Case Report

Simultaneous Multiple Cerebral and Systemic Embolisms: A Case Report

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Abstract

Atrial fibrillation (AF) is one of the most common causes of cardiac embolism, but cerebral and peripheral embolisms can be also observed in approximately 30% of the patients with AF. Simultaneously encountered multiple embolism due to AF is a relatively rare condition. The aim of this study is to present a case with paroxysmal AF who had simultaneous renal, pulmonary and bilateral anterior cerebral embolisms.

Keywords: Atrial fibrillation, bilateral anterior cerebral infarct, renal infarct, pulmonary infarct

INTRODUCTION

Infarction of the anterior cerebral artery (ACA) territory accounts for only 0.3-4.4% of all cerebral infarctions[7]. Bilateral infarction is even relatively rare. Bilateral ACA infarction is usually caused by vasospasm after subarachnoid hemorrhage due to rupture of the aneurysm. On the other hand, atrial fibrillation (AF) is associated with an important risk of systemic embolisms and hypercoagulability[8]. Especially patients with chronic AF have higher risk for thromboembolism compared with those with paroxysmal AF[4,9,11,12]. The incidence of thromboembolism in patients with AF is about 4% to 7.5% per year[10]. Also existence of additional risk factors could increase the risk of thromboembolic events in patients with AF[3]. But simultaneously multiple embolisms due to AF including with other risk factors are relatively rare.

The aim of this report is to present a patient with paroxysmal AF who had simultaneously renal, pulmonary and bilateral anterior cerebral artery embolisms.

CASE PRESENTATION

A 61-year-old man with hypertension was admitted with a history of unconsciousness for three days. At the time of admission his blood pressure was 140/100mmHg. He was a heavy smoker and alcoholic for 30...
years. Family history was unremarkable. On admission he was unconscious but his vital signs were stable. His neurological examination revealed global aphasia, loss of swallowing reflex. Muscle strength was 4/5 in both upper and 1/5 in lower extremities. Babinski reflex was bilaterally positive and deep tendon reflexes were globally decreased. Sensorial and cerebellar examination could not be performed. A cranial magnetic resonance imaging (MRI) showed acute bilateral anterior cerebral arterial (ACA) infarction with hemorrhagic components and subacute right middle cerebral artery (MCA) infarction areas (Picture 1A, 1B).

Detailed etiological investigations were planned on this patient diagnosed with ischemic stroke. Among all laboratory investigations, white blood cells were 21600/mm³, sedimentation 52 mm/h, urea 91 mg/dl (20-50), creatine 1.19 mg/dl (0.6-1.5), creatin phosphohinase (CPK) 1156 U/L (0-190), alkaline phosphatase 170 (25-100 IU/L), C-reactive protein (CRP) 153.9 mg/l (0-5), glutamic-oxaloacetic transaminase (SGOT) 88 U/L (5-37), lactate dehydrogenase (LDH) 3237 U/L (220-450), gama glutamil transpherase (GGT) 69 U/L (7-49) and D dimer was 728 mic/L (50-228). Mild proteinuria and hematuria was also seen. Secondary causes of cerebrovascular diseases in young adults such as hypercoagulability and vasculitis were searched for and all markers were resulted to be normal.

The patient was consulted with cardiology, respiratory diseases and internal medicine departments. Thorax, abdominal and pelvic CT were performed. Thorax CT revealed passive atelectatic changes in lower posterior lobe of the right lung, consolidation and hypodense areas in the right lower lobe, ground-grass appearance in upper lobe anterior segment of the right lung, right pleural effusion, milimetric nodular lesions in the right lung and right descending pulmonary artery was noticed as hypodence. Abdominal and pelvic CT showed right renal arterial embolism (Picture 2). All investigations revealed that the patient had renal, pulmonary and bilaterally cerebral embolisms.

Picture 1 A, B: Bilateral anterior cerebral artery and right middle cerebral artery infarctions
Cranial MR angiography showed occlusion starting from the A1 segment of bilateral ACA and mural irregularity in M 2 segment of the right MCA (Picture 3A, 3B). Cerebral angiography revealed no anatomical or morphologic variations in the bilateral ACA territory and circle of Willis.

Electrocardiogram (ECG) revealed right ventricular hypertrophy and negative T waves at the time of admission, but AF was observed on the seventh day of the hospitalization. Detailed history taken from the family revealed that he had paroxysmal AF for 10 years.

Neuro-Doppler ultrasonography (USG) showed atheromatous plaque leading to stenosis of 50% in the left ICA. Lower extremity venous Doppler USG was normal. Transthoracic and transoesophageal echocardiogram were all normal.

Renal s Burligraphy showed renal function of 8% in the right kidney and 92% in the left.

All detailed investigations were performed to detect the etiology of multiple systemic infarctions, and the clinical picture was attributed to paroxysmal AF with the addition of other risk factors such as, hypertension, alcoholism and cigarette smoking. Anticoagulation was started in the ninth day of the hospitalization.

Following next days the patient was noticed to be more alert. His speech and cognitive functions were gradually improved. Muscle weakness evaluated in the tenth day was 2/5 in the lower and 5/5 in the upper extremities. He started oral feeding. 15 days after the hospitalization his treatment protocol was arranged and he was sent to a physical therapy and rehabilitation hospital.
DISCUSSION
Infarction of the ACA territory accounts for 0.3-4.4% of all cerebral infarctions. According to Bogousslavsky et al. 63% of ACA territory infarctions result from cardiogenic embolism or artery-to-artery embolism\(^1\). Bilateral ACA infarction is rare. Bogousslavsky et al. reported 27 cases of ACA territory infarction among 1,490 cases of cerebral infarction; however, there were only two cases of bilateral involvement. Kumral et al. reported two cases of bilateral ACA infarction among 48 cases\(^5\).

Kumral et al. emphasized that the main risk factors of ACA infarcts were hypertension in 58% of patients, diabetes mellitus in 29%, hypercholesterolemia in 25%, cigarette smoking in 19%, atrial fibrillation in 19%, and myocardial infarct in 6%\(^5\).

Atrial fibrillation is a strong risk factor for systemic embolism and hypercoagulability\(^9\).

Infarction of renal arteries is relatively rare. But if present, one of the most probable causes that can be accused should be cardiac embolisms (atrial fibrillation, cardiac valve disease, etc.). Gasparini et al. stated that 55% of all the renal infarcts originate from AF\(^2\). In our patient, the renal infarction was found to be due to paroxysmal AF. Renal infarctions are rarely accompanying the cerebral embolisms. Neurologists for this reason did not order detailed tests routinely. Deep vein thrombosis (DVT) is a well-known complication of stroke and frequently develops in acute stroke patients. Immobility in stroke patients increases the risk of DVT and pulmonary embolism\(^6\). Therefore early anticoagulation therapy is important to prevent complications.

In conclusion; an unusual case was presented here since he had simultaneous multiple embolisms. Detailed history and further studies are necessary to identify and prevent complications that may increase mortality.

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