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Case Report

# Cerebral venous thrombosis secondary to ulcerative colitis: A case report with a literature review ☆,☆☆,★

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ABSTRACT

Ulcerative colitis is an idiopathic inflammatory bowel condition that may be worsened by thromboembolic events such deep vein thrombosis, cerebral venous thrombosis, and pulmonary embolism. Cerebral venous thrombosis is a rare but critical consequence of ulcerative colitis characterized by high mortality and morbidity rate. It is thought to be caused by the hypercoagulable state that occurs during ulcerative colitis relapse. Cerebral venous thrombosis is a reversible condition with good outcomes when detected early and treated properly. In this study, we describe the case of a young woman who presented with cerebral venous thrombosis secondary to ulcerative colitis complicated by venous infarction with petechial cerebral hemorrhage.



## Introduction

Ulcerative colitis (UC) is an idiopathic inflammatory condition of the colon with an unknown etiology. It constitutes with Crohn's disease an entity called inflammatory bowel disease (IBD). UC can be complicated by various extra-intestinal manifestations such as venous thromboembolic events, particularly in the active phase of the condition and eventually due to its prothrombotic state [1].

The association between venous thromboembolic events and IBD was first reported in 1936, and its incidence ranges from 0.5% to 6% [2]. Despite the significant prevalence of venous thromboembolic events, clinicians still do not fully understand this risk. We present an unusual case of a young

*Abbreviations:* UC, ulcerative colitis; IBD, inflammatory bowel disease; CVT, cerebral venous thrombosis; MRI, magnetic resonance imaging; MRV, magnetic resonance venography; SSS, superior sagittal sinus.

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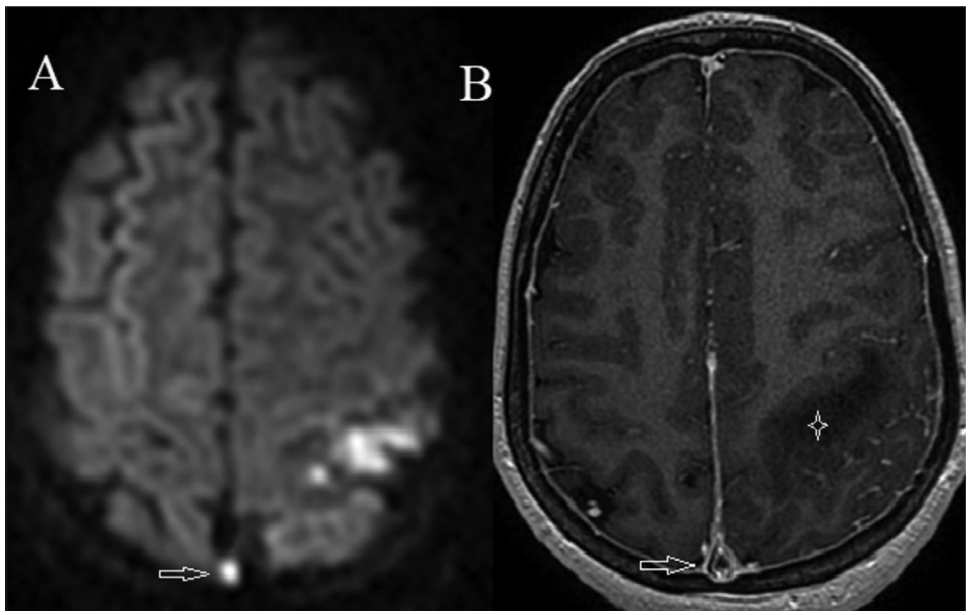


**Fig. 1 – Cerebral MRI on diffusion sequence (A) shows high signal intensity of the left parietal lobe with punctiform hyperintensity of the superior sagittal sinus (arrow). Axial T1 contrast-enhanced sequence (B) demonstrates delta sign of the superior sagittal sinus (arrow) with hypointensity in the left parietal lobe (asterisk).**

woman who presented with cerebral venous thrombosis (CVT) concurrent with a relapse of UC complicated by venous infarction with petechial cerebral hemorrhage.

## Case report

A 34-year-old woman with a history of ulcerative colitis for 3 years was hospitalized at the gastroenterology and hepatology department for bloody diarrhea and diffuse abdominalgia. She was treated by 5-aminosalicylic which was stopped 9 months ago. The patient has never experienced a COVID-19 infection. She received 2 doses of the Pfizer/BioNTech vaccine 18 months ago without any notable complications. For the relapse of her inflammatory bowel disease, the patient received 60 mg per day of intravenous methylprednisolone during 5 days.

