Triad of Emboli in Acute Flare of Ulcerative Colitis

Abstract

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Arterial thrombosis is rare in ulcerative colitis (UC). Our case report described a triad of arterial embolism in a UC patient who presented with bilateral lower limb claudication associated acute chest pain, confusion, ataxia and diplopia. Investigations confirmed bilateral femoral and popliteal artery occlusion, occipital infarct and a sub-endocardial infarct secondary to embolic disease.

Introduction

Venous thrombotic complications are well known features of acute exacerbation of ulcerative colitis, with many fatal episodes relating to pulmonary embolism. Arterial thrombosis is significantly rarer, including cerebral, retinal, hepatic, aorto-iliac, intra-cardiac and splenic thrombus. The relationship between active disease and inflammatory process resulting in a prothrombotic state is the key factor in these complications.

Case Report

A 38-year old man with a 15-year UC history, maintained on azathioprine, presented with a 6-month history of bilateral lower limb claudication. Occasional exacerbations of his colitis had been successfully controlled with corticosteroids. The onset of claudication coincided with an acute flare-up 6 months ago. He also had an episode of severe chest pain, followed by an episode of acute confusion with associated ataxia and diplopia lasting less than 24-hours prior to claudication onset. Bilateral popliteal, dorsalis pedis and posterior tibial pulses were absent. Ankle-brachial indices were 0.98 on the right and 0.30 on the left. A computerized tomographic angiogram showed bilateral popliteal artery occlusion secondary to emboli at the popliteal bifurcation, with proximal propagation of thrombus (Figure 1).

Subsequently, the patient had a magnetic resonance imaging (MRI) of the brain revealing signal changes in the right medial temporal lobe extending posterior to the occipital lobe consistent with an embolism. Cardiac investigations were carried out in search of a central source for these embolic events. Trans-thoracic echocardiogram showed a hypokinetic left ventricle with no evidence of atrial or valvular thrombus. Follow-up trans-oesophageal echocardiogram did not confirm a patent foramen ovale. Subsequent cardiac MRI confirmed a sub-endocardial infarct involving the mid and apical inferior segment of the left ventricle (Figure 2). Coronary angiogram revealed no evidence of atherosclerotic disease. Thrombophilia screen was also negative. Given the patients history of UC, a colonoscopy was performed investigating for occult neoplasm. Biopsies of pseudo-polyps in the transverse colon were benign. Computed tomography of the thorax, abdomen and pelvis did not show an occult neoplasm. The patient was commenced on warfarin, but re-presented several weeks later with a further exacerbation of his colitis. He was re-commenced on high dose steroids combined with infliximab which controlled his condition.

Discussion

There are sparse reports on acute arterial thrombosis in patients with inflammatory bowel disease (IBD). A Mayo Clinic study of 7,199 patients with IBD observed only 7 patients with arterial thrombosis. It is postulated that patients with UC confers a higher risk of arterial thrombosis than those with Crohn’s disease. Though collocty has been advocated for patients with pan-colonic UC to reduce the risk of thrombosis, its benefit has not been accurately quantified. The inflammatory response to acute colitis results in a pro-thrombotic state. Up-regulation of coagulation factors V, VIII, VIII, thrombin, fibrinogen and thromboplastin combined with increased levels of cytokines, tissue necrosis factor alpha (TNF α), interleukin-6 and inhibition of fibrinolysis make the perfect storm for thrombus formation. Studies have shown that IBD medications including sulphasalazine, methotrexate, azathioprine, corticosteroids and anti-TNF agents predispose to pro-thrombotic conditions. Hyperhomocysteinemia is also a recognized independent risk factor for thrombosis. The frequency of hyper-homocysteinemia in patients with IBD is estimated to range between 11-52% and is associated with medications such as methotrexate and sulphasalazine. The treatment of thromboembolism in IBD remains debatable. The British Society of Gastroenterology recommends prophylactic subcutaneous low-molecular weight heparin for all patients experiencing an exacerbation of UC. Studies have shown that heparin and members of the glycosaminoglycans have noteworthy anti-inflammatory properties that are beneficial in acute flare-ups. However this treatment is less feasible in the community, as more than 300 patients would need to be treated to prevent one thromboembolic event.

The incidence of arterial thrombosis during an exacerbation of UC is rare. We report an unusual case describing a triad of arterial thromboembolism. We alert physicians to the potential serious vascular complications associated with UC. However, the role of pharmacological agents still remains unclear.

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References


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