

Left Ventricular Thrombus and Cardioembolic Stroke in a Patient with Ulcerative Colitis: A Case Report

Hardeep Kaur Grewal¹, Manish Bansal¹, Arun Garg², Ravi R. Kasliwal¹, Anil Bhan³, Dheeraj Gautam⁴

Departments of ¹Clinical and Preventive Cardiology, ²Neurology, ³Cardiac Surgery and ⁴Laboratory Medicine, Pathology and Blood Bank, Medanta-The Medicity, Gurgaon, Haryana, India

Abstract

Left ventricular (LV) thrombi usually occur in the setting of global or regional LV systolic dysfunction and are extremely rare in the absence of LV wall motion abnormalities. We report here a case of a 23-year-old female who presented with cardioembolic stroke due to ulcerative colitis. To determine the cause of stroke, several investigations and evaluations were carried out, but the results were mostly normal or unremarkable. Transthoracic echocardiography revealed an oscillating pedunculated globular mass, which was eventually resected due to recurrent transient ischemic attacks. The histopathology of the excised mass revealed it to be an organized thrombus with acute and chronic inflammatory cells and fibroblasts. The uncommon etiology combined with the unusual appearance of the thrombus presented a major diagnostic and therapeutic dilemma for this exceedingly rare cause for intracardiac thrombus formation. Therefore, it would be useful to have a low threshold for screening patients with active inflammatory bowel disease for possible ventricular thrombosis before discharge, especially if other risk factors are present.

Keywords: Cardioembolic stroke, Crohn's disease, inflammatory bowel disease, intracardiac clot, left ventricular thrombus, ulcerative colitis

Address for correspondence: Dr. Manish Bansal, Clinical and Preventive Cardiology, Medanta-The Medicity, Sector 38, Gurgaon - 122 001, Haryana, India.
E-mail: manishaiims@hotmail.com

Submitted: 20-Nov-2019 **Revised:** 16-Apr-2020 **Accepted:** 20-Aug-2020 **Published:** 26-Dec-2020

INTRODUCTION

Patients with inflammatory bowel disease (IBD) are at increased risk of developing thromboembolic complications.^[1-6] The venous system is most commonly involved, manifesting as peripheral venous thrombosis, with or without pulmonary embolism. In a large series involving patients with IBD flare-up, the absolute risk of venous thromboembolism (VTE) in hospitalized patients was 37.5/1000 person-years as compared to 13.9/1000 person-years in the control group.^[7] The corresponding figures for the ambulatory patients were 6.4 and 0.4/1000 person-years, respectively. Unlike VTE,

intracardiac thrombus formation, especially on the left side, with systemic embolism is exceedingly rare, with only five such cases having been reported in patients with ulcerative colitis (UC)^[8-12] and three in patients with Crohn's disease.^[13,14] We report here a case of a young female who presented with a cardioembolic stroke due to UC. The unexpected etiology and the unusual appearance of the thrombus, as discussed below, presented a major diagnostic and therapeutic dilemma for the clinical team. This case highlights the need for keeping a high index of suspicion for intracardiac thrombosis in all patients with IBD who present with vascular complications.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Grewal HK, Bansal M, Garg A, Kasliwal RR, Bhan A, Gautam D. Left Ventricular thrombus and cardioembolic stroke in a patient with ulcerative colitis: A case report. Saudi J Med Med Sci 2021;9:67-70.

Access this article online

Quick Response Code:



Website:

www.sjmms.net

DOI:

10.4103/sjmms.sjmms_525_19

CASE REPORT

A 23-year-old female presented with persistent diarrhea for 2–3 weeks and was diagnosed with UC. She was started on mesalamine 2.4 g/day and prednisolone 40 mg/day, which resulted in prompt resolution of her abdominal symptoms. However, 3 weeks later, she developed sudden-onset weakness on the right side of the body along with aphasia. She presented to the emergency room within 1 h of the symptom onset. Urgent computed tomography (CT) scan of the brain was done, which showed a left middle cerebral artery territory infarct. She immediately underwent thrombolytic therapy with tissue plasminogen activator (tPA), resulting in significant neurological recovery (improvement in the right upper and lower limb motor power to Grade 4/5 with resolution of aphasia). This was followed by subcutaneous, weight-adjusted enoxaparin (40 mg) twice daily. Over the next 5 days, she had two more episodes of transient ischemic attacks (TIAs). Repeat CT did not show any specific new change, and thus, it was decided to continue with conservative treatment only. She denied any abdominal, respiratory and cardiovascular symptoms during this hospital stay.

