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CEREBRAL VEIN SINUS THROMBOSIS, AN UNUSUAL COMPLICATION OF INFLAMMATORY BOWEL DISEASE

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ABSTRACT

Cerebral venous sinus thrombosis (CVST) is a rare but severe complication of inflammatory bowel disease (IBD). We present 4 cases (3 cases of ulcerative colitis, 1 of Crohn's disease) with CVST over a 10-year period (2014–2024). Symptoms included confusion, headache, seizures, and focal deficits, often initially misdiagnosed. Diagnosis required computed tomography/magnetic resonance venography. Therapeutic anticoagulation and IBD control (steroids, biologics, or surgery) led to favourable outcomes in 3 cases. One patient required thrombectomy. Thrombophilia screening revealed protein C deficiency and *JAK2* mutation in 2 cases. This series highlights CVST as a critical consideration in IBD patients with neurological symptoms. Physicians should pay attention in patients who have pre-existing risk factors and in acute flare of IBD are susceptible to this unusual complication, and advocate for prompt imaging and anticoagulation.

KEYWORDS

Cerebral venous sinus thrombosis (CVST), inflammatory bowel disease (IBD), ulcerative colitis (UC), Crohn's disease (CD)

LEARNING POINTS

- Isolated cerebral venous sinus thrombosis (CVST) without deep venous thrombosis or pulmonary embolism is rare in inflammatory bowel disease (IBD).
- Clinical presentation is often vague, even unenhanced computed tomography scan can miss CVST, acute care physicians should be aware of this complication of IBD.
- Why certain patients develop isolated CVST calls for future research.
- Prompt anticoagulation is the key in management of CVST.

INTRODUCTION

Patients with inflammatory bowel disease (IBD) have an increased tendency to develop venous thromboembolism (VTE), leading to devastating complications and even fatalities[1,2]. Multiple factors contribute to this, including inflammation and its effects on the coagulation cascade[3]. Various clinical factors: age, disease phenotypes, severity, hospitalization, pregnancy, surgical interventions, and medications like corticosteroids, Janus kinase (JAK) Inhibitors are well known factors for VTE[2].

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Cerebral vein sinus thrombosis (CVST) is an uncommon but serious complication of IBD. We present 4 cases with known IBD who presented to our hospital with CVST, over 10-year period, (2014-2024).

CASE DESCRIPTION

Case 1: A 27-year-old woman with a history of ulcerative colitis (UC), who was diagnosed 2 years prior, presented with sudden onset headache, nausea and vomiting to the emergency department (ED). She was on 5-aminosalicylate (5-ASA) and azathioprine for the treatment of colitis. Prior to this index presentation, patient had 2 flare-ups requiring steroids. In ED, her non contrast computed tomography (CT) brain and venogram were normal and subsequent cerebrospinal fluid examination was unremarkable. She was diagnosed as a case of migraine; therefore, reassured and discharged with analgesics.

One week later she re-presented with left side hemiparesis, incoordination and seizures. Bloods showed anaemia with haemoglobin (HB) 87 g/l, albumin 32 g/l and C-reactive protein (CRP) 17 mg/l, CT venogram revealed a right cortical vein thrombus with right parenchymal haemorrhage. She was transferred to the stroke unit in the tertiary hospital and started on anticoagulation with heparin infusion. The gastroenterology team started high dose oral steroids for treatment of colitis. After few days her condition, including the neurological symptoms, improved. She was started on warfarin for anticoagulation and discharged. Unfortunately, due to ongoing active severe disease, she underwent colectomy after failing medical therapy with a tumour necrosis factor (TNF) alpha inhibitor (golimumab). After surgery patient had a stable disease course. She made a full recovery from the CVST. She occasionally has a flare in her rectal stump, for which she receives topical 5-ASA with good effect.

