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Case report

Renal Artery Thrombosis Due to Paroxysmal Atrial Fibrillation in a Patient with Multiple Thrombosis History

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Abstract

Atrial fibrillation (AF) is the most common persistent cardiac arrhythmia. When left untreated, atrial fibrillation often causes systemic embolization. We present a case of multiple thrombosis possibly associated with paroxysmal atrial fibrillation presenting with abdominal pain and renal artery thrombosis.

Keywords: renal artery thrombosis, atrial fibrillation, embolization

Introduction

Atrial fibrillation is the most common persistent cardiac arrhythmia and the lifetime prevalence is approximately 25% [1]. Atrial fibrillation is associated with a 3 to 4-fold increased risk of ischemic stroke and a 2-fold increased risk of death [2]. Known risk factors for atrial fibrillation include advanced age and age-related diseases such as hypertension, myocardial infarction and heart valve disease [3]. It is expected that the prevalence of atrial fibrillation will increase about 2 times in the 2010-2060 period, following the ongoing demographic change and improvement in survival in western societies, as well as the predisposition to heart diseases such as myocardial infarction [4]. Atrial fibrillation is an important public health problem and it is increasingly important to determine the risk of cardiovascular complications targeting preventive strategies [5]. Atrial fibrillation or flutter (AFF) creates an intra-atrial stasis with the potential to cause thrombus formation and embolization [6].

AFF has been associated with increased risk of other arterial and venous events beyond increasing ischemic stroke risk [7]. The patient was an 81-year-old woman with a past medical history remarkable for myocardial infarction, cerebrovascular disease and mesenteric ischemia who was referred to the Internal Medicine Clinic with a chief complaint of worsening abdominal pain.

Case Report

An 81-year-old female patient was admitted to the emergency room due to abdominal pain which started 3-4 days before, was not associated with food, and became increasingly severe. The patient was transferred to the Internal Medicine Clinic for workup of severe abdominal pain. The patient's past medical history was significant for an operation history associated with mesenteric ischemia, myocardial infarction, cerebrovascular disease. She was also treated with clopidogrel. On physical examination, the general condition of the patient was moderate, conscious open, cooperative. The patient was oriented to time and place. Vital signs revealed a blood pressure of 110/70 mmHg, a regular pulse of 76 bpm and a temperature of 36.2°C. The abdomen was non-tender. ECG (electrocardiography) showed normal sinus rhythm. The patient's admission labs were significant for creatinine of 1.1 mg/dl, hemoglobin of 13 gr/dl, white blood cell count of 18,000 mm3 and LDH of 1051 ul. The rest of the lab tests were as follows: PLT: 264.000/ul, urea: 26 mg/dl, creatinine: 1.1 mg/dl, glucose: 122 mg/dl, Na: 140 mmol/l, K: 3.86 mmol/l, Ca:9.3 mg/dl, AST:30 U/l, ALT:17 U/l, lipase: 16 U/l. Routine urine analysis was normal. An echocardiogram was performed that reveale normal chamber sizes. Defense and rebound findings were not detected. Right costovertebral region was slightly sensitive on palpation. Abdominal ultrasonography scan was normal. Abdominal angio-CT (computed tomography angiography) was performed for abdominal pain etiology. In angio-CT, hypoperfusion was found in more than 50% of the right kidney, and the distal end was found to be thrombosed when the right main renal artery was open (Figure 1). SMA (superior mesenteric artery) was detected in a thrombosed appearance after 3 cm from the initial segment. The splenic artery was open but the spleen had

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an infarct area secondary to distal microthrombosis. Celiac truncus, IMA (inferior mesenteric artery) and left renal artery were patent and no stenosis was detected. The patient with renal artery thrombosis was admitted

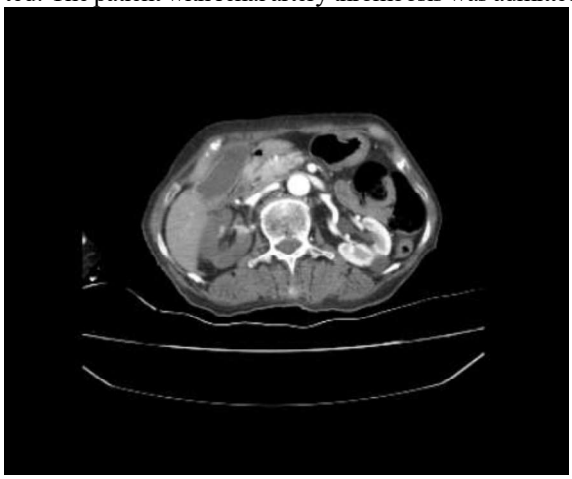

Fig. 1. Distal end thrombosis of right renal artery and hypoperfusion of right kidney

to the Internal Medicine Clinic for examination and treatment of the etiology of multiple thrombosis. A consultation was requested from the Interventional Radiology Clinic for thrombi formed in the renal arteries and branches. As the first 24-hour time limit for intra-arterial thrombolytic therapy was exceeded, no intervention was considered for the patient. Low-molecular-weight heparin therapy was initiated with clopidogrel therapy discontinued. PNH (paroxysmal nocturnal hemoglobinuria) panel, autoimmune markers and lupus anticoagulants, tumor markers for malignancy and imaging studies were requested for pathologies that may produce thrombosis susceptibility. No pathology was found in the requested examinations. Medical Genetics Department was consulted for clopidogrel insensitivity. Blood sample was sent for *CYP2C19* genotyping, which is responsible for clopidogrel metabolism. No evidence of clopidogrel resistance was detected in the gene analysis. A 24-hour ECG monitor (Holter ECG) was performed in the patient without documented cardiac arrhythmia, in terms of paroxysmal atrial fibrillation (PAF). Frequent atrial and ventricular premature beats and atrial fibrillation was detected in 2 times in Holter ECG. The patient was discharged after direct-acting oral anticoagulant (Dabigatran 110 mg 2x1) was initiated for PAF.

Discussion

The patient's past medical history did not include any cardiac rhythm disturbances. Although there were no known arrhythmia and documented AF in our case, 2 times AF was detected in the Holter ECG. AFF is associated with increased myocardial infarction, peripheral embolism, hemorrhagic stroke, and venous thromboembolism within 1 year, especially during the first 30 days after AFF, as well as being a risk factor for ischemic stroke [8]. The incidence of peripheral thromboembolic events associated with AFF is lower than the incidence of cerebrovascular thromboembolic events [8]. This is thought to be due to the fact that the majority of cerebral arteries are functional end-arteries, whereas many other arterial occlusions are protected by collateral circulation [9]. In our patient with renal artery thrombosis who had multiple thrombosis, no pathology was detected in terms of hypercoagulability, hematologic, oncologic and rheumatologic parameters and examinations which may cause thrombosis tendency. Cardiac causes were investigated in terms of thrombosis etiology, and echocardiography and ECG showed no pathological findings. In 1 of 20 acute stroke patients, AF was defined with systemic ECG monitoring [10]. This rate is much greater than that established by standard 12-lead electrocardiography (ECG) recordings. AF may remain undiagnosed for a long period of time (silent AF) and many patients with AF are not admitted to the hospital [10]. For this reason, the true prevalence of AF is likely to be close to 2% of the population [10]. Despite the absence of known arrhythmia and documented AF in our patient, two episodes of AF in the Holter ECG were detected. AFF is associated with an increased risk of developing cerebrovascular and peripheral thrombosis. As in our case, patients with multiple and recurrent thromboses should be evaluated with a Holter ECG and PAF should be kept in mind even if AFF is not detected with standard ECG. The differential diagnosis is expansive; however the presentation contains several clues to help focus the clinician's further evaluation. This case illustrates the importance and the value of Holter ECG in the evaluation of a patient with multiple and recurrent thrombosis. The patient manifests as a collection of findings highly suggestive of renal artery thrombosis due to paroxysmal atrial fibrillation.

Conflict of interest statement. None declared.

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