After the initial acute management, she underwent a further evaluation to find the cause of stroke. Blood investigations revealed microcytic, hypochromic anemia (hemoglobin 9.24 g/dL, hematocrit 30.7%, mean corpuscular volume 68.2 fL and mean corpuscular hemoglobin 20.4 pg) with raised white blood cell and platelet counts (17,500 cells/mm³ and 581,000 cells/mm³, respectively). Renal and liver function tests were within normal limits. C-reactive protein (26.3 mg/L), erythrocyte sedimentation rate (47 mm in the first hour), serum procalcitonin (0.10 ng/mL) and beta-2-microglobulin (1890 ng/mL) were raised, suggesting that she was in active state of inflammation. Chest X-ray was normal, and the autoimmune profile was negative, except for antinuclear antibody being positive at 1:80 dilution, which was considered clinically insignificant by the immunologist. Her low-density lipoprotein cholesterol was 75 mg/dL and high-density lipoprotein cholesterol was 92 mg/dL.

The patient also underwent a cardiac evaluation to rule out any cardiac source of embolism. Both 12-lead electrocardiogram and 24-h Holter monitoring were unremarkable. However, transthoracic echocardiography revealed an oscillating pedunculated globular mass attached to left ventricular (LV) apex. The mass was measuring 1.9 cm × 1 cm in size and had a thin stalk [Figure 1]. The LV systolic function was normal with no regional wall motion abnormality. There was no other remarkable finding on

echocardiography. Transesophageal echocardiography was also done, which confirmed these findings, and at the same time, excluded other potential sources of cardioembolism such as left atrial appendage thrombus, patent foramen ovale and aortic atheroma. Based on the echocardiographic findings alone, it was not possible to ascertain the etiology of the mass, but thrombus was considered the first possibility, as it is the most common cause of a cardiac mass. However, certain features of the mass were not consistent with the thrombotic nature, i.e., normal LV contractility, pedunculated morphology of the mass and no reduction in its size despite continued anticoagulation. The patient's thrombophilia scan (including factor V Leiden, cardiolipin antibodies, lupus antibodies and protein C and protein S) was also completely normal. These findings raised the possibility of an alternative etiology of the mass, possibly a tumor, but an LV tumor is much less common than an LV thrombus. Cardiac magnetic resonance imaging (MRI) was contemplated but not performed because the mass was very mobile, and it was believed that MRI would not be able to image it adequately.

As the patient had recurrent episodes of TIAs despite being on anticoagulation and was at risk of having a major embolic catastrophe, surgical excision of the LV mass was undertaken, despite the risk of perioperative intracranial bleed. The histopathology of the excised mass revealed it to be an organized thrombus with acute and chronic inflammatory cells and fibroblasts [Figure 2]. There was no perioperative complication, and she was discharged to home 6 days after surgery in a stable condition. Her discharge medications included enteric-coated aspirin 75 mg daily, warfarin (dose-adjusted according to prothrombin time/international normalized ratio) and mesalamine 1.2 g (two tablets in the morning and one tablet in the evening). Aspirin was discontinued after a few weeks but warfarin and mesalamine were intended for long-term. She remained stable during the follow-up and last visited our hospital at approximately three years after her initial presentation.

DISCUSSION

The incidence of systemic thromboembolic events in IBD patients ranges from 1% to 8% in clinical studies^[1,2] and approximately 40% in postmortem studies.^[3-5] The risk of thrombosis is higher when the disease is active and most of the thrombotic complications occur during an exacerbation of IBD.^[7] In a large study involving 13,756 patients with IBD and 71,672 matched controls, the overall risk of VTE in IBD was 2.6/1000 person-years, which increased to 9.0/1000 person-years during an active flare.^[7] The risk was much greater in hospitalized patients (37.5/1000 person-years) as

