

Cortical venous thrombosis in ulcerative colitis : A rare caseAnurag Lavekar¹, Aradya Venu², Suryaprakash Kothe¹, H P Nandeesh³**ABSTRACT**

Extraintestinal manifestations of ulcerative colitis (UC) are common. Cortical venous thrombosis (CVT) is a potentially fatal complication of UC if remained undiagnosed. A high index of suspicion and early recognition of symptoms is required for the diagnosis of CVT in a UC case. CVT is usual after 60 years in UC and active phase of the disease. The absence of guidelines regarding CVT management in UC case adds to the complexity of treatment. We report an unusual case in a 36 years old male patient with partial UC remission presented as CVT which was managed successfully (patient recovered without any neurodeficit) with heparin and warfarin with ongoing treatment for UC.

Keywords : ulcerative colitis, cortical venous thrombosis.

Introduction :

Ulcerative colitis (UC) is a disease complicated by various extra intestinal complications like arthritis, uveitis, primary sclerosing cholangitis, and thromboembolism. Among them, the incidence of cortical venous thrombosis (CVT) as a complication of ulcerative colitis is around 6.5%.¹ The manifestation of cortical venous thrombosis in UC is rare, yet can lead to serious complications. At times, diagnosis of Inflammatory bowel disease (IBD) is established only after the CVT episode.^{2,3} Patients with active IBD are at an increased risk of thromboembolism with primary CVT being rarer in young adults.³ This case report highlights the fact that cerebral venous thrombosis has to be suspected in patients with ulcerative colitis who present with acute onset neurological deficit.

Case Report :

A 36-year-old male patient who was a known case of UC presented to the casualty with sudden onset headache followed with generalised tonic-clonic seizures and vomiting the previous day. He had no bowel complaints and had a regular frequency of stools with no blood in stools. He was regularly

taking the medications mesalamine (2.4 mg / day) and Azathioprine (50 mg / day) for last two years for UC, and had achieved remission of his symptoms on above medications. On examination, he was conscious and had faciobrachial monoparesis on left side.

MR venography examination revealed loss of flow void in the superior sagittal sinus, cortical veins, left transverse sinus and sigmoid sinus suggestive of thrombosis.

His complete blood picture was normal. His liver function & renal function tests, serum electrolytes, fasting blood sugar and coagulation profile were normal. His antithrombin III was normal with 118% (normal range 83-128%). However, his Protein C was 56% (normal range 70-140%) which was less. Protein S was also found to be less 58% (74.1-123.7%). He had recently undergone colonoscopic biopsy (one week before presentation) which showed partial histological remission. He was continued with the ongoing treatment for UC and was started on low molecular weight heparin later changed to warfarin. Once on warfarin, during his hospital stay, regular monitoring of the prothrombin time was done. He showed a rapid neurological improvement in power within seven days. He did not have any bleeding complications. In follow-up one month later, he recovered with no residual symptoms.

Discussion :

The precise mechanisms which lead to a thrombotic state in ulcerative colitis are poorly understood.⁴ It

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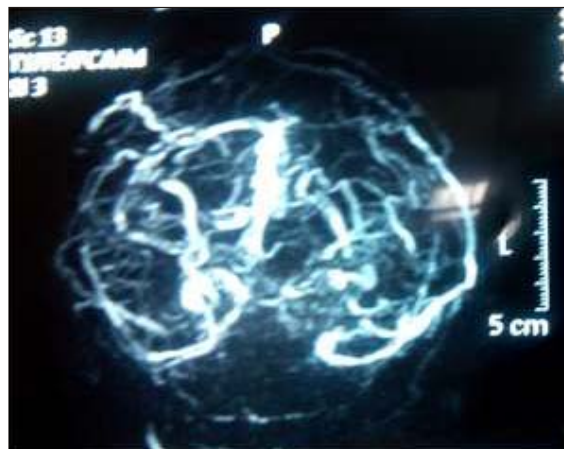
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can be postulated that hypercoagulable state, hyperfibrinolysis and immunological abnormalities (antiphospholipid antibodies) play a role in thrombosis.⁵ However, there is no sufficient evidence to enforce this assumption. Hyperhomocysteinemia secondary to altered folate metabolism can also be one of the possible explanations for thrombosis in ulcerative colitis. One of the causes for decrease in protein C and S in this patient may be attributed to the activation of complement system due to UC leading to increased binding of these proteins.



Hemorrhagic infarct in the frontoparietal region

Prognosis of CVT in ulcerative colitis revolves around early recognition of neurological dysfunction and its treatment; as seen in our case. There is a paucity of data or guidelines regarding the approach to the treatment of cortical vein thrombosis in ulcerative colitis. Therefore, guidelines used for patients of CVT otherwise; are also used while treating CVT in ulcerative colitis patients.^{6,7} Low molecular weight heparin is usually the first line of therapy for treating CVT which was similarly followed in our case.⁸ Unfractionated heparin is best avoided for fear of GI bleeding; particularly in ulcerative colitis patient. Trial with endovascular thrombolysis has been reported to have favourable outcome and safety profile. ACG guidelines advocate the use of thromboprophylaxis in active disease.⁹



Absent blood flow in superior sagittal sinus, transverse sinus, sigmoid sinus and jugular vein

Thrombosis is usually encountered after the age of 60 years.¹⁰ Our patient presented at a relatively younger age. CVT is commonly associated with active stage of the Ulcerative colitis, whereas our patient presented when he was in partial remission. The use of anticoagulant medications against thrombosis primarily related to UC is debatable due to risk of hemorrhage from the gastrointestinal tract. Fortunately, our patient who had extensive involvement of almost all cortical venous sinuses responded well to low molecular weight heparin therapy with no residual neurological deficits or GI Bleed.

Conclusion :

Our case underlines the importance of early recognition of CVT in ulcerative colitis as it can manifest even in the inactive stage of ulcerative colitis. It also emphasises the role of anticoagulation in the treatment of CVT in known Ulcerative colitis patients.

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