Probable Left Atrial Myxoma Presenting as Concurrent Cerebral and Myocardial Infarctions

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ABSTRACT

Concurrent cerebral and coronary artery embolization is a theoretically possible, but extremely rare complication of an atrial myxoma. We present a patient with a left atrial mass (a probable myxoma) who presented with concurrent cerebral and myocardial infarctions due to emboli of tumor origin. An 84-year-old woman presented with an acute cerebral infarction of the middle cerebral artery territory. Several hours after admission, she complained of chest pain consistent with a myocardial infarction. Transthoracic and transesophageal echocardiographic studies revealed the presence of a large, mobile, heteroechoic mass with a few daughter nodules in the left atrium, compatible with a myxoma. Coronary angiography disclosed subtotal occlusion of the ramus intermedius branch and visible tumor vascularization adjacent to the right coronary artery. With medical treatment, including anticoagulation, the patient was stabilized and had an uneventful clinical course for the ensuing 6 months since discharge. (Korean Circ J 2008;38:622-626)

KEY WORDS: Myxoma; Myocardial infarction; Cerebral infarction.

Introduction

The most common primary tumor of the heart is an atrial myxoma, which may cause symptoms of left atrial outflow obstruction and systemic embolization. Concurrent coronary and cerebral artery embolism is an extremely rare, but theoretically possible complication of left atrial (LA) myxoma. We report a patient with a left atrial mass (a probable myxoma) who presented with concurrent cerebral infarction in the left middle cerebral artery (MCA) territory and a non-ST segment elevation myocardial infarction.

Case

An 84-year-old woman was admitted to the Emergency Department of our institution with a 2-hour history of sudden dysarthria and right side weakness. Her risk factors for atherosclerosis were hypertension and hypercholesterolemia. Several hours after admission to the Neurology Department, she complained of substernal chest pain and dyspnea, while her right side weakness showed slight improvement. At the time that she complained of chest pain, the physical findings included a regular heart rate of 94 beats/minute, a blood pressure of 170/90 mmHg, and a body temperature of 36.9°C. Neither carotid bruits nor jugular venous engorgement were present. Thoracic auscultation revealed no cardiac murmurs, but inspiratory rales were audible in both lower lung fields. An electrocardiogram (ECG) revealed a normal sinus rhythm with intermittent ventricular premature complexes, and diffuse non-specific ST segment changes. There was no significant change compared with the initial ECG (Fig. 1). A chest X-ray showed mild cardiomegaly and mild pulmonary congestion. A brain MRI showed a left MCA infarction, but the brain MR angiogram findings were negative (Figs. 2A and 3B). The peak creatinine phosphokinase level was 492 U/L with 70 units of MB isoenzyme fraction present and the peak troponin-T level was 2.08 ng/mL. The total serum cholesterol level was 173 mg/dL and the low density lipoprotein (LDL)-cholesterol was 118 mg/dL. A trans-thoracic echocardiographic study revealed the presence of a large mass with heterogenic echogenicity in the...
left atrium, originating from the interatrial septum and oriented toward the posterior mitral leaflet, compatible with an atrial myxoma (Fig. 3A). The mass did not prolapse into the mitral valve orifice during diastole and the left ventricular systolic function and wall motion were normal. The patient was transferred to the Cardiology Department and was given aspirin, unfractionated heparin, an angiotensin receptor blocker (candesartan), a statin (rosuvastatin), and nicorandil. On the following day, transesophageal echocardiography (TEE) and coronary angiography were performed. The TEE showed a large, polypoid mass in the LA with several daughter nodules (Fig. 3B). The coronary angiography disclosed a subtotal occlusion of the small ramus intermedius (RI) with reduced flow, thrombolysis in myocardial infarction (TIMI) grade 2, and with thrombi-like haziness (Fig. 4). Tumor vascularization was visible adjacent to the right coronary artery (RCA) (Fig. 5). Percutaneous coronary intervention was not performed because of the small size of the culprit vessel.

