Coexistence of Takayasu’s arteritis, ulcerative colitis, and stroke: A letter to the editor

Sir,

A 35-year-old right handed woman brought to the emergency department of Bu-Ali Sina Hospital, Sari, Iran, with sudden seizure, right hemiparesis, and dysarthria. During this acute phase the patient had a significant tremor and was frequently rolling on the floor and screaming. At the time of the first examination, she was found to have facial paresis, Broca’s aphasia, and right hemiplegia. About 2.5 years ago she called in a doctor with chief complaint of bloody defecation and tenesmus. A colonoscopy showed decreased vascular pattern and mucosal edema with submucosal hemorrhage. A biopsy was taken from ileum, cecum, and rectum of the patient. Pathologic results verified ulcerative colitis (UC) [Figure 1]. Asacol 800 mg tablets (4.8 g/day) prescribed for her. Treatment is ongoing and the patient does not have any digestive problem. In September 2010 she referred to neurology clinic with paresis in her upper limb. Also she had weight loss, back pain, vertigo, diplopia, blurred vision, and imbalance. At this time the patient had no digestive complaint. The patient was a case of asthma too that was confirmed by spirometry. Spirometry showed reduced forced expiratory volume (FEV)₁, FEV₁/forced vital capacity (FVC) ratio, and peak expiratory volume (PEF) suggesting of obstructive pattern. Her recent problems started 6 months ago. In the physical examination bilateral radial pulses of the upper limbs and distal pulses of lower limbs were different. In laboratory tests erythrocyte sedimentation rate (ESR) was 95 (normal limit (NL) = 0–30). She admitted with a diagnosis of Takayasu’s arteritis (TA) that was verified by color Doppler sonography and angiography. The upper limb had monophasic flow with decreased peak systolic velocity (PSV) in arterial color Doppler sonography. Proximal part of brachial and auxiliary arteries was occluded. Decrease of the lumen due to defuse wall thickness in large arteries especially common carotid and left brachiocephalic arteries were seen. Angiography showed complete obstruction in ascending aorta, right and left subclavian, and both sides of carotid arteries. Four months ago she was brought to our hospital for sudden convulsion with decreased consciousness. Brain MRI showed an ischemic cerebrovascular accident in the occipital part [Figure 2]. She had several experience of convulsions from that time until now. Now he is bedridden for the next stroke. She has a past history of anemia from 2 years ago, but does not have any other risk factors like: Hypertension, hyperlipidemia, diabetes, and positive family history of cardiovascular diseases. On admission she was not febrile in physical examinations. Blood pressure at brachial artery not detected on both right and left sides. Pulse rate of the right hand was 92/min, but the left side was so weak. Right and left dorsalis pedis pulses were well palpable and symmetric. Auscultation of the heart, lung, and also carotid and subclavian arteries were normal. In neurological examinations, she has Broca’s aphasia, facial paresis, and right hemiplegia. Babinski reflex was positive in right foot. Sensory evaluations of right side of the body are impaired. Laboratory data were as

Figure 1: (Low power field) Cecal mucosa reveals surface and glandular epithelium with marked polymorphonuclear leukocyte (PMN) exocytosis resulted to crypt abscess, goblet cell depletion, and reparative changes. The congested lamina propria shows severe lymphocytic, moderate neutrophilic and eosinophilic infiltration with foci of lymphocytic aggregation and lymphoid follicle formation

Figure 2: Brain computed tomography (CT) scan shows infarction in right occipital lobe
follows: White blood cell count 14,200/mm³ (lymphocytes 25%, monocytes 2%, neutrophils 73%), red blood cell count 436 × 10⁶/mm³, hemoglobin 12.5 g/dl, platelet count 455 × 10⁹/mm³, erythrocyte sedimentation rate (ESR) 120, and C-reactive protein (CRP) 102.3. Prothrombin time was within normal limit. Immunological evaluations revealed CH50 98 U/ml, C3 181.75 mg/dl, C4 32.4 mg/dl, antinuclear antibody (enzyme-linked immunosorbent assay (ELISA)) 4.1 U/ml and anti-double stranded deoxyribonucleic acid (dsDNA) 9.6 IU/ml. Urine analysis and stool examination were normal. In radiology examinations, chest X-ray was normal. Magnetic resonance imaging (MRI) showed occipital infarction as a signal intensity changing at the right occipital lobe and subcortical region with gyral pattern. Finally in the management of patients with TA, some other inflammatory diseases such as UC should be considered and vice versa.[1,2] And also perform more diagnostic evaluations to prevent progression of disease toward cerebral vessel involvement and dangerous complications such as stroke.[1,4]

ACKNOWLEDGEMENT

We really thank Bu-Ali Sina pathology staff members for their help. And acknowledge with grateful appreciation the kind assistance Mrs Mina Rostami for her help in editing the manuscript.

REFERENCES


Ebrahim Khoshnama¹, Mehrdad Taghipour², Rayka Sharifian³, Omid Emadian Saravi⁴

¹Assistant Professor of Neurology, Department of Neurology, Mazandaran University of Medical Sciences, Sari, Iran,
²Baqiyatallah University of Medical Sciences, Tehran, Iran,
³Mazandaran University of Medical Sciences, Sari, Iran
⁴Assistant Professor of Pathology, Mazandaran University of Medical Sciences, Sari, Iran

Address for correspondence: Dr. Mehrdad Taghipour, Nephrology and Urology Research Center, Baqiyatallah University of Medical Sciences, Tehran, Iran (021) 8248 3252. E-mail: mehrdadtaghipour@gmail.com