

Agitation as the only symptom of cerebral venous sinus thrombosis in a patient with Crohn's disease

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Neurologic manifestations in inflammatory bowel disease (IBD) are not precisely estimated. Cerebral venous sinus thrombosis (CVT) is rare and only 1.6% of total CVTs are associated with IBD [1-5]. CVT occurs in only 1.3-6.4% of adults and 3.3% of children with IBD [2-5].

A 44-year-old female presented to the emergency department with fever of up to 38°C, abdominal pain and hemorrhagic diarrhea. Initially she was diagnosed as having ulcerative colitis, but 5 years ago the diagnosis was changed to Crohn's disease (CD) and was treated with adalimumab.

Physical, neurological and fundoscopic examinations were unremarkable apart from a temperature of 38°C. She had clotted external grade III hemorrhoids and perianal abscess.

Hematology examination revealed hematocrit of 27%, hemoglobin of 8.5 mg/dL and platelet count of 506,000 /mm³. Biochemical investigation was normal. The antithrombin level and the activities of the proteins C and S were normal. At that time the event was considered as a relapse of CD. The patient underwent surgical procedure for the abscess, and was treated with IV meropenem, and methylprednisolone. A prophylactic dose of enoxaparine was introduced.

On the 3rd day of hospitalization she became agitated, with anxiety attacks, ideas of impending doom, suspiciousness and insomnia. The symptoms were compatible with acute paranoid reaction or organic psychosyndrome. The brain MRI showed a subacute thrombus in the superior sagittal sinus. MR venography confirmed the absence of venous flow (Fig. 1).

The patient was treated with a therapeutic dose of enoxaparine for six months. Follow-up MR brain venography and neurological examination at three months after hospital discharge were normal.

There are limited reports in the literature concerning CVT associated with CD [3-5]. In our patient mainly the elevated platelets were indicative of increased activity of the coagulation system but she was receiving a prophylactic dose of enoxaparine. Mental disorders are a common side effect of corticosteroids [6]. These events were against the diagnosis of CVT.

Factors that promote thrombosis in IBD are: generalized inflammatory activity, intestinal loss of circulating anticoagu-

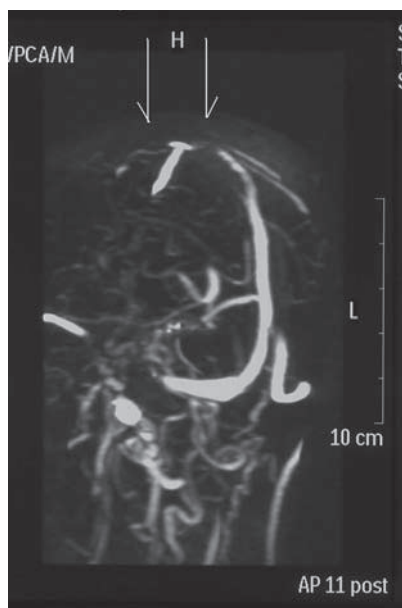


Figure 1 MR venography showing absence of flow on superior sagittal sinus

lants and transient abnormalities of the coagulation system during the active course of the disease such as: thrombocytosis, increased levels of factor V, VIII and fibrinogen and decreased antithrombin III and protein S [3-5]. High-dose corticosteroid treatment is associated with an increased risk of CVT [7]. Notably, there is one case reported with ulcerative colitis at anti-tumor necrosis factor (TNF) treatment which developed CVT [5]. Of all these mentioned factors our patient had thrombocytosis, she was receiving corticosteroids and anti-TNF.

The usual neurological symptom is persistent, global headache. The increased intracranial pressure results in vomiting and papilledema. The most common focal symptoms are: seizures, hemiplegia, tetraplegia, vision deficits, altered consciousness, aphasic disorders and confusion [2-5]. Rarely, CVT could present with psychiatric abnormalities if thalami or cortex are affected [2,3,5]. To our knowledge, pure psychiatric manifestations without a structural lesion have not been reported.

The current therapeutic approach recommends initial treatment with heparin which should be switched to oral anticoagulation for 3 to 6 months if a risk factor is not identified or is transient, otherwise treatment should be longer, even indefinitely [2-5,8].

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Conflict of Interest: None

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