Cerebral Venous Sinus Thrombosis in a Patient with Ulcerative Colitis

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ABSTRACT

Introduction: Cerebral venous sinus thrombosis is a condition in which cerebral venous drainage is interrupted by thrombus formation within dural venous sinuses, which may be further complicated by increased intracranial pressure, cerebral edema and occurrence of cerebral venous infarction or hemorrhage. It is uncommonly seen in the patients of ulcerative colitis.

Case report: We report a case of a 33 years old male with previous history of ulcerative colitis who was diagnosed with cerebral venous sinus thrombosis. He was presented with acute right side hemiparesis preceded by headache for one week. CT brain showed hyperdense and thickened mid part of the superior sagittal sinus. Urgent Brain MRI with contrast and Magnetic Resonance Venography MRV studies confirmed loss of signal void and no contrast opacification in the vast majority portion of the superior sagittal sinus particularly the anterior and mid segments with minimal residual patent portion posteriorly.

Conclusion: He was treated with therapeutic dose of Low Molecular Weight Heparin, intravenous fluids in addition to his previous medications of Ulcerative Colitis (Prednisolone and Mesalazine). On fourth day patient improved and he was discharged on long-term warfarin therapy.

Keywords: Cerebral Venous Sinus Thrombosis, Magnetic Resonance Venography, Ulcerative Colitis

INTRODUCTION

Cerebral venous sinus thrombosis is a condition in which cerebral venous drainage is interrupted by thrombus formation within dural venous sinuses, which may be further complicated by increased intracranial pressure, cerebral edema and occurrence of cerebral venous infarction or hemorrhage. It is a rare cerebrovascular disease that accounts for 0.5% of strokes. It usually manifests as headache, seizures, focal neurological deficits, and altered mental state. Due to its low incidence and various manifestations, it is not easy to suspect CVT, which causes delays in treatment and little impact on the results.[1]

Ulcerative colitis causes inflammation and ulcers (sores) in the digestive system. The innermost lining of your large intestine (colon) and rectum are affected by ulcerative colitis. Symptoms of ulcerative colitis appear gradually.[1] Cerebral venous sinus thrombosis (CVT) is a rare but potentially fatal complication of ulcerative colitis (UC), with an annual incidence ranging from 0.5% to 6.7%. It's thought to be a result of the hypercoagulable condition that occurs after a disease recurrence.[2]

CASE REPORT

A 33-year-old male patient, with past history of migraine and ulcerative colitis which diagnosed 8 years previously, presented to emergency department with acute right side weakness and right side facial weakness started two hours before arrival. He admits increasing frontal headache for the last week after doing upper and lower GI endoscopy. On assessment he looks underweight, his vitals were normal, fully conscious, oriented (GCS 15), normal speech, he had right upper motor neuron facial weakness, other cranial nerves were intact, right hemiparesis with power grade 3/5 in upper limb and 4/5 in lower limb on MRC scale. Sensory examination, coordination and bedside confrontation tests were normal. His NIHSS was 4 on initial assessment. Stroke code announced and patient directed to CT room.

Investigation

CT brain showed hyperdense and thickened mid part of the superior sagittal sinus (Figure 1). Urgent Brain MRI with contrast and MRV studies confirmed loss of signal void and no contrast opacification in the vast majority portion of the superior sagittal sinus particularly the anterior and mid segments with minimal residual patent portion posteriorly (Figure 2). Few tiny suspicious foci in the left high frontal para sagittal region showing faintly restricted signal on DWI and corresponding low signal on ADC raise the likelihood of potentially early small venous infarctions.[3]

Treatment

He was admitted to ICU, started on therapeutic dose of Low Molecular Weight Heparin, intravenous fluids and his previous medications of UC (Prednisolone and Mesalazine) continued until reviewed by gastroenterologist who decided to continue the same. Nine hours after admission, he developed sudden decrease in conscious level, so intensivists decided to intubate him and to be connected to mechanical ventilator. Brain CT repeated showed no interval changes compared to initial study. Fundus examination showed no papilledema. Next day of admission, patient regained full consciousness, he was kept on mechanical ventilation minimal settings then sedation gradually stopped and was extubated on 3rd day of admission. Physical and occupational therapy started early

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How to cite this article: Akkad A, Gamea M, Ibrahim R. Cerebral venous sinus thrombosis in a patient with ulcerative colitis. International Journal of Contemporary Medical Research 2021;8(9):I5-I7.