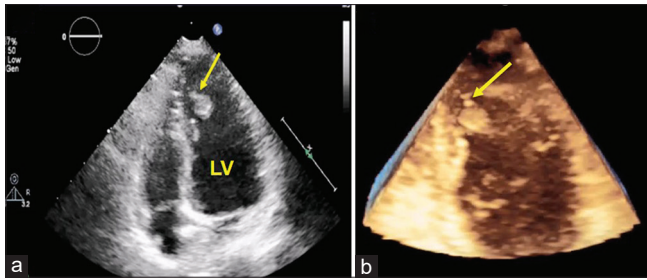


Figure 1: (a) Apical four-chamber view showing globular pedunculated mass attached to the left ventricular apex with a thin stalk (yellow arrow); (b) the same finding in three-dimensional echocardiographic image. LV – Left ventricular

compared with ambulatory patients (6.4/1000 person-years). Most of the thrombotic events involve peripheral vessels, with venous thrombosis being several times more common than arterial thrombosis. In a study from the Mayo Clinic, only 7 of 7199 patients of IBD were found to have arterial thrombosis.^[6] Intracardiac thrombus formation is even rare, as discussed below.

Cardiac involvement in IBD has been reported mainly in the form of sporadic case reports. Pericarditis is the most frequent type of cardiac complication encountered in IBD, whereas myocarditis and perimyocarditis occur much less commonly. Autoimmunity, drug-related side effects and idiopathic origin are some of the mechanisms implicated in causation of these complications. Endocarditis can also occur as a result of septicemia, resulting from prolonged use of total parenteral nutrition catheters or immunosuppression.

LV thrombus formation is extremely rare in IBD patients. On reviewing the literature, we only found five such cases in those with UC^[8-12] and three in those with Crohn's disease.^[13,14] Chin *et al.* reported a patient with UC and cocaine abuse who presented with an LV thrombus with lower limb arterial embolism requiring urgent popliteal embolectomy.^[9] Another case report described a patient with UC who had initially presented with abdominal symptoms and bloody diarrhea but subsequently developed stroke. Echocardiography revealed a large LV thrombus with normal LV contractility.^[8] Lutz *et al.* described a pedunculated LV apical thrombus in a patient with UC, similar to our case. The thrombus was incidentally detected but underwent robot-assisted surgical excision in view of its pedunculated nature and high risk of embolization.^[10]

Thrombosis in IBD is believed to be multifactorial in origin.^[15] The underlying inflammatory milieu and autoimmunity result in increased circulating levels of various cytokines and prothrombotic factors that

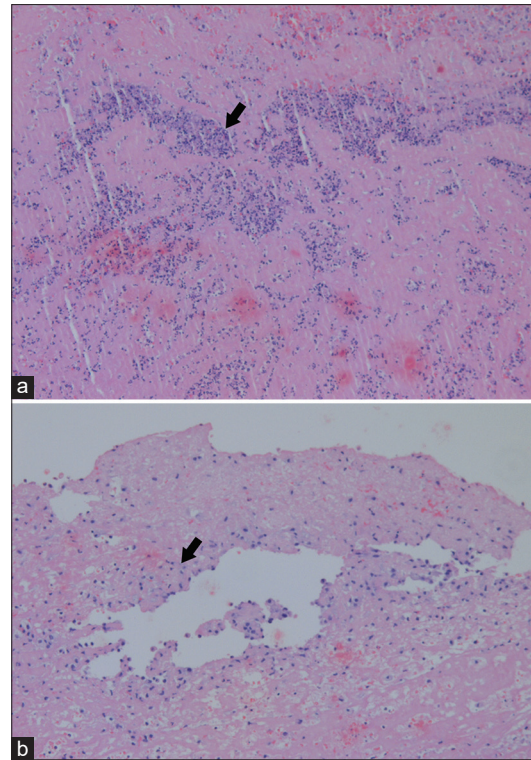


Figure 2: Histopathology of the excised mass confirming it to be a thrombus. (a) Organized thrombus with neutrophilic infiltrate (arrow); (b) fibroblasts (arrow) within the thrombus

heighten thrombogenicity. Apart from these, some of the IBD medications including sulfasalazine, methotrexate, azathioprine, cyclosporine and prednisolone also indirectly create a prothrombotic state.^[15] Conversely, decreased tPA activity and increased plasminogen activator inhibitor levels result in depressed fibrinolysis. Finally, hyperhomocystinemia is also a recognized independent risk factor for thrombosis in IBD patients.^[16,17] In our patient, we attributed the LV thrombus to UC because she was in an active state of inflammation, was receiving prednisolone and did not have any alternative identifiable cause for LV thrombus formation.

