br>Heparin, warfarin, anticoagulation                | Cure      |

CVST: Cerebral venous sinus thrombosis; ICH: Intracerebral hemorrhage.

neurological or blood system diseases.

### Physical examination

After admission, the patient's body temperature was 36.7 °C, respiratory rate was 16 bpm, heart rate was 42 bpm, and blood pressure was 135/72 mmHg. There was no detectable rash, bradycardia, arrhythmia, or murmur, and both lungs sounded clear with no rales. Abdominal distension without tenderness was noted, and the patient's neurological examination was normal.

### Laboratory examinations

On September 6, 2020, white blood cell count was elevated ( $12.6 \times 10^9/L$ ) with an absolute eosinophil count (AEC) of  $0.97 \times 10^9/L$ , hemoglobin concentration of 155 g/L, platelet (PLT) count of  $238 \times 10^{12}/L$ , and C-reactive protein concentration of 110 mg/L. On September 9, 2020, PLT count decreased to  $60 \times 10^{12}/L$ , AEC increased to  $2.41 \times 10^9/L$ , D-dimer increased to 50.22 mg/L, prothrombin time (PT) was 12.8 s, and activated partial PT was 36.2 s. Total immunoglobulin E was 389.4 IU/mL, erythrocyte sedimentation rate was 21 mm/h, and glycan antigen was 125 U/mL, but a serological allergen screen was negative. Tests for parasites in the stool were negative. Anti-neutrophil cytoplasmic, antinuclear, and cardiolipin antibodies were negative. Procalcitonin, troponin, cardiac enzymes, liver and kidney function, folic acid, vitamin B12, and thyroid function were all normal.

Serology for human immunodeficiency virus, hepatitis viruses, and *Treponema pallidum* was normal. Bone marrow biopsy showed normal cellularity with increased eosinophils (35%). *FIPL1* and platelet-derived growth factor receptor (*PDGFR*) A/B gene fusion and chromosomal analysis were normal. However, AEC progressively

increased up to  $7.65 \times 10^9/L$ , PLT count progressively decreased to a minimum of  $14 \times 10^9/L$ , and D-dimer continuously increased to 69.76 mg/L (Figure 1).

### Imaging examinations

On September 6, 2020, abdominal computed tomography (CT) revealed swelling with peripheral exudation changes in the wall of the transverse and sigmoid colon. On September 9, 2020, brain CT revealed thrombosis of the left jugular vein to the left transverse sinus vein (Figure 2A). Chest CT demonstrated inflammation in both lungs.

Electrocardiography showed significant sinus bradycardia with irregularity, sinus arrest, and frequent borderline escape, with normal myocardial enzymes and troponin. Echocardiography showed no abnormal findings. On September 15, 2020, brain CT revealed right temporal lobe and left centrum semiovale ICH (Figure 2B and C). Chest CT showed bilateral pleural effusion. Duplex ultrasonography showed bilateral intermuscular venous thrombosis of the calf.

On September 30, 2020, enhanced cranial magnetic resonance venogram showed stenosis of the left internal jugular vein, transverse sinus, sigmoid sinus, confluent sinus, straight sinus, and inferior sagittal sinus (Figure 3A).

## FINAL DIAGNOSIS

HE induced CVST, and the cause of HE was unknown, so the diagnosis changed from HE to idiopathic HE syndrome (HES). Thrombocytopenia may have been related to consumptive reduction by thromboembolism, and ICH occurred secondarily to thrombocytopenia.

## TREATMENT

The patient was treated with antibiotic agents due to suspected colitis, and low-molecular-weight heparin was initiated after the diagnosis of CVST. When PLT count decreased to  $14 \times 10^9/L$ , we terminated anticoagulation and antibiotic agents and performed PLT transfusion. We used intravenous immunoglobulin (32 g/d for 5 d) for suspected heparin-induced thrombocytopenia (HIT) and prednisolone 80 mg/day due to HE after ICH. Hematoma removal and decompressive craniectomy for right ICH were performed after PLT count increased to  $80 \times 10^9/L$  after PLT transfusion. The patient was treated with anti-coagulation again (heparin and warfarin) 2 wk after surgery. Corticosteroids were gradually tapered, and the total treatment course was 3 mo. Warfarin was continued, and the patient still uses it today.