During admission, she presented with sudden right-sided weakness, without any seizures or visual disturbance. On physical examination, the patient was conscious, apyretic with no sign of meningitis. The motor examination revealed 3/5 right hemiparesis with right muscle hypotonia. Deep tendon reflexes were absent with a right Babinski sign. The sensory exam revealed a right-sided tactile and thermo-algic hypoesthesia with normal motor coordination. The examination of the cranial nerves was unremarkable.

A few hours later, the patient presented with progressive headaches and dizziness followed by a generalized tonic-clonic seizure. Urgent magnetic resonance imaging (MRI) and venography (MRV) were performed, revealing a thrombosis of the superior sagittal sinus (SSS), complicated by a left frontoparietal venous infarction with petechial hemorrhagic lesions (Figs. 1 -3).

The opening pressure of cerebrospinal fluid was 160 mmH2O (normal opening pressure range, 60-200 mmH2O), with normal cytochemical examination. The patient had iron deficiency anemia, with 8.5 g/dL hemoglobin (reference range, 12-15 g/dL) and a ferritin level of 7 μg/L (reference range, 20-220 μg/L). Further laboratory tests (prothrombin time, fibrinogen level, D-dimers level, serum electrolytes, renal and hepatic tests, and thyroid hormone) were normal. The antinuclear and anticardiolipin antibodies were negative. Her prothrombotic workup (antithrombin III, protein C and protein S, factor V and factor VIII) was normal.

An immediate subcutaneous anticoagulation therapy with low-molecular-weight heparin (Enoxaparin 0.6 mL/12 h) was administered for 6 days. Acenocoumarol medication was continued for 6 months. The patient also received prednisolone with azathioprine as disease-modified therapy for UC. After 3 months of treatment, the clinical evolution was favorable with the resolution of neurological and digestive manifestations. After 9 months of follow-up, there was no recurrence.

## Discussion

UC is a chronic idiopathic IBD caused by a complex interaction of genetic and environmental risk factors [3]. It can be considered a multisystem disorder with various extraintestinal symptoms. Neurologic complications are diverse, uncommon, and often severe. CVT is an uncommon cerebrovascular event of UC with a significant risk of morbidity. The prevalence of CVT in IBD is reported to be between 0.5% and 6% [1].

The pathophysiology of CVT linked with UC is multifactorial and remains unknown. Excessive inflammation dur-

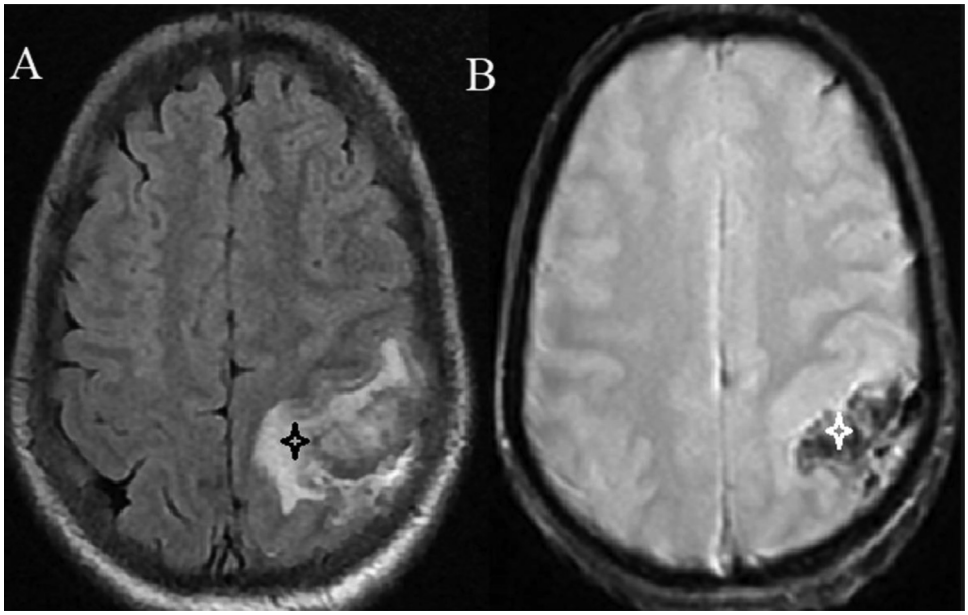


**Fig. 2 – Cranial MRI on axial Flair sequence (A) shows hypersignal intensity of the left precentral gyrus surrounding hypointense signal in the left parietal lobe (black asterisk). T2*-weighted gradient-echo sequence (B) demonstrates hypointensity lesion in the left parietal lobe (white asterisk).**

