Case of Cerebral Venous Sinus Thrombosis and Hemorrhagic Infarct in a Patient with Ulcerative Colitis

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Introduction

Ulcerative colitis (UC) is an inflammatory bowel disease (IBD) characterized by inflammation of the colonic mucosa. While patients with IBD have a three-fold higher risk of venous thromboembolism (VTE), cerebrovascular involvement is rare. We present a case of a pediatric patient whose ulcerative colitis flare was complicated by a cerebral venous sinus thrombosis (CVST) and a hemorrhagic infarct.

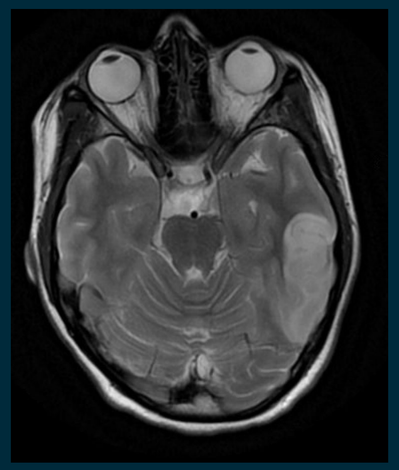
Patient Presentation

This is a 15 year old female with mild developmental delay who was diagnosed with ulcerative colitis five months prior to presentation. Patient was admitted to our pediatric hospital for management of a UC flare after an outpatient colonoscopy revealed pancolitis. At home, her management consisted of infliximab which she tolerated well until the past several weeks when her symptoms worsened. Stool cultures were negative, along with a negative *Clostridium difficile* PCR. She admitted to initiate intravenous steroids and oral tacrolimus.

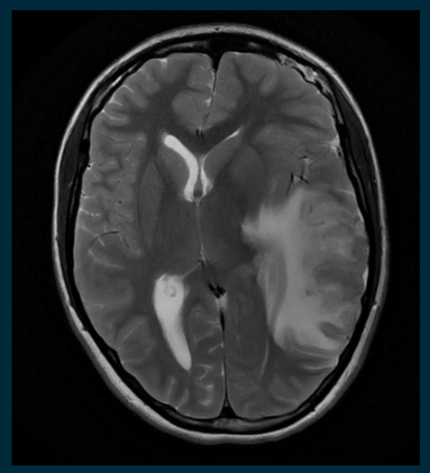
Clinical Course

- Three days after admission, the patient started complaining of a headache and had a witnessed tonic clonic seizure.
- Stat head CT showed a hyperattenuation in the area of the left transverse and sigmoid sinuses in the left posterior cranial fossa, and follow-up MRI/MRA/MRV were suggestive of CVST and hemorrhagic infarct.
- Routine repeat imaging was performed four days later and showed increase in the size of a hemorrhagic infarct with new uncal herniation and a left-to-right midline shift.
- The patient underwent emergent left decompressive hemicraniectomy with improvement on post-operative imaging.
- The patient recovered to her neurologic baseline within a week and started to undergo physical, occupational, and speech therapy.
- She was placed on enoxaparin with consultation of hematology.
- The patient has since undergone total colectomy as treatment for her UC.

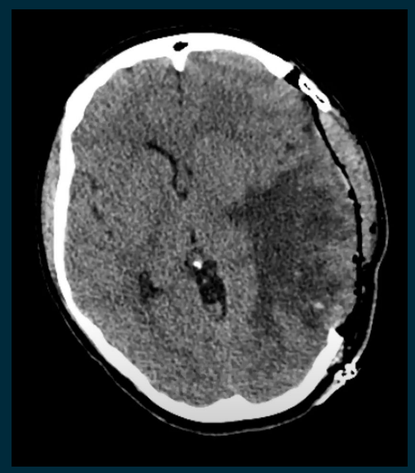
CT Head: Hyperattenuation concerning for for possible deep venous sinus thrombosis



MRI Brain T2: Deep venous sinus thrombosis in transverse/sigmoid sinuses and hemorrhagic infarct within the left temporoparietal lobe.



MRI Brain T2: Interval increase in the size of a hemorrhagic venous infarct centered in the left temporoparietal lobe with new uncal herniation and 9 mm left-to-right midline shift



Head CT: Post craniectomy with near resolution of uncal herniation and midline shift.

Additional Workup

Hypercoagulability Workup

- Overall, the patient's hypercoagulability workup was grossly unremarkable.
- Lupus Anticoagulant: negative, ANA: negative, Complement C3: 138, C4: 21.9, Rheumatoid factor: <10, Antithrombin III activity: 126
- Protein C activity: 114(↑), Protein S activity: 68
- Thrombin time: 14.7(↑)
- Factor 5 assay: 112, Factor VIII activity: 256 (↑)
- Homocysteine: 4.9, Vitamin B6: 19.5 (↓)

Discussion

- IBD is thought to be associated with a hypercoagulable state leading to thromboembolic complications. Oftentimes, coagulation studies will show clotting abnormalities as well as abnormal values of fibrinolysis, nutrition factors, platelets, and endothelial abnormalities.
- CVST is an uncommon but devastating thromboembolic complication of UC seen in approximately 1.3%-7.5% of cases yearly. The use of prophylactic anticoagulation is controversial. Some guidelines recommend low molecular weight heparin for prevention of venous thrombosis despite the presence of a hemorrhage infarct, while others suggest avoiding thromboprophylaxis when severe bleeding is present. In our case, it was felt that the risk of further bleeding outweighed the potential benefits of initiating anticoagulation.
- Supratherapeutic tacrolimus has also been shown to exhibit neurological side effects including leukoencephalopathy, posterior reversible encephalopathy syndrome and reversible cerebral vasoconstriction syndrome. However, in our case, the patient's tacrolimus levels were low during seizure onset and the present imaging results made tacrolimus induced neurotoxicity less likely.

References

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