DOI: http://dx.doi.org/10.21276/ijcmr.2021.8.9.10

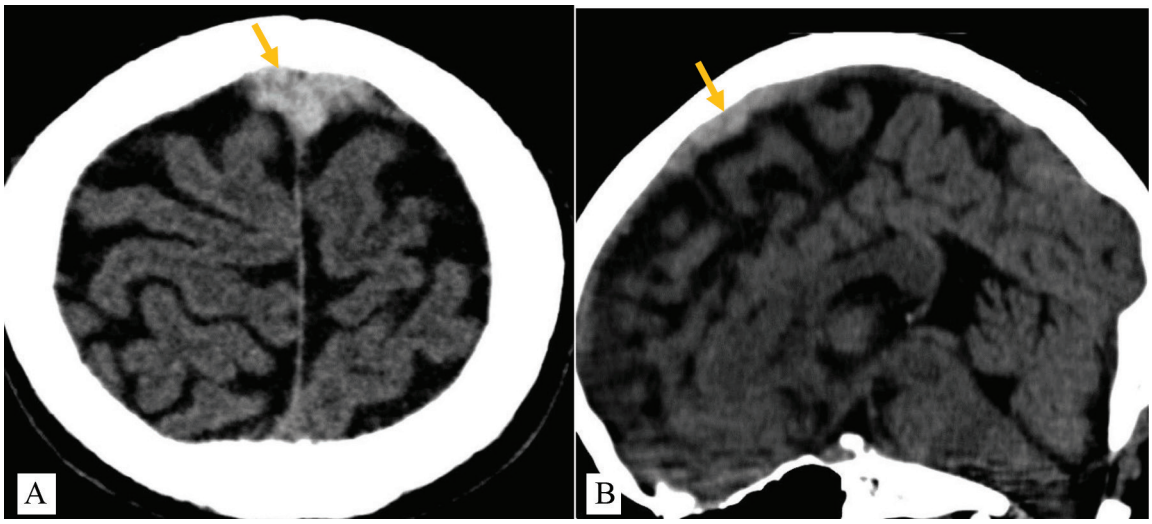

Figure-1: Brain CT scan shows hyperdense and thickened mid part of superior sagittal sinus (arrows) in axial cut (A) and sagittal cut (B).

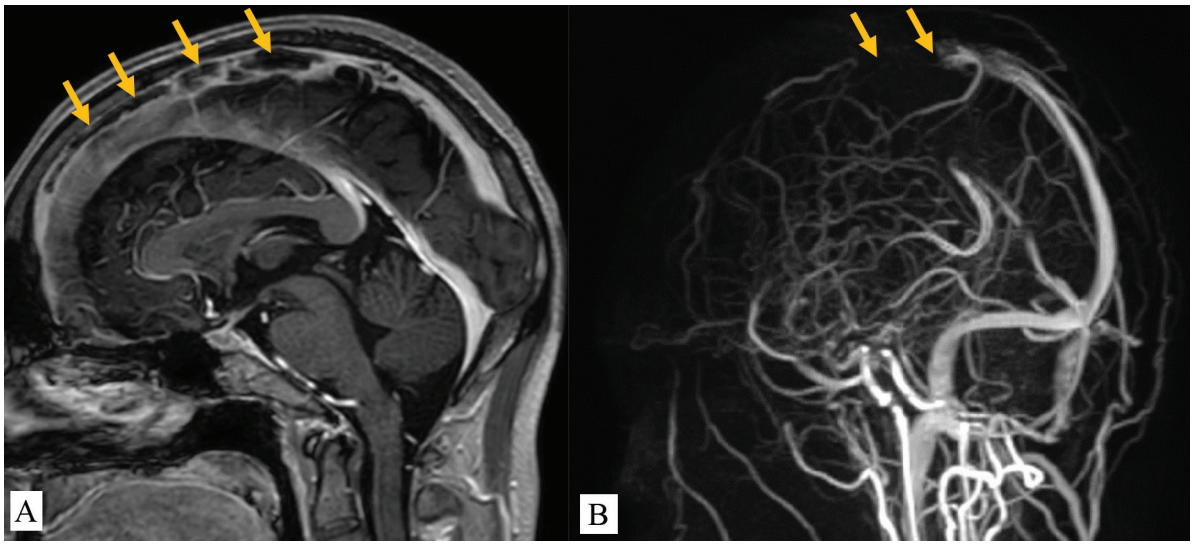

Figure-2: (A) Brain MRI -T1WI with contrast showed loss of contrast opacification (arrows) due to thrombosis of anterior and mid segment of superior sagittal sinus. (B) MRV study shows loss of signal void (arrows) in anterior and mid part of superior sagittal sinus.

