The Heart Surgery Forum #2008-1015
11 (3), 2008 [Epub June 2008]
doi: 10.1532/HSF98.20081015

Online address: http://cardenjennings.metapress.com

Surgical Resection of Left Atrial Myxoma Presenting with Acute Multiple Hemorrhagic Cerebral Infarctions: A Case Report

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ABSTRACT

Brain ischemia resulting from left atrial myxoma embolization has been well documented. In contrast, the link between the development of intracerebral hemorrhage and myxoma in these patients has little coverage in the literature. The main theory describing this relationship stems from the fact that cardiac myxoma cells metastasize to the brain's vessels, causing destruction of the arterial wall with subsequent formation of fusiform aneurysm and further intracranial bleeding. It is assumed that when a diagnosis of left atrial myxoma with neurologic manifestations is made, surgical resection should be performed without delay to prevent repeated tumor embolization; however, systemic anticoagulation treatment during cardiac surgery with cardiopulmonary bypass is not recommended immediately after intracerebral hemorrhage occurs because of the possibility of extending the infarct's size. We describe a patient with acute hemorrhagic brain infarction and an echocardiographically demonstrated left atrial myxoma that was surgically resected successfully in the acute phase after the onset of the neurologic symptoms.

INTRODUCTION

Myxomas account for approximately 50% of primary neoplasms of the heart. Brain ischemia resulting from direct embolization of left atrial myxoma has been well documented [Burke 1993; Swartz 2006]. In contrast, intracerebral hemorrhage due to intracranial aneurysm in myxoma patients is a rare manifestation that has little coverage in the medical literature [Suzuki 1994; Herbst 2005; Sabolek 2005].

We describe a patient with acute hemorrhagic infarction due to 3 parenchymal intracerebral hemorrhages with a subarachnoid component. The echocardiogram showed a left atrial myxoma, which was surgically resected in the acute phase after the onset of neurologic symptoms. The pathogenetic mechanisms of intracranial bleeding in patients with myxoma and the dilemma of surgical treatment are discussed.

Received February 3, 2008; accepted March 25, 2008.

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CASE REPORT

A 59-year-old white woman was admitted to our hospital because of the sudden development of right-side hemiparesis, dysarthria, visual disturbances in the right eye, and dizziness. There was no family history of cerebrovascular disease or cardiac tumors.

A physical examination revealed normal vital signs. A cardiovascular examination revealed a grade 2/6 systolic murmur at the upper left sternal border and a late diastolic murmur at the apex. Peripheral pulses were normal.

The neurologic examination demonstrated the patient to be oriented. Observation of the cranial nerves showed right homonomous hemianopia, decreased visual acuity in the left eye (6/30), mild pallor of the left eye optic disc, and weakness in the lower part of the right facial musculature. A motor examination revealed right hemiparesis with 2/5 strength in the arm and 4/5 strength in the leg. The patient exhibited brisk, 3+ reflexes on the right side. The patient's coordination and sensory capacities were intact.

The results of routine laboratory studies, including a complete blood count, a urinalysis, and clotting factor and blood chemistry tests, were normal. The C-reactive protein level was increased to 3.6 mg/L. The results of a serologic test for syphilis were negative. An electrocardiogram and a chest radiograph showed no obvious abnormalities.

An initial computed tomography (CT) scan of the brain on admission demonstrated multiple foci (hyperdense and hypodense) in the left hemisphere, some of them with subarachnoid hemorrhages, as shown in the left frontal lobe (Figure 1A). Subsequent transthoracic and transesophageal echocardiographic evaluations demonstrated a large mass ($2.5 \times 2.5 \times 6$ cm) in the left atrium that had prolapsed through the mitral valve into the left ventricle.

A repeat CT scan 4 days later showed that the foci had become more prominent with a new hyperdense focus in the left frontal lobe (Figure 1B). We decided to perform

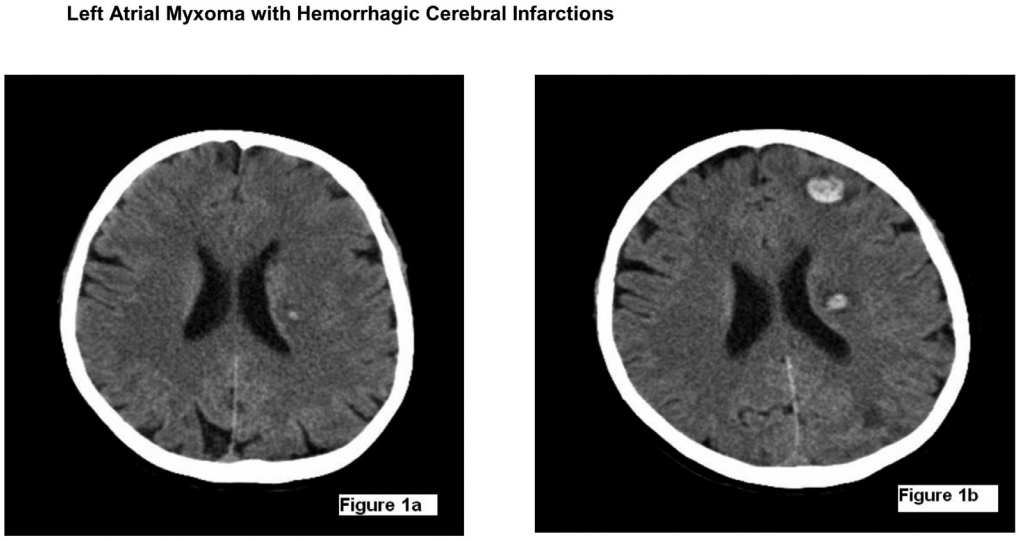

Figure 1. A, Computed tomography scan demonstrating multiple foci (hyperdense and hypodense) in the left hemisphere, some with minimal subarachnoid hemorrhage, as is shown in the left frontal lobe. B, A follow-up scan 4 days later revealed the foci to be more prominent with a new hyperdense focus in the left frontal lobe.

a resection of the cardiac myxoma and carried out this surgery 2 weeks later.

Surgical Technique

Surgery was carried out via a median sternotomy with standard cardiopulmonary bypass. Myocardial protection was achieved with antegrade and retrograde cold blood intermittent cardioplegia and moderate hypothermia (32°C). We used a modified biatrial approach. After making an incision into the right atrium and septum, we opened the dome of the left atrium. The incision was then enlarged caudally to expose the tumor. We resected the full-thickness interatrial septum and the myxoma and reconstructed the septum with autologous pericardium. The postoperative course was uneventful, and the patient was discharged 6 days after surgery.

Pathologic Findings

The removed left atrial myxoma was 6 × 4 × 3 cm. The gross appearance was typical of a myxoma; histologically, the tumor was composed of isolated spindle and stellate cells with a small amount of eosinophilic cytoplasm.

Follow-up

At the 1-year follow-up, the patient had recovered a good portion of her motor power and remained functionally independent. A