Case 2: A 40-year-old man presented to the ED, with mild headache and sudden onset confusion noted by his girlfriend, who called the ambulance. He was recently diagnosed with Crohn's disease (CD) as he had a history of bloody diarrhoea for the past few months and sought advice from a private clinic 5 weeks prior to admission and underwent colonoscopy which showed colonic CD. He was started on prednisolone 40 mg and mercaptopurine (6-MP).

On examination, he was found to have receptive aphasia. Investigations showed HB 103 g/l, white blood cell (WBC) count 16.5 $\times 10^9$/l, platelets: 515 $\times 10^9$/l, and mildly elevated CRP 12 mg/l.

Brain imaging revealed multifocal areas of acute intra- and extra-axial haemorrhage. CT brain angiography showed haemorrhagic venous infarction due to sinus venous thrombosis of the transverse and sigmoid sinuses on the left, later confirmed by magnetic resonance venography (MRV) *(Fig. 1, for comparison with normal Fig. 2)*[4]. He was started on low molecular weight heparin (LMWH) and levetiracetam. For CD, he was started on intravenous (IV) steroids and anti-TNF therapy (infliximab) as an inpatient. A few days later, upon improvement of his condition, he was discharged with a regular follow up in the IBD clinic. For anti-coagulation, the haematology team put him on oral rivaroxaban. He was found to have protein C deficiency, which may have contributed to his higher thrombotic tendency, in addition to hisCD. He made a full recovery from the CVST and was able to return to work.

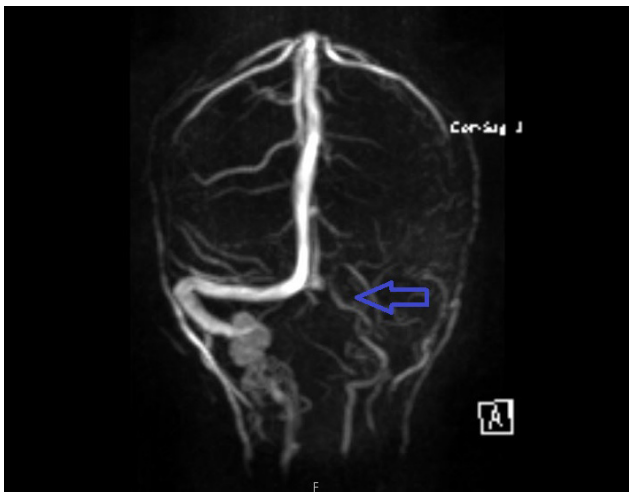


Figure 1. Magnetic resonance venography of the patient showing occluded left transverse and sigmoid sinus thrombosis (blue arrow).

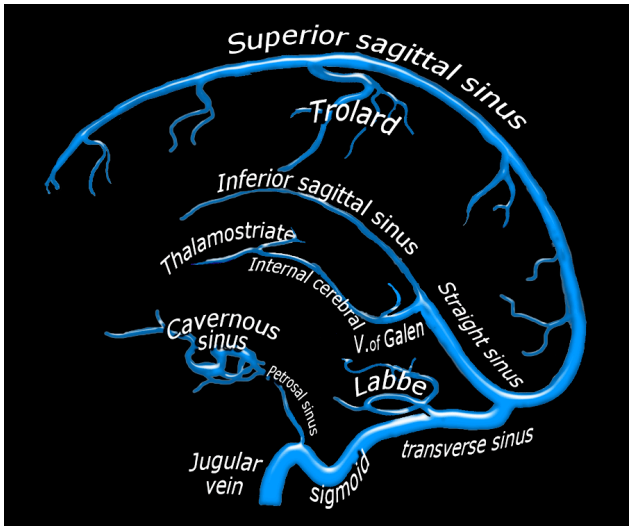


Figure 2. Diagram of cerebral venous sinuses, adopted from the radiologyassisstant.nl[5].