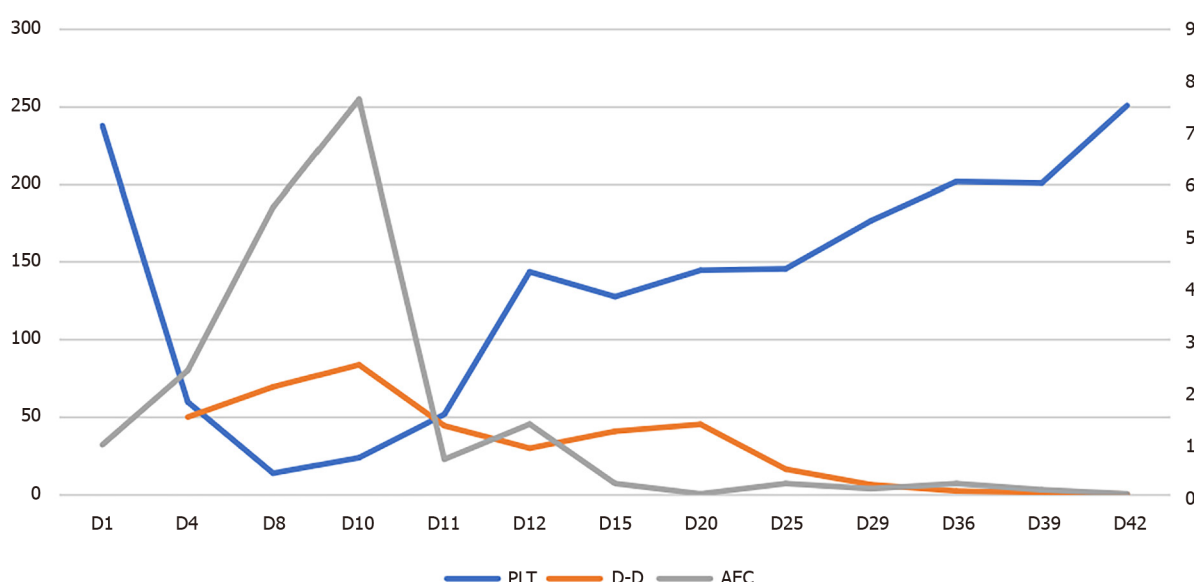
## OUTCOME AND FOLLOW-UP

A 6-mo follow-up showed that the patient did not experience any further symptoms. However, thrombosis in the left internal jugular vein, transverse sinus, sigmoid sinus, and confluent sinus, as well as partial venous thrombosis in the straight sinus and inferior sagittal sinus, was unchanged (Figure 3B).

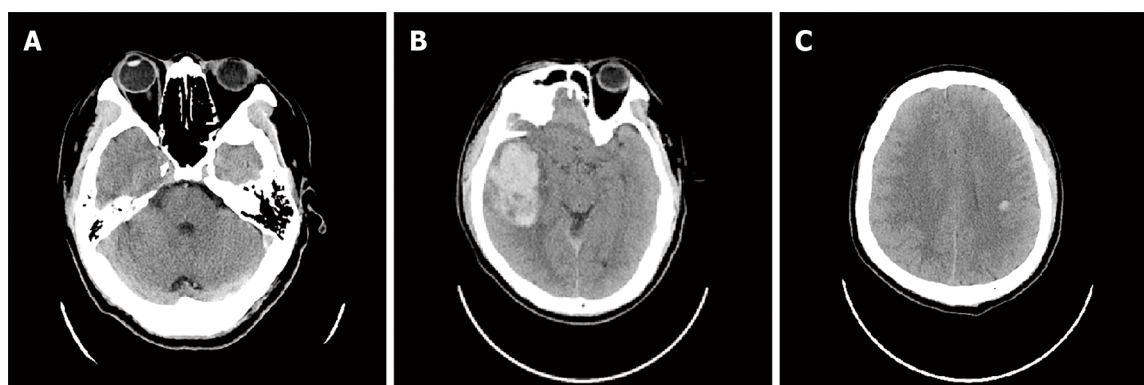
## DISCUSSION

Eosinophils are multifunctional granular leukocytes that represent approximately 3%–5% of circulating blood leukocytes with an AEC in healthy adults of  $0.35\text{--}0.5 \times 10^9/L$ . Eosinophils are normally present in gastrointestinal tract, except in the squamous esophagus, and are important in homeostasis and reconstitution of tissue[9]. Eosinophilia encompasses a broad range of non-hematologic (secondary or reactive) and hematologic (primary or clonal) disorders with potential for end-organ damage[10]. HE is usually linked to allergies, infections (parasitic or fungal), drugs, neoplastic disorders, autoimmune diseases, and atopy[9,10]. When blood HE induced organ damage, the diagnosis changes from HE to HES[9].

Of the cases reported in the literature, three occurred secondarily to eosinophilic granulomatosis, one was secondary to an allergic reaction to a fungus, and three was idiopathic HES. Our case was also idiopathic HES.



**Figure 1 Blood indexes.** Platelets, D-dimer, and absolute eosinophil count gradually recovered after addition of corticosteroid and anti-coagulation therapy. PLT: Platelets; D-D: D-dimer; AEC: Absolute eosinophil count.