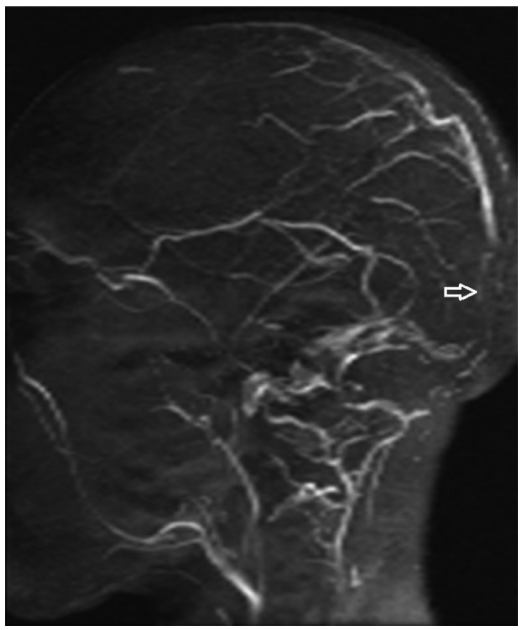

**Fig. 3 – Sagittal magnetic resonance venography shows the absence of opacification of the superior sagittal sinus (arrow).**

ing the exacerbation of UC is a severe condition that predisposes to thrombosis and dramatically raises the risk of venous thromboembolic events [4].

The hypercoagulable and hypofibrinolytic conditions caused by UC are essential risk factors for CVT. Decreased levels of protein C, protein S, and antithrombin with high levels of fibrinogen and factor VIII represent the hypercoagulable state. The hypofibrinolytic state is caused by endothelial dysfunction, platelet abnormalities with increased PAI-1 and lipoprotein [5].

Thrombocytosis and anemia have also been linked to an increased risk of CVT in patients with UC. Moreover, hospitalization, steroid treatment, and IBD-related operations have all been identified as significant risk factors for venous thromboembolic events in patients with IBD [6].

CVT clinical symptoms differ from patient to patient. Depending on the site of the thrombosis, the most common presenting symptoms are headache, convulsions, focal neurological deficits, visual disturbances, and impaired mental state [2]. CVT is usually challenging to diagnose clinically since it can present with various signs that resemble various neurological pathologies.

Neuroimaging is fundamental in the diagnosis of CVT. Although cranial CT without contrast had a relative sensitivity, it remains a useful technique for eliminating other diagnosis like hemorrhagic or ischemic stroke. MRI and MRV should be performed to provide accurate visualization of the thrombus to confirm CVT [7]. The superior sagittal sinus and the lateral sinus are the most prevalent locations of CVT. In this study, MRV and MRI confirmed the diagnosis of CVT by showing thrombosis of the SSS complicated by frontoparietal venous infarction.

The management of CVT in patients with UC is based on dissolving the thrombus and preventing its propagation. Conventional anticoagulant with low molecular weight or unfractionated heparin is recommended. Anticoagulation treatment in patients with UC does not cause more mucosal hemorrhage [8]. Parenteral anticoagulation is continued with oral vitamin K antagonists for 6 months to 1 year [9].

The prognosis of CVT tends to be more critical in patients with UC. It could be due to delay in diagnosis and reluctance to initiate anticoagulation in individuals with a high risk of gastrointestinal bleeding. Patients who did not receive heparin treatment were the most at risk of death [10]. The American College of Gastroenterology recommends preventive anticoagulation for hospitalized patients to avoid venous thromboembolic events in active IBD [9].

## Conclusion

UC is an inflammatory and prothrombotic condition especially in the active phase, which could be complicated by venous thromboembolic events. Our clinical case demonstrates the clinical obstacles and difficulties related to the suspicion, identification, and management of CVT in patients with UC. The diagnosis of CVT should be considered in UC's patients with newly onset neurological symptoms. MRI and MRV continue to be the gold standards for diagnosing CVT even though CT scan is essential to rule out some differential diagnoses. Even after the onset of remission and the removal of symptoms, early and appropriate anticoagulant therapy should be continued. We need further research to develop detailed recommendations for treating CVT in individuals with IBD.

## Intellectual property

We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property.

## Research ethics

Written consent to publish potentially identifying information, such as details or the case and photographs, was obtained from the patient(s) or their legal guardian(s).

## Authorship

All listed authors meet the ICMJE criteria. We attest that all authors contributed significantly to the creation of this manuscript, each having fulfilled criteria as established by the ICMJE.

We confirm that the manuscript has been read and approved by all named authors.

We confirm that the order of authors listed in the manuscript has been approved by all named authors.

## Patient consent

I qualify as the corresponding author to this manuscript and warrant that I have informed the patient of this scientific manuscript and confirm that I obtained his written and informed consent for the publication of this article.

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