

Case Report

Ulcerative colitis flare with splenic vein thrombosis

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Abstract: Patients with ulcerative colitis (UC) have an increased risk of thromboembolic events. Here, we present a 28-year-old man with active ulcerative pancolitis presenting via splenic vein thrombosis and left renal superior infarct that was not associated with a surgical procedure.

Keywords: Ulcerative colitis, splenic vein thrombosis, renal infarct

Thrombotic events related with ulcerative colitis are not frequent but might cause severe complications as deep venous thrombosis, pulmonary emboli, portal vein thrombosis, cerebral venous sinus thrombosis, Budd Chiari syndrome, retinal vein thrombosis, myocardial infarction, visceral ischemia [1-3]. The overall incidence rate of venous thromboembolism (VTE) in IBD patients has been estimated to be 1-8% [2-4]. Here, we report a case of splenic vein thrombosis and left renal superior infarct that had not been reported before, in a young man with active ulcerative pancolitis.

A 28-year-old man was admitted with hemorrhagic, mucous diarrhea and abdominal pain. He was suffering from bloody diarrhea 8 to 10 times daily for a month and lost 6 kgs (10% of original weight). He had no smoking, no surgical procedure and his body mass index was 22 kg/m². On the physical examination he had abdominal pain and bowel sounds were hyperactive. Laboratory findings showed thrombocytosis (612,000/mm³), increased erythrocyte sedimentation rate (62 mm/h), and high serum C-reactive protein (147 mg/L) levels. He had no proteinuria. All coagulation parameters, such as prothrombin time (pt), activated partial thromboplastin time (aptt), protein C, protein S and anti-thrombin III levels were normal. Serum homocysteine level was 8, 45 (N: 0-10) mg/dL. Serum folate, vitamin B12 were within normal limits. G20210A, methylenetetrahydrofolate

reductase (MTHFR) (677 CC) mutations, JAK2 (p.V617F) were normal. Factor V G1691A (Leiden) mutation was heterozygous.

He was treated with mesalazine p.o. and enema. His bloody diarrhea and stool frequency returned to normal limits in two days but his abdominal pain and high serum C-reactive protein continued. His contrast enhanced abdominal computed tomography reported splenic vein thrombosis and left renal superior infarct (**Figure 1**). Low-molecular-weight heparin was given for prophylaxis of thrombosis recurrence. Transthoracic echocardiography, lower extremity venous Doppler examination were normal. One month later after discharge he were asymptomatic and he was taken control contrast enhanced abdominal computed tomography which were reported normal. Mesalazine and anticoagulant treatment were continued.

Here, we reported a young man with active ulcerative pancolitis complicated by splenic vein thrombosis and left renal superior infarct. Thromboembolic events are rarely seen and might be related with the activity of the disease. Underlying mechanisms are not clear, and thrombosis might also play a role in the pathogenesis of UC. The etiopathogenesis of thrombotic events related with IBD is widely debated, and coagulation alterations and fibrinolysis have been suggested to play the major role in this process [5]. Grainge MJ et al showed asso-



Figure 1. Splenic vein thrombosis (Thin arrow) and left renal superior infarct (Thick arrow).

ciation VTE and IBD flares in a cohort study [6]. According to the data from this assessment, the risk of VTE was increased most prominently during a flare of IBD, compared with periods of chronic activity and periods of clinical remission. Bernstein et al also showed higher VTE rates in hospitalized UC patients than in non-UC hospitalized patients regardless of age [7]. Nguyen et al compared the risk of VTE between hospitalized IBD patients and randomly selected hospitalised non-IBD patients and reported that IBD patients had an adjusted 1.7-fold increased rate of VTE compared with non-IBD patients [8].

The most prevalent thrombophilia reported in IBD patients is factor V mutation. However, the prevalence of factor V mutation in thrombotic IBD patients has been shown to be significantly higher than that in IBD patients without thrombosis, suggesting that factor V Leiden, when present, increases the risk of IBD-associated VTE [9]. Two recent meta-analyses confirmed this conclusion [10, 11]. The factor V Leiden mutation was associated with a significantly higher risk of thromboembolism in IBD patients. When genetic risk factors occur, patients with IBD (compared with healthy controls) are more likely to suffer thromboembolic complications, suggesting that hereditary thrombophilia and inflammation-associated thrombogenicity have at least an additive effect for the risk of VTE in IBD.

In conclusion, in IBD, there is an increased risk of thromboembolic events due to inflammation, nutritional deficiencies, hospitalization, surgery and inherited prothrombotic factors. Thromboembolic events might be presented as abdominal pain. We must be aware of this situation, especially in young and hospitalized IBD pati-

ents. Importantly, VTE appears to carry a poorer prognostic outcome for patients with IBD than for the general population. Though a routine approach for this situation is not clear, consideration should be given to heparin, low-molecular-weight heparin or other medical choices for the treatment or prevention of thrombotic events. Further studies are needed to reach a consensus for the treatment of this troubling complication.

Disclosure of conflict of interest

None.

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References

- [1] Murthy SK, Nguyen GC. Venous thromboembolism in inflammatory bowel disease: an epidemiological review. *Am J Gastroenterol* 2011; 106: 713-718.
- [2] Yuhara H, Steinmaus C, Corley D, Koike J, Igarashi M, Suzuki T, Mine T. Meta-analysis: the risk of venous thromboembolism in patients with inflammatory bowel disease. *Aliment Pharmacol Ther* 2013; 37: 953-962.
- [3] Tan VP, Chung A, Yan BP, Gibson PR. Venous and arterial disease in inflammatory bowel disease. *J Gastroenterol Hepatol* 2013; 28: 1095-1113.
- [4] Scoville EA, Konijeti GG, Nguyen DD, Sauk J, Yajnik V, Ananthakrishnan AN. Venous thromboembolism in patients with inflammatory bowel diseases: a case-control study of risk factors. *Inflamm Bowel Dis* 2014; 20: 631-6.
- [5] Rankin GB. Extraintestinal and systemic manifestations of inflammatory bowel disease. *Med Clin North Am* 1990; 74: 39-50.
- [6] Grainge MJ, West J, Card TR. Venous thromboembolism during active disease and remission in inflammatory bowel disease: a cohort study. *Lancet* 2010; 375: 657-663.
- [7] Bernstein CN, Nabalamba A. Hospitalization-based major comorbidity of inflammatory bowel disease in Canada. *Can J Gastroenterol* 2007; 21: 507-511.
- [8] 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: 2272-2280.
- [9] Guédon C, Le Cam-Duchez V, Lalaude O, Ménard JF, Lerebours E, Borg JY. Prothrombotic

- inherited abnormalities other than factor V Leiden mutation do not play a role in venous thrombosis in inflammatory bowel disease. *Am J Gastroenterol* 2001; 96: 1448-1454.
- [10] Zhong M, Dong XW, Zheng Q, Tong JL, Ran ZH. Factor V Leiden and thrombosis in patients with inflammatory bowel disease (IBD): a meta-analysis. *Thromb Res* 2011; 128: 403-409.
- [11] Liang J, Wu S, Feng B, Lei S, Luo G, Wang J, Li K, Li X, Xie H, Zhang D, Wang X, Wu K, Miao D, Fan D. Factor V Leiden and inflammatory bowel disease: a systematic review and meta-analysis. *J Gastroenterol* 2011; 46: 1158-1166.