Early detection and intervention are critical in patients with LV thrombi as there is approximately 2%–3% risk of systemic embolization.^[18] There is no single laboratory test that can predict the occurrence of thrombosis. Therefore, it would be desirable to have a low threshold for screening patients with active IBD for possible ventricular thrombosis before discharge, especially if other risk factors are present. At the same time, additional modifiable risk factors should be minimized.

CONCLUSIONS

This case highlights the thrombogenic potential of IBD,

especially in the active phase of the disease. Although venous thrombosis is more common, thrombus formation can very rarely also involve cardiac chambers, even in the absence of regional wall motion abnormality. Therefore, a low threshold should be kept for using imaging techniques in these patients, especially when they present with embolic complications.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Giannotta M, Tapete G, Emmi G, Silvestri E, Milla M. Thrombosis in inflammatory bowel diseases: What's the link? *Thromb J* 2015;13:14.
2. Freeman HJ. Venous thromboembolism with inflammatory bowel disease. *World J Gastroenterol* 2008;14:991.
3. Murthy SK, Nguyen GC. Venous thromboembolism in inflammatory bowel disease: An epidemiological review. *Am J Gastroenterol* 2011;106:713-8.
4. Yuhara H, Steinmaus C, Corley D, Koike J, Igarashi M, Suzuki T, *et al.* Meta-analysis: The risk of venous thromboembolism in patients with inflammatory bowel disease. *Aliment Pharmacol Ther* 2013;37:953-62.
5. Tan VP, Chung A, Yan BP, Gibson PR. Venous and arterial disease in inflammatory bowel disease. *J Gastroenterol Hepatol* 2013;28:1095-113.
6. Talbot RW, Heppell J, Dozois RR, Beart RW Jr. Vascular complications of inflammatory bowel disease. *Mayo Clin Proc* 1986;61:140-5.
7. Grainge MJ, West J, Card TR. Venous thromboembolism during active disease and remission in inflammatory bowel disease: A cohort study. *Lancet* 2010;375:657-63.
8. Saleh T. Left ventricular thrombosis in ulcerative colitis. *Case Rep Gastroenterol* 2010;4:220-3.
9. Chin WW, van Tosh A, Hecht SR, Berger M. Left ventricular thrombus with normal left ventricular function in ulcerative colitis. *Am Heart J* 1988;116:562-3.
10. Lutz CJ, Bhamidipati CM, Ford B, Swartz M, Hauser M, Kyobe M, *et al.* Robotic-assisted excision of a left ventricular thrombus. *Innovations (Phila)* 2007;2:251-3.
11. Baratta P, Pasqualini P, Misuraca L, Minelli S, Chiavarelli M, Severi S, *et al.* Thrombotic storm in a teenager with previously diagnosed ulcerative colitis: A rare case of left ventricular thrombosis. *G Ital Cardiol (Rome)* 2017;18:664-7.
12. Willner N, Schiff E, Goitein O, Rainis T. Left ventricular thrombosis in a young ulcerative colitis patient: Review with a case report. *Eur J Case Rep Int Med* 2015;2:doi: 10.12890/2015_000268.
13. Rasalingam R, Saeed IM, Woodard PK, Pérez JE. Left ventricular thrombus in the setting of normal left ventricular function in patients with Crohn's disease. *Echocardiography* 2016;33:145-9.
14. Iyer A, Marney L, Ipp S, Bough G, McCoombe D, Tam R, *et al.* Recurrent left ventricular thrombus in Crohn's disease: A rare presentation. *Asian Cardiovasc Thorac Ann* 2014;22:86-8.
15. Magro F, Soares JB, Fernandes D. Venous thrombosis and prothrombotic factors in inflammatory bowel disease. *World J Gastroenterol* 2014;20:4857-72.
16. Banait VS, Sandeep MS, Shetty S, Bapat MR, Rathi PM, Ghosh K, *et al.* Hypercoagulable state in idiopathic ulcerative colitis: Role of hyperhomocysteinemia and hyperfibrinogenemia. *Indian J Gastroenterol* 2008;27:94-8.
17. Oussalah A, Guéant JL, Peyrin-Biroulet L. Meta-analysis: Hyperhomocysteinemia in inflammatory bowel diseases. *Aliment Pharmacol Ther* 2011;34:1173-84.
18. O'Sullivan CJ, Eberli FR. Left ventricular thrombus formation after acute myocardial infarction: Vigilance still required in the modern era. *Swiss Med Wkly* 2015;145:w14158.