Case Report

Cerebral Venous Thrombosis and Crohn's Disease: A Case Report and Literature Review

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Abstract

Cerebral venous thrombosis (CVT) can increase cerebral venous pressure leading to decrease cerebral perfusion; resulting in ischemic injury, parenchymal hemorrhage and vasogenic edema. The clinical manifestations of CVT are: headache (more common), vomiting, seizure, vertigo, ataxia and photophobia. In this paper we report a 19-year-old girl with Crohn's disease. Our patient is a known case of Crohn's disease for about 14 years and is suffered from hyper coagulopathy state.

Keywords: Cerebral Venous Thrombosis; Crohn's Disease; case report

Introduction:

Inflammatory bowel disease (IBD), is an autoimmune disease with unknown etiology that is caused by inappropriate immune response to intestinal microflora. IBD is divided into two major types: ulcerative colitis (UC - that just affects colon) and Crohn's disease (that can involve every part of gastrointestinal tract, from mouth to anusm with transmural and skip lesions pattern). IBD can also be associated with extra-intestinal manifestations such as musculoskeletal, hepatobiliary, skin, pulmonary, ophthalmology and coagulation cascades problems (1-6). Patients with IBD are in higher risk of venous thrombosis compared to general population (2). The reason could be the hypercoagulable states with an unknown reason; however, in this situation, increased fibrinogen, factors V and VIII is seen along with thrombocytosis, decreased antithrombin, factor III and protein S (4). Overall 1.3%-6.4% of IBDs, experience cerebral venous thrombosis (CVT) in their life time (5-6) and this percentage increases to about 3.4% in children (5). The incidence of CVT in UC is more than Crohn's (2). CVT is more common in young patients, however coincident of IBD and CVT occurs in younger ages more than CVT alone (5).

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Case Presentation:

A 19 years old female was admitted with chief complaints of abdominal pain and diarrhea since 4 days prior to admission. She had no history of fever or night sweating. Another symptom was headache; which was progressive till 2 days ago, without proper control by analgesics and coexistent with paresthesia, limb weakness and blurred vision. There were no photophobia, vertigo or ataxia. The patient suffered from Crohn's disease since 14 years ago and was on medication (Asacol, Ferfolic and Prednisolone). The patient was admitted in GI service. On physical exam the patient was moderately pale and drowsy with vital signs: Blood pressure: 100/60, Pulse rate: 75, Respiratory rate: 18, Temperature: 37.1 Centigrade, O2 sat: 95%. She had pale skin and pale conjunctiva. The chest was clear with normal breathing sounds and bilaterally symmetrical expansion. Her abdomen was soft, flat, with normal bowel sounds without any tenderness, rebound tenderness or guarding. Extremities was ok with no pitting edema. The neurologic examination revealed 3mm pupillary size with no abnormalities, and reactive to light. No extra ocular abnormality was detected. Motor system examinations showed normal tone, with brisk reflexes. Sensory examination was unremarkable. Plantar reflexes were bilaterally flexor. Deep tendon reflex was 1/2 and all muscle power were normal (5/5). Her ECG was normal sinus rhythm and normal axis.

Lab findings showed on table 1. Because of patient's condition and her headache, Brain CT scan was done and neurologic consultation was requested. Brain CT scan's report: Hyper density in superior sagittal sinus and left transverse sinus is noted suggestive of cerebral vascular thrombosis (CVT). We confirmed diagnosis via Brain MRI & MRV: complete obstruction of superior sagittal, left transverse and sigmoid sinus by thrombosis and partial obstruction in right transverse and sigmoid sinus is noted. The additional lab tests requested for her such as: Lupus anticoagulant (LAC): >120 (NL: 28-45), ANA, Anti Ds-DNA, C3, C4, APLA, C-ANCA, P-ANCA, B2 Glycoprotein all were within normal limits.

On admission; anticoagulant (Heparin – Caspian Tiamin Rasht) was administered intravenously to treat CVT. For prevention of convulsion, levetiracetam (Stragen Daroo) was started. After 10 days, patient developed fresh rectal bleeding which resulted in decreased hemoglobin and hypovolemic shock, so the decision was to stop heparin, start hydration and a brief period of norepinephrine and transfuse blood products. Emergency GI consult, endoscopy and colonoscopy were done. Colonoscopy and endoscopy reported moderate erythema, diffuse and multiple polypoid lesions (Pseudopolyps), loss of vascular marking, diffuse granularity from rectum to cecum. Few small ulcers were also seen in ileum up to 20cm.

After acceptance of GI man, low dose heparin injection was started again, however after 2 days, platelets dropped and it seems that heparin induced thrombocytopenia (HIT) was occurred. So heparin injection

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was stopped again and the patient was discharged.

Discussion:

The risk factors for CVT are genetic and acquired thrombophilia, OCP consumption, pregnancy and hypercoagulable state. The problem of hypercoagulable state in IBD is not completely solved as the cause seems to be the abnormality in coagulative factors such as increased fibrinogen and leiden factor V, decreased antithrombin 3 which is involved in this process (3,6). Coagulation activity can be related to the activity and colonic extension of the disease that develops during days (3). Seizure may occur in 40% of cases. Other neurological presentation such as unilateral focal deficit, sign and symptoms of increase ICP is also found in patients with IBD (3). In one of the literatures, the prevalence of neurological manifestations is including: Headache (80%), seizure (35%), vomiting (29%), hemiplegia and quadriplegia (29%), altered consciousness (21%), visual deficit (12%), and pupil edema (7%). The mean age of CVT prevalence is 29 years old and females are suffered more than males (37% vs 28%) (2). CT scan can exclude the structural abnormalities but due to poor sensitivity, we should use CT-Venography and MRI or MRV as the main diagnostic methods (4). The most common site of CVT is superior sagittal sinus (50%). Treatment of venous thromboembolism (VTE) in patients with IBD is the same as patients without VTE (1).

The most common strategy which improves the outcome of CVT is the administration of anticoagulant with intravenous heparin or subcutaneous LMWH. If the patient is stable, then the treatment can be continued by oral anticoagulants for about 3-6 months. If there are contraindications for this method, endovascular thrombolytic therapy, can be used (1). Our patient is a known case of Crohn's disease for about 14 years and is suffered from hyper coagulopathy state.

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Table 1: Laboratory findings of the patient

Parameter	Value	Parameter	Value
WBC	5.7×10^9/L	Na	139 mEq/L
Platelet	140×10^9/L	K	3.7 mEq/L
Hemoglobin	7.9 g/L	AST	15 U/l
PT	16 second	ALT	9.0 U/l
PTT	29.9 second	Alkaline phosphatase	142 IU/l
INR	1.55	Total bilirubin	0.3 mg/dl
BUN	7.0 mg/dL	Total protein	4.0 g/dL
Creatinine	0.4 mg/dL	Albumin	2.3 g/dL

AST: aspartate transaminase; ALT: alanine transaminase; PTT: Partial thromboplastin time; PT: prothrombin time; INR: International Normalized Ratio; BUN: blood urea nitrogen;

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