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Ulcerative Colitis Presenting with Bilateral Thalamic Infarcts Due to Cerebral Sinus Thrombosis

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Keywords

Ulcerative colitis; cerebral sinus thrombosis; stroke; hypercoagulable; MRV

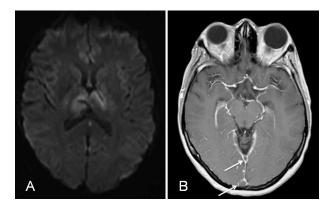


Figure 1. (A) Sagittal MR venogram showing absence of flow through the superior sagittal sinus (white arrow) and confluence of sinuses (black arrow). (B) Coronal imaging demonstrates absence of flow in superior sagittal sinus (black arrow) and transverse sinus (white arrow).

Case

A 35-year-old woman with ulcerative colitis was transferred to our hospital because of 2-day history of altered mental status and headache. Apparently, 2 weeks prior to the transfer she had been having increased nausea, vomiting, and diarrhea. She was diagnosed and reported with extensive thrombosis of the intracranial venous system. There was thrombosis of internal cerebral veins, the superior sagittal sinus, straight sinus, and transverse and sigmoid sinuses with extension into jugular veins. There was also acute ischemia of thalami bilaterally. Homocysteine and protein C levels were normal. Protein S and thrombin levels were slightly low. Subsequently,

Figure 2. (A) MR diffusion weighed imaging showing bilateral thalamic infarcts. (B) T1 MRI with gadolinium demonstrates empty delta sign in the superior and inferior sagittal sinuses (white arrows).

anticoagulation was initiated with continuous intravenous infusion of heparin.

Comment

Ulcerative colitis (UC) is an idiopathic chronic inflammatory bowel disease usually affecting patients between the ages of 20 and 30 years old with a second peak age between the ages of 70 and 80 years old.¹ UC's CNS manifestations include thrombotic and cerebrovascular disease, myelopathy, cerebral vasculitis, multiple sclerosis, and acute disseminated encephalomyelitis. Patients suffering from UC are at risk of developing a hypercoagulable state and the two most common thrombotic complications are deep venous thrombosis and pulmonary thromboembolism.² The frequency of cerebral vein and sinus thrombosis (CVST) ranges from 1.3% to 7.5% of cases yearly.³ The etiology of hypercoagulation and

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thromboembolism in IBD remains to be elucidated. Factor abnormalities such as elevated levels of fibrinogen, factor V, factor VII, etc., increase in circulating thrombin–antithrombin complexes, and decreased antithrombin III have been described.⁴ In conclusion, cerebral sinus and vein thrombosis is a serious and often fatal complication of idiopathic inflammatory bowel disease if undiagnosed. It should be considered in 51 any patient with little or unknown vascular risk factors presenting with a severe headache and other focal or diffuse neurological signs.

References

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