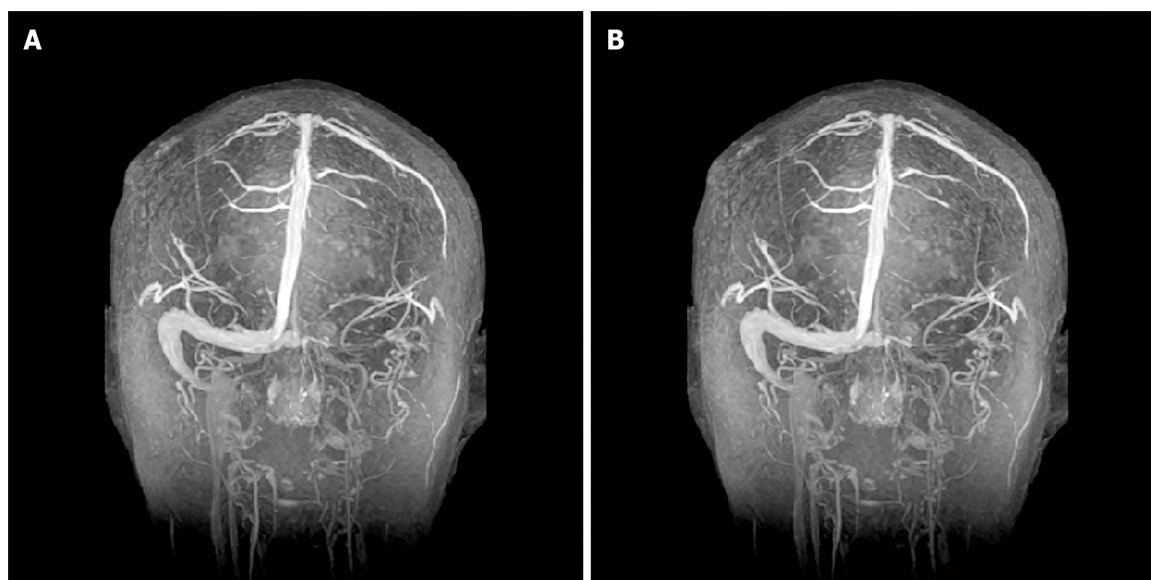


**Figure 2 Brain computed tomography.** A: Thrombosis of the left jugular vein to the left transverse sinus vein; B: Right temporal lobe intracerebral hemorrhage; C: Left centrum semiovale intracerebral hemorrhage.

Peripheral blood eosinophilia can occur in patients with inflammatory bowel disease[11-13], and peripheral blood eosinophilia may be a biomarker of disease severity. Eosinophilic colitis (EC) should be suspected in any patient with intestinal symptoms with peripheral blood eosinophilia, but EC is a rare condition[14]. When accompanied by peripheral blood HE, colitis can occur as an isolated gastrointestinal disorder or as part of HES[15]. Our patient's colitis may be a part of HES.

Thrombosis is one of the most serious HE-related organ damage. It has been suggested that approximately one-quarter of patients with HES develop thromboembolic complications and that 5%–10% die as a result of these complications[16]; however, CVST is rare. Eosinophils release rich tissue factors (TFs)[17] and provide a procoagulant phospholipid surface that supports TF-mediated thrombin generation[18]. Eosinophil cationic protein activates factor XII, which promotes internal coagulation[19]. Major basic protein (MBP) and eosinophil peroxidase activate PLTs[20,21]. Binding of MBP to thrombomodulin inhibits anticoagulant activities[22,23]. Tissue and endothelial damages through direct cytotoxic effects or indirect recruitment and activation of other inflammatory cells increase vascular permeability, which may contribute to a procoagulant state.

Thrombocytopenia is more common than thrombocytosis in patients with HE (31% vs 16%, respectively)[24]. The mechanisms of thrombocytopenia in HE are not fully understood, but may be related to consumptive thrombocytopenia caused by thromboembolism. In a previous study, thrombocytopenia was present in five of ten patients with central nervous system involvement but no thrombosis of HE[25]. In our review, two of eight cases reported in the literature developed thrombocytopenia,



**Figure 3 Magnetic resonance venogram.** A: Thrombosis in the left internal jugular vein, transverse sinus, sigmoid sinus, confluent sinus, partial venous thrombosis in the straight sinus and inferior sagittal sinus; B: The thrombus was smaller than before.

which may have been underpinned by an immunological mechanism. One case of thrombotic thrombocytopenic purpura with HE was caused by an ADAMTS13 inhibitor[26]. Other cases of HE with an initial presentation of idiopathic thrombocytopenia have been reported[27]. Spontaneous HIT is rare and may be associated with antibodies against platelet factor-4, but it may occur without previous heparin exposure[28,29].

HE with ICH was accompanied by CVST in five of eight cases reported in the literature. Of these, three deaths were observed due to cerebral herniation. Multiple mechanisms may account for ICH. ICH may occur secondarily to CVST or thrombocytopenia, as a side effect of anticoagulant drugs, or due to direct endothelial injury or vasculitis caused by eosinophilic infiltration.

Corticosteroids are a first-line therapy for PDGFRA/B-negative HE[10]. HE can induce CVST, and we need to focus on eosinophil counts in patients with CVST. Early initiation of steroid therapy can potentially prevent disease progression. Persistent eosinophilia is associated with a shorter time to thromboembolism relapse[30], so the goal of therapy is to maintain the eosinophil count below 1500/ $\mu$ L.

## CONCLUSION

HE can induce CVST, and we need to focus on eosinophil counts in patients with CVST. Corticosteroids are a useful first-line therapy for PDGFRA/B-negative HE and HES.

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