

J Med Allied Sci 2017; 7 (1): 64-66

www.jmas.in

Print ISSN: 2231 1696 Online ISSN: 2231 170X

Journal of Medical & Allied Sciences

Case report

Unusual case of stroke in young

Dhavala Aashitha¹, T. Umesh¹, Mohammed Aejaz Habeeb²

¹Department of Neurology and ²Department of Gastroenterology, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad-500058, Telangana, India.

Article history

Received 23 November 2016 Accepted 23 December 2016 Early online 05 January 2017 Print 31 January 2017

Corresponding author

T. Umesh

Professor, Department of Neurology, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad-500058, Telangana, India. Phone: +91-9248033372 Email: drumesh40@hotmail.com Thromboembolism is a rare extra-intestinal manifestation of inflammatory bowel disease. Thromboembolic event involving CNS in a case with inflammatory bowel disease (IBD) is very rare & leads to increase in mortality. The mortality of ulcerative colitis with thromboembolic complications have been reported to reach 25 percent. Here, we present a case of a young adult with a two years old history of ulcerative colitis with a sudden onset of stroke with no other associated co-morbidities for cerebrovascular disease.

Abstract

Key words: Anticoagulation therapy, homocysteine, hypercoagulable states, stroke, thromboembolic event, ulcerative colitis

DOI: 10.5455/jmas.249352

© 2017 Deccan College of Medical Sciences. All rights reserved.

Licerative colitis (UC) which is an inflammatory disease of the bowel rarely leads to thromboembolic complications which lead increased mortality and morbidity. Deep vein thrombosis (DVT) and pulmonary thromboembolism are the most common thromboembolic complications in IBD cases¹ whereas stroke and myocardial infarction are rare.

Case report

A 27 year old male patient visited to our hospital with recurrent abdominal pain, vomiting and bloody diarrhea of 2 years duration, earlier diagnosed as ulcerative colitis. During a week of hospitalization, the patient had transient loss of consciousness lasting for few minutes following which he developed sudden loss of power in right arm and leg with cessation of speech and deviation of angle of his mouth towards the left. He did not have significant family history.

His blood pressure recorded was 110/80 mm Hg, heart rate was 88 beats/min and normal temperature. He had pallor, grade 2 clubbing and pitting edema of right leg. Neurologically, he was conscious, alert, comprehending with reduced word output. He had right facial weakness with grade 0-1 power in right limbs.



Fig 1. Colonoscopy (A) Ascending colon showing few scattered ulcers in healing phase with normal intervening mucosa and (B) Transverse colon showing multiple healing pseudopolyps and few healing ulcers - Early remission phase of ulcerative colitis

Detailed biochemical investigations revealed low hemoglobin (8 gm/dl) with lower normal range platelet count (1.5 lakhs/cumm), normal total leukocyte count (8,000 cells/cumm). Hypercoagulability work up revealed raised partial thromboplastin time (PTT), raised prothrombin time/international normalized ratio (PT/INR), reduced protein-C, reduced factor V and reduced antithrombin III levels. Antinuclear antibodies (ANA) levels were within normal limits. Ultrasonography showed mild to moderate ascites with hepatomegaly and portal hypertension. Echocardiogram was normal. Colonoscopy showed multiple healing ulcers (Fig 1). Computed tomography of brain revealed left side middle cerebral artery (MCA) cortical infarct (Fig 2).



Fig 2. CT Brain (Plain) showing infarct in left parieto-temporal region

The patient was given blood transfusions to compensate his ongoing blood loss. He was given anticoagulants, aspirin, statins, ACE inhibitors and cerebroprotein hydrolysate. Patient was hospitalized for 2 weeks; during his stay he did not show any signs of improvement but left with residual right hemiplegia subsequently. Patient was discharged advising rehabilitation program.

Discussion

The association of inflammatory bowel disease (IBD) with thrombosis was first described by Bargen and Baker in 1936². Four decades later it was Patterson, who described thromboembolic event involving central nervous system (CNS) in a child with IBD³. During 1970-1980 a study revealed thromboembolic complications in 92 cases (1.3 %) among 7,199 cases with IBD⁴. Thromboembolic events involving CNS in IBD patients are unusual and devastating. Arterial thromboembolisms are rare. Reports claim that two thirds of thromboemboli are venous and one third are arterial⁵. Houissa et al described arterial thrombosis in 4 cases, 3 of them younger than 25 years⁶. Arterial thromboembolisms are usually post surgery cases.

Ulcerative colitis (UC) cases are reported to be more commonly complicated with thromboembolic events. Pan-colonic disease has been reported as a major risk factor for stroke⁷. Women are reported to be effected more than men; this could be due to the use of oral contraceptives. Stroke usually occurs during the active stage of IBD in young adults or during gastrointestinal (GI) complications like abscess or fistula⁸.