after admission and surprisingly the right facial weakness and right side weakness improved to full power on 4th day. Warfarin started under cover of LMWH until therapeutic INR (above 2) was reached, then LMWH was stopped and the patient was discharged in a stable condition on long term warfarin therapy. [4]

DISCUSSION

CVST is a relatively rare cause of stroke that affects young individuals (under the age of 50).[7] CVST is also a rare extraintestinal manifestation of IBD. The mean age of IBD patients at the time of the CVST is significantly younger when compared with CVST patients without IBD; in the reviewed case reports, the mean age of IBD patients at the time of the CVST was <29 years. Additionally, anemia and thrombocytosis have been suggested as significant risk factors for CVST. Coagulation abnormalities (increased fibrinogen, elevated factor VII, elevated factor VIII, antithrombin III deficiency, and protein C and protein S deficiency) often coexist with IBD and return to normal values when the intestinal inflammation is controlled. In this study our patient is 33 years male suffering from CVST.[5]

The variable presentation of CVST and its insidious course often contribute to delays in diagnosis. Headache is the most common and sometimes only presenting symptom, which makes it a clinical challenge to diagnosis CVST. Further increased intracranial hypertension may cause vomiting, diplopia, visual impairment, and decreased level of consciousness. Focal neurologic symptoms may suggest the presence of a venous infarction and hemorrhagic conversion, which has been reported in more than one-third of patients. In this case, the patient initially complained of a headache only, which progressed to a decreased level of consciousness and focal neurologic deficit. Patient of this study had right side facial weakness and frontal headache. He had right upper motor neuron facial weakness, other cranial nerves were intact, right hemiparesis with power grade 3/5 in upper limb and 4/5 in lower limb. In conclusion, the presence of a severe headache or neurologic signs or symptoms in a patient with IBD should prompt clinicians to suspect the possibility

of CVST, which may be the presenting symptom for a newly diagnosed active phase of IBD.[6]

Thus, the patient is admitted to CT room. Non-contrast head CT, which is the most frequently performed imaging study for the evaluation of patients with neurological deficit, has poor sensitivity to detect CVT.MRI/magnetic resonance venogram is recommended as a preferred brain image to diagnose CVT. The most commonly affected venous sinus is the superior sagittal (62%) as in our case; however, multiple cerebral thrombosis has been reported in half of the patients.[7] Anticoagulation treatment is the standard treatment for patients with CVT. Anticoagulation prevents further thrombus growth and facilitates recanalization. Previous studies revealed anticoagulation treatment was associated with a lower mortality in patients with CVT. Therefore, the current guidelines suggest that patients with CVT should be treated with low molecular weight heparin or intravenous heparin, even in the presence of intracranial hemorrhage. Previous studies have shown that IBD patients with CVT who did not receive anticoagulation treatment showed a poorer outcome compared to those who received it. In our case, the patient was treated with prednisolone, 5-aminosalicylic acid, and anticoagulation was initiated with intravenous LMW heparin and continued with warfarin. Patients with multiple cerebral thrombosis and hemorrhagic infarction in the basal ganglia have been reported to have a higher risk of mortality. In this case, CVT may not only be associated with UC but possibly also with antiphospholipid syndrome.[8] Our patient had thrombosis limited to superior sagittal sinus with small subcortical venous infarcts that were not hemorrhagic, he was recovered without residual neurologic deficit.

Corticosteroid treatment in IBD patients with CVT is still up for debate. Although some previous studies considered that steroid treatment does not increase the risk for VTE, but rather promote anti-inflammatory effects and decrease hypercoagulability in IBD patients, other studies reported high dose steroid treatment is associated with an increased risk of developing VTE, especially in patients with other risk factors (e.g., inherited coagulopathies, oral contraceptive use). Our patient was treated without corticosteroids out of concern for a possible adverse effect of corticosteroids.[9]

CONCLUSION

Cerebral venous sinus thrombosis is an uncommon form of stroke that could be life threatening or leads to severe disability. Its prognosis is directly related to early diagnosis and appropriate treatment. CVST is considered an uncommon extraintestinal complication of UC that usually occurs during active phase. It should be suspected in any patient with a known risk factor that may cause a hypercoagulable state e.g. chronic autoimmune disease, pregnancy, post-partum state, vasculitis, inflammatory bowel disease or cancer presenting with focal or diffuse neurologic symptoms or signs suggest increased intracranial pressure like headache, vomiting, visual disturbances or limb weakness. Our aim is to raise the awareness among clinicians about this uncommon complication of UC as early diagnosis and treatment commonly leads to a favourable outcome.

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Source of Support: Nil; **Conflict of Interest:** None

Submitted: 08-08-2021; **Accepted:** 30-08-2021; **Published:** 30-09-2021