Arterial and venous thrombosis with right ventricular thrombus in Crohn's disease

Sujay Halkur Shankar[1], Prabhat Kumar[1], Kumble S. Madhusudan[2]

[1]Department of Medicine, All India Institute of Medical Sciences, New Delhi, India
[2]Department of Radio-diagnosis, All India Institute of Medical Sciences, New Delhi, India

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Dear Editor,

Inflammatory bowel disease (IBD) is a known hypercoagulable state, and thrombosis could be the initial manifestation in patients with this disease. Arterial thrombosis is rare in these patients compared with venous thrombosis, and ventricular thrombus formation is even rarer. Here we describe a rare case of Crohn's disease with cerebral infarction, proximal venous thrombosis, and right ventricular thrombus.

A 24-year-old man presented with complaints of right lower limb swelling since 15 days prior. He had a history of intermittent, colicky lower abdominal pain, which was associated with an increased frequency of stools since 1 month prior. He also had a significant, documented weight loss over the previous 4 months. Five months back, he had acute-onset headache associated with dysarthria, forgetfulness, and inability to recognize faces. A magnetic resonance (MR) imaging of the brain at that time showed bilateral middle cerebral artery infarction. MR angiography revealed significant attenuation of the M2, M3, and posterior frontoparietal cortical branches of the left middle cerebral artery. He was extensively worked up for hypercoagulable state which turned up negative. His symptoms improved over time with minimal residual defects during the present admission. On examination, he had a swollen and tender right lower limb with local temperature increase. Blood investigation results revealed iron deficiency anemia and increase in acute phase reactants. A bilateral lower limb Doppler was performed, which revealed acute deep venous thrombosis of the bilateral common femoral veins, superficial femoral veins, and popliteal veins, extending into the bilateral external iliac veins, common iliac veins, and inferior vena cava. A transthoracic echocardiogram revealed a partially mobile right ventricular apical thrombus measuring 28×16 mm in size, with normal left ventricular function. A subsequent computed tomography (CT) pulmonary angiography revealed normal pulmonary arteries with right ventricular

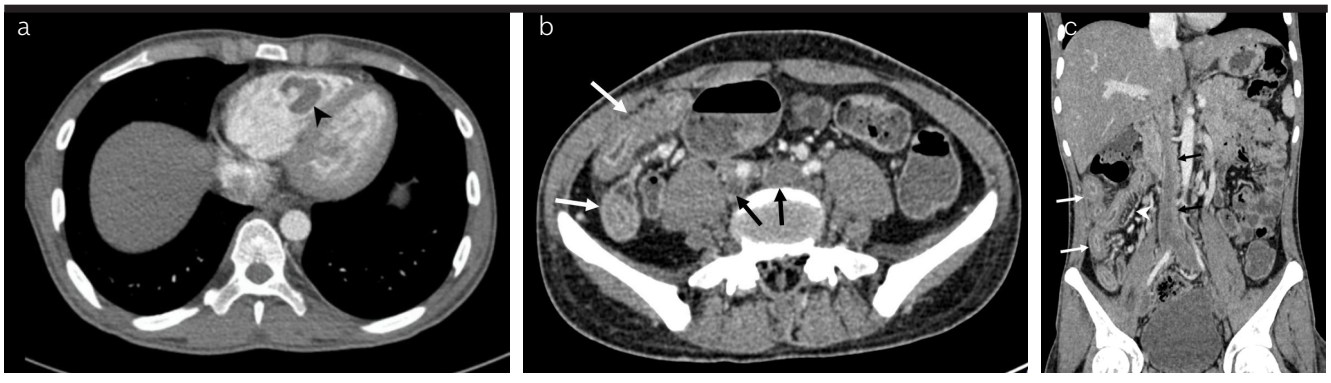

Figure 1. a-c. CT pulmonary angiography showing thrombus in right ventricle (arrow) (a); Axial contrast enhanced CT scan shows long segment thickening of an ileal loop (white arrows) with stratified enhancement and thrombosis of bilateral common iliac veins (black arrows) (b); Coronal contrast enhanced CT scan shows the thickened ileal loop (white arrow) with prominent mesenteric vascularity (arrow head) and thrombosis of the inferior vena cava (black arrows) (c).

Corresponding Author: ***Prabhat Kumar; drkumar.prabhat@gmail.com***
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thrombus [Figure 1A]. Repeat MR imaging of the brain during the present admission was consistent with an old vascular infarct with a normal MR angiogram. A work-up for congenital and acquired hypercoagulable state was negative. Furthermore, serum antinuclear antibody (ANA), anti-neutrophil cytoplasmic antibody (ANCA), and HLA B51 tests were negative, thereby ruling out a possibility of any underlying vasculitis.

CT enterography features were consistent with the diagnosis of Crohn's disease [Figure 1B, 1C]. Colonoscopy was suggestive of terminal ileitis with multiple aphthous ulcers in the terminal ileum. Histopathology of the ileal biopsy revealed chronic inflammation with no granulomas. The Crohn's disease activity index was calculated to be 290, providing evidence of active disease. Following diagnosis, the patient received an induction therapy of infliximab at a 5mg/kg dose at 0, 2, and 6 weeks with low-molecular-weight heparin. His symptoms resolved on treatment, and his current Crohn's disease activity index is calculated to be 155, demonstrating response to treatment. Repeat lower limb Doppler and echocardiography after 3 months revealed significant resolution of thrombus, and the patient is currently being maintained on oral anticoagulation.

The risk for deep venous thrombosis and pulmonary embolism in IBD is increased by almost three-fold compared with that in the general population (1). Arterial thrombosis has been also documented with IBD, albeit less commonly (2). Cardiac involvement in the form of ventricular thrombus is rarely seen, with only few sporadic cases reported. Ventricular thrombus has been reported more frequently in the left ventricle, with only one reported case of right ventricular thrombus in IBD (3, 4). The exact pathogenesis of hypercoagulable state in IBD is not well understood. However, it is postulated that endothelial dysfunction, inflammation, alteration in coagulation cascade, and fibrinolytic system might play a role in thrombus formation (5,6). Treatment to decrease the acute inflammatory state along with anticoagulation remains the mainstay of IBD management. In the acute setting, heparin or low-molecular-weight heparin is being used followed by warfarin as maintenance therapy. Once the patient overcomes the acute phase, prognosis is usually good.

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