CASE REPORT

A Case of Transient Ischemic Attack in a patient with Ulcerative Colitis

Valavanis A, Tegos T

1st Neurological Department, Aristotle University of Thessaloniki, AHEPA General Hospital, Thessaloniki, Greece

Abstract

Arterial stroke is a rare complication of Ulcerative Colitis (UC) and so far there are no guidelines for the treatment of stroke in these patients. The pathogenesis of thrombosis in UC remains uncertain. This case is one of the few published reports on the relationship between stroke associated with UC and the factor V Leiden mutation.

Key Words: Transient ischemic attack, ulcerative colitis

Corresponding Author: Athinodoros Valavanis, 1st Neurological Department of Aristotle University, 54636 Thessaloniki, Greece, tel: +306974501202, e-mail: athis78@hotmail.com

Introduction

Thromboembolic complications with extra intestinal manifestation are well recognized in inflammatory bowel disease (IBD) since 1936[1]. Ulcerative Colitis (UC) is frequently discussed together with Crohn's disease under the heading IBD, nevertheless are discrete entities. Arterial stroke is rare in UC and scattered cases are reported to the literature. No guidelines are available at present to help manage this severe complication. A case of transient ischemic attack in a patient with UC is described here.

Case Report

A 49 year old, non-smoker, Caucasian man, with negative family history for thromboembolic disease, presented with headache and left sided hemiparesis. A diagnosis of UC had been made one year previously and was started on sulfasalazine 2g daily. On admission he was well nourished, normotensive and his body temperature was 37,3 ºC. Cardiovascular examination was normal. Neurological examination revealed sensory loss and hemiparesis with pyramidal signs on left side. Carotid Doppler and Computed Tomography of the brain were normal.

Laboratory tests revealed a normochromic, normocytic anemia with a hemoglobin concentration of 12.3g/dl, white cell normal count, an elevated sedimentation rate of 41 mm/h, and a raise of CRP of 27mg/L. Autoimmune markers, tests for VDRL, cryoglobulins, anticardiolipin and lupus erythematosus were negative. The fundus of the eyes was normal. These investigations excluded vasculitis. A coagulation test and haemostatic parameters were normal. Laboratory tests revealed homozygous factor V Leiden gene mutation for wild type. Magnetic resonance imaging 24 hours after admission was normal. The patient treated with aspirin with full recovery of neurological deficits.

Discussion

Deep vein thrombosis and pulmonary embolism remains the most common vascular complication of IBD. Thrombotic events involving the central nervous system are much less common, especially stroke. Active disease was associated with the occurrence of thrombotic events, but there are reports[2] that patients in remission are in haemostatic imbalance in favour of coagulation. Thus, clinical activity of the disease may increase the risk, but it is probably of minor importance.

The pathogenesis of thrombosis in UC remains uncertain. Attention has been drawn to protein C and its cofactor protein S, which are naturally occurring vitamin K dependent plasma anticoagulants. The prevalence of vitamin K[3] deficiency in patients with UC suggested as a cause of temporarily deficiency of protein C, protein S and prothrombin but there is lack of evidences for the pathogenetic role of several thrombotic factors in what is considered as a hypercoagulable state[4] associated with UC[5]. Hyperhomocysteinaemia is common in IBD patients, as a result of malabsorption and medication with folate depleting agents, but according to some authors elevated total homocysteine levels do not contribute to the development of thrombotic complications[6] , although there is an argument[7].

All the above thrombogenic factors and a long list of autoimmune causes of cerebral vasculitis excluded. Our patient was also investigated for the presence of the factor V Leiden mutation and was found homozygous for wild-type. There are few published reports[8] on the relationship between thrombosis associated with IBD and the factor V Leiden mutation.

There is no consensus in the literature regarding the treatment of stroke in UC. Prophylactic doses of LMWH are used in patients with IBD under the recommendation by British Society of Gastroenterology[9]. Anticoagulation is controversial because of the increased risk of intracerebral and GI bleeding. In conclusion, further research is needed to illuminate the hypercoagulable state in UC and guidelines are required in the management of stroke in UC.

References

1. Bargen JA, Barker NW. Extensive arterial and venous thrombosis complicating chronic ulcerative colitis. Arch Intern Med. 1936; 58:17-31.
2. van Bodegraven AA, Schoorl M, Baak JP, Linskens RK, Bartels PC, Tuynman HA. Hemostatic imbalance in active and quiescent ulcerative colitis. Am J Gastroenterol. 2001; 96: 487-493.
3. Krasinski SD, Russel RM, Furie BC, Kruger SF, Jacques PF, Furie B. The prevalence of vitamin K deficiency in chronic gastrointestinal disorders. Am J Clin Nutr. 1985; 41: 639-643.
4. Shafer AI. The hypercoagulable states. Ann Intern Med. 1985; 102: 814-828.
5. Danese S, Papa A, Saibeni S, Repici A, Malesci A, Vecchi M. Inflamation and coagulation in inflammatory bowel disease: The clot thickens. Am J Gastroenterol. 2007; 102: 174-186.
6. Oldenburg B, Fijnheer R, van der Griend R, vanBerge-Henegouwen GP, Koningsberger JC. Homocysteine in inflammatory bowel disease: a risk factor for thromboembolic complications? Am J Gastroenterol. 2000; 95: 2825-2830.
7. Scheid R, Teich N. Neurologic manifestation of ulcerative colitis. Eur J Neurol. 2007; 14: 483-493.
8. Liebman HA, Kashani N, Sutherland D, McGehee W, Kam AL. The factor V Leiden mutation increases the risk of venous thrombosis in patients with inflammatory bowel disease. Gastroenterology. 1998; 115: 830-834.
9. Carter MJ, Lobo AJ, Travis SP; IBD Section, British Society of Gastroenterology. Guidelines for the management of inflammatory bowel disease in adults. Gut. 2004; 53: V1-V16.