Thromboembolism associated with IBD usually presents with early atherosclerosis and increased homocysteine⁹. Hyperhomocysteinemia associated with increased thrombosis is due to increase in factor V, factor VIII and platelet abnormalities¹⁰. Increased homocysteine levels are associated with vitamin B6, vitamin B12 and folate deficiency which are involved in homocysteine metabolism while vitamin D deficiency could be due to malabsorption or reduced dietary intake.

Inherited hypercoagulable states were reported to be a cause of stroke¹¹. High prevalence of thrombosis in IBD case could be associated with hereditary factors such as methylene tetrahydrofolate reductase (MTHFR), factor V, prothrombin gene mutation, antithrombin III deficiency, protein C and protein S abnormalities. Our case had reduced protein C levels, raised PTT and PT/INR.

In IBD cases, aggregation of platelets in mesenteric vessels leads to release of platelet activating factor and thromboxane production leading to further thrombus formation.

Association of maternal active ulcerative colitis with fetal infarcts was reported by Scher¹². Consideration of other co-morbidities such as sepsis, post surgery, immobility, contraception, smoking, obesity, etc should be done.

Conclusion

Thromboembolic events should not be overlooked in cases with IBD and prophylactic treatment should be given. Prophylactic alteration in modifiable risk factors such as smoking, obesity, diet, contraception should be done. Anticoagulation therapy should be started in high risk cases¹³. While use of low molecular weight heparins (LMWH) are still under debate¹⁴. Few reports suggest the use of TNF inhibitors as pro-inflammatory cytokines TNF and IL-6 are said to be increased in these patients¹⁵. Prophylactic protocolectomy has been reported to prevent thromboembolic events in IBD cases while few reports deny it.

Acknowledgments: None

Conflict of interest: None

References

- Srirajaskanthan R, Winter M, Muller AF. Venous thrombosis in inflammatory bowel disease. Eur J Gastroenterol Hepatol 2005 Jul; 17(7):697-700.
- Bargen JA, Barker NW. Extensive arterial and venous thrombosis complication chronic ulcerative colitis. Arch Intern Med 1936; 58(1):17-31.
- Patterson M, Castiglioni L, Sampson L. Chronic ulcerative colitis beginning in children and teenagers. Am J Dig Dis. 1971 Apr; 16(4):289-297.
- Talbot RW, Heppell J, Dozius RR, Beart RW Jr. Vascular complications of inflammatory bowel disease. Mayo Clin Proc. 1986 Feb; 61(2):140-145.
- Joshi D, Dickel T, Aga R, Smith-Laing G. Stroke in inflammatory bowel disease: a report of two cases and review of literature. Thromb J. 2008 Mar 21; 6:2.
- Houissa F, Salem M, Bouzaidi S, Rejeb MB, Mekki H, Debbeche R, Moussa A, Trabelsi S, Said Y, Najjar T. Cerebral thrombosis and inflammatory bowel disease: a report of four cases. J Crohns Colitis. 2011 Jun; 5(3):249-252.
- Solem CA, Loftus EV, Tremaine WJ, Sandborn WJ. Venous thromboembolism in inflammatory bowel disease. Am J Gastroenterol. 2004 Jan; 99(1):97-101.
- Nguyen GC, Sam J. Rising prevalence of venous thromboembolism and its impact on mortality among hospitalized inflammatory bowel disease patients. Am J Gastroenterol. 2008; 103(9):2272-2280.

- Roifman I, Sun YC , Fedwick JP, Panaccoine R, Buret AG, Liu H, Rostom A, Anderson TJ, Beck PL. Evidence of endothelial dysfunction in patients with inflammatory bowel disease. Clin Gastroenterol Hepatol. 2009 Feb; 7(2):175-182.
- Zezos P, Papaioannou G, Nikolaidis N, Vasiliadis T, Giouleme O, Evgenidis N. Hyperhomocysteinemia in ulcerative colitis is related to folate levels. World J Gastroenterol. 2005 Oct 14; 11(38):6038-6042.
- Owczarek D, Cibor D, Glowacki MK, Rodacki T, Mach T. Inflammatory bowel disease: epidemiology, pathology and risk factors for hypercoagulability. World J Gastroenterol. 2014 Jan 7; 20(1):53-63.
- Scher MS. Maternal ulcerative colitis and fetal brain injury: long term neurologic outcome. J Child Neurol. 2007 Nov; 22(11):1293-1296.
- Ra G, Thanabalan R, Ratneswaran S, Nguyen GC. Predictors and safety of venous thromboembolism prophylaxis among hospitalized inflammatory bowel disease patients. J Crohns Colitis. 2013; 7(10):e479-485.
- 14. Pastorelli L, Saibeni S, Spina L, Signorelli C, Celasco G, de Franchis R, Vecchi M. Oral, colonic-release low-molecularweight heparin: an initial open study of Parnaparin-MMX for the treatment of mild-to-moderate left-sided ulcerative colitis. Aliment Pharmacol Ther. 2008; 28(5):581-588.
- Nielson OH, Ainsworth MA. Tumor necrosis factor inhibitors for inflammatory bowel disease. N Engl J Med. 2013 Aug 22; 369(8):754-762.