Takayasu arteritis and ulcerative colitis; coexistence or misdiagnosis?

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Abstract. Takayasu arteritis is a chronic inflammatory disease that primarily affects large arteries such as the aorta and its proximal branches. The association between Takayasu arteritis and ulcerative colitis is an extremely rare condition. Ulcerative colitis is an inflammatory bowel disease, clinical presentation is not specific and may mimic Crohn's disease, radiation colitis, ischemic colitis, a variety of infectious processes, and colitis related to medications. Herein we report a case of Takayasu arteritis who had been misdiagnosed and treated as ulcerative colitis. (Sarcoidosis Vasc Diffuse Lung Dis 2012; 29: 53-54)

Key words: Takayasu arteritis, ulcerative colitis, diagnostic errors

Introduction

Takayasu arteritis (TA) is a chronic inflammatory disease that primarily affects large arteries such as the aorta and its proximal branches. It is more common in young females. The association between TA and ulcerative colitis (UC) is an extremely rare condition which may have genetic basis (1). UC is an inflammatory bowel disease involving the colonic mucosa, which is, from its immunological findings, accepted as an autoimmune disorder (2). Clinical presentation is not specific and may mimic Crohn's disease, radiation colitis, ischemic colitis, a variety of infectious processes, and colitis related to medications (3). Diagnosis of UC can usually be established by the characteristic history, typical endoscopic appearance of the mucosa and histological confirmation seen on colonic biopsy.

Vasculitides are rare causes of ischemic colitis than atherosclerosis and thrombotic processes. TA sometimes may result ischemic colitis due to mesenteric arteries involvement. Herein we reported a patient with the diagnosis of TA that was masqueraded as UC for two years.

Case report

Twenty-five years old female patient was hospitalized with the diagnosis of pneumonia and renal failure. She had been treated with sulfasalazine for one year with the diagnosis of UC. However under sulfasalazine treatment, her bowel symptoms including abdominal pain, watery diarrhea and bloody stool did not resolved. Her chest x-ray demonstrated enlargement of mediastinum. Peripheral arterial pulses could not be detected. Initial laboratory examinations showed slight elevation in serum creatinin and urea concentrations and erythrocyte sedi-
ementation rate (ESR) was 118 mm/h. Ultrasonography of the kidneys pointed asymmetry of kidney volumes; right kidney was measured as 80 mm of length which was 32 mm shorter than the left one. Thoraco-abdominal computerized tomography (CT) of the patient revealed thickening of the aorta starting from descending aorta, involving abdominal aorta until 3 cm proximal of iliac bifurcation. Magnetic resonance angiography of the aorta showed stenosis in both renal and superior mesenteric arteries. Colateral circulation has been detected from inferior mesenteric artery to transverse colonic segment. Colonoscopic examination showed edematous and fragile mucosa at hepatic flexure and proximal part of transverse colon. Histopathological findings of the biopsy specimens were compatible with nonspecific colitis. According to these findings the patient was diagnosed as ischemic colitis due to TA. A common genetic basis has been speculated for TA and UC and specific haplotype but there has been no genetical examination was performed for this patient. Thereafter sulfasalazine therapy was stopped and steroid treatment was initiated for TA. One week after treatment urea and creatinin concentrations decreased to normal range with a marked improvement in clinical symptoms including abdominal pain, watery and bloody diarrhea.

Discussion

Takayasu arteritis is a chronic vasculitis and the initial vascular lesion frequently occurs in the left middle or proximal subclavian artery. As the disease progresses, the left common carotid, vertebral, brachiocephalic, right middle or proximal subclavian artery, right carotid and vertebral arteries and the aorta may also be involved. The inflammatory processes cause thickening of the walls of the affected arteries. In most cases the diagnosis is based upon suggestive clinical features and imaging of the arterial tree by magnetic resonance imaging (MRI), CT or angiography.

The co-existence of UC and TA has been reported previously (4-6) and a common genetic basis has been speculated because of the high frequency of specific HLA typing for these two diseases. Inflammatory bowel diseases (IBD) may overlap and sometimes are associated with auto-immune and rheumatologic disorders. Vasculitis is often considered in the differential diagnosis of IBD. The association of TA with IBD suggests speculation on a common pathophysiology and could be explained by a cross-reactivity between autoantigens in the arterial wall and colonic mucosa. On the other hand, impaired perfusion of blood to the bowel due to TA may lead to ischemic colitis.

In the present case, she was followed up and also was treated with the misdiagnosis of UC for one year and the bowel symptoms did not associated with co-incidence of UC and TA. After endoscopic and histopathologic examinations, the diagnosis of UC was excluded.

In conclusion the clinicians should be kept in mind TA in the differential diagnosis of indeterminate colitis especially in younger women with high ESR.

References