Case 3: A 39-year-old lady presented to the ED in a post ictal state. She had had a witnessed seizure episode at home. When examined in the ED, she was complaining of headache, but no neurological deficits were noted. Her background history was UC and was oral and topical 5-ASA (mesalazine). She had a recent flare of symptoms few months prior requiring an increase in the dose of her medications. Her blood tests were unremarkable apart from leucocytosis (14.9 $\times 10^9$/l), but CT scan of the head showed a right parietal area of low attenuation. CT venogram showed right dural venous sinus thrombosis including the transverse and sigmoid sinuses and extending into the jugular bulb, *(Fig. 3)* and magnetic resonance imaging of the brain revealed a small infarct. She was reviewed by the neurologist and



Figure 3. Computed tomography venogram of the patient showing right transverse and sigmoid venous thrombosis (blue arrow).

commenced anticoagulation with LMWH. She made a quick recovery with no residual neurological deficits and had no further seizures, so she was discharged.

On follow up by the haematologist, the tyrosine Janus kinase (JAK-2) mutation was found to be positive in her case. This mutation may have been an additional risk for CSVT in addition to her UC. She was started on rivaroxaban for anticoagulation. She remained well on follow up examination and her colitis is in remission.

Case 4: The patient was a 43-year-old man with a complex history of UC, of 7 years duration. He initially failed anti-TNF therapy but responded to a α4-β7 integrin inhibitor (vedolizumab). His medical history was remarkable for 'obsessive-compulsive disorder with psychosis' and fatty liver. He was admitted to the hospital with acute fulminant UC. He was started on intravenous steroids. In hospital, his colitis course became complicated with superadded infection with *Clostridium difficile*, requiring antibiotics. Limited bowel examination with flexible sigmoidoscopy revealed severe colitis. Given the ongoing severity of symptoms, the surgical team proposed subtotal colectomy, but the patient declined. Given his mental illness, the treatment plan became very challenging. He was kept under close monitoring and treatment for colitis, upadacitinib (selective JAK-1 inhibitor) was commenced. While he was improving from the colitis point of view, on day 7 he suddenly developed headache and confusion. CT head showed focal hypodense area in the left parietal region with multiple internal haemorrhages. CT venogram revealed superior sagittal sinus thrombosis.

The patient was transferred to the tertiary neurology unit, underwent thrombectomy, and started on anticoagulation. His colitis treatment was changed again to a different TNF alpha inhibitor (adalimumab). He had prolonged rehabilitation and discharged home. Unfortunately, he had further flare up of his colitis and underwent colectomy. He is requiring supportive rehabilitation care.

DISCUSSION

In these 4 cases of IBD presented, there was increased disease activity which appear to be a risk factor for CVST. CVST is a rare and dangerous complication of IBD associated with high mortality and morbidity. The clinical presentation is often non-specific and vague, requiring high clinical suspicion. In addition, non-contrast CT scan can miss these lesions. It is important to note that none of our patients had previous episodes of deep vein thrombosis or pulmonary embolism, which are the most common presentations of VTE.

Aggressive disease control of IBD and anti-coagulation for CVST are the key management strategies. Early recognition and therapeutic intervention ended in a favourable outcome in 3 out of our 4 cases. Patients with pre-existing risk factors for thrombosis, including treatment with small molecules increases the risk for CVST. The true incidence of CVST in IBD has not been ascertained and there is little evidence available in the literature. A retrospective study in China showed cumulative incidence of CVST is nearly 1 out of every 200 cases of IBD[5], which likely to be an underestimation as east Asian populations have lower risk of IBD associated VTE than Western populations[6].

Our case series shows that accurate history, thorough examination and targeted workup are crucial in managing IBD patients with an unusual presentation, and emphasizes that early identification is the key, as prompt anticoagulation is safe and effective, and is associated with favourable outcomes. CVST should be considered in IBD patients presenting with acute neurological symptoms, particularly during disease flares or in the presence of other thrombotic risk factors. Our case series underscores the need for heightened clinical vigilance among emergency and acute care providers and supports further research into the risk stratification and the true incidence of CVST in patients with IBD.

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