Despite our suggestion to surgically remove the mass, the patient decided against open heart surgery. Medical treatment, including anticoagulation with warfarin, was instituted as the main treatment modality. The pathologic diagnosis of the presumptive myxoma could not be confirmed in the absence of surgery. On the basis of her echocardiographic and angiographic findings, we thus concluded that the patient’s final diagnosis was concurrent cerebral and myocardial infarctions, possibly caused by embolization from a LA myxoma. The uneventful
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A course of medical treatment with anticoagulation and the improvement of neurologic and cardiac symptoms resulted in the patient’s discharge on the 15th day after admission. At the last follow-up visit, 6 months after discharge, the patient was well and reported an uneventful clinical course.

Discussion

The most common presentation of a LA myxoma is dyspnea due to obstruction of the mitral valve, but can include dizziness, palpitations, syncope, and other non-specific constitutional symptoms. Symptoms of systemic embolism, the second frequent initial presentation of an atrial myxoma, have been variably reported in 16-40% of patients. Cerebral infarction is the most frequently observed embolic event in patients with cardiac myxomas.2-5 However, the occurrence of myocardial in-
fraction due to coronary embolization is rare and has been reported in only 0.006% of all cases. Panos and colleagues, noting the low coronary embolization rates, suggested two possible explanations: 1) the perpendicular disposition of the coronary ostia in relation to the aortic blood flow, thereby decreasing the possibility for an embolus to enter the coronary artery; and 2) during cardiac systole the coronary ostia are protected by the opening of the aortic valve leaflets.

Recently, there was an anecdotal case report of a patient who presented with multiple concurrent embolic events, including occlusion of the cerebral, ulnar, and popliteal arteries. However, because concurrent cerebral and coronary artery embolization is a very rare condition, there were only two cases in the worldwide previously reported literature.

Diagnosis of cardiac myxoma depends on a high index of suspicion and can almost be made by echocardiography; 90% originate from the LA, and the echocardiographic appearance is heterogenous with small lucencies. Two different morphologic types of myxomas have been determined by means of echocardiography: 1) the round type, which is solid and smooth with a non-mobile surface; and 2) the polypoid type, which is soft and irregular in shape with a mobile surface. Ha and coworkers showed that prolapsing and polypoid tumors were associated with a higher incidence of embolism. The more irregular and friable the myxomas were, the higher the likelihood was for emboli to form. In our case, the LA mass was polypoid in shape with a relatively irregular surface and a few daughter nodules, but it did not prolapse across the mitral valve orifice.

Since the first report by Marshall et al., there have been many reports concerning the coronary angioGraphic findings of cardiac myxomas, which have suggested that myxomas exhibit abnormally dilated atrial branches supplying the tumor, clusters of tortuous vessels, and contrast pooling or tumor blush on selective coronary angiography. Our patient’s angioGraphic findings also showed tortuous vessels supplying the tumor and tumor blush on right coronary angiography. Although we could not confirm the mass by pathologic evaluation, we judged the mass to be an atrial myxoma because its distinctive echocardiographic and angioGraphic findings were most compatible with a myxoma.

Thrombolytic therapy is not usually recommended for patients with embolic infarctions caused by cardiac myxomas because of the risk of embolism and hemorrhage. There are two possible explanations why thrombolytic agents cause embolic events: 1) the agents may cause lysis of an accumulated thrombus, and 2) in the presence of hemorrhagic areas and a rich vascular supply, thrombolysis could increase hemorrhage and cause rupture of small fragments. Although coronary artery myxomatous embolization is a rare condition which can be the cause of acute myocardial infarction, echocardiographic evaluation should be preceded before performing thrombolysis or urgent catheterization with intracoronary thrombolysis. If there is total or subtotal occlusion of the coronary artery, coronary angioplasty or coronary artery bypass surgery may be necessary. In our case, coronary angioplasty was not performed because of the small size of the culprit artery. In general, resection of the tumor is the only efficient treatment to ensure recovery of the patient; however, due to the patient’s decision to not undergo surgery, removal of the mass could not be performed.

Although concurrent cerebral and coronary artery embolization is a very rare condition, it should be considered in cases in which the emboli are of cardiac origin, such as myxomas of the heart. Prompt echocardiographic evaluation can ensure a swift and accurate diagnosis. Coronary angiography can help decide the therapeutical strategy for myocardial infarction and surgical resection is the treatment of choice for cardiac myxomas.

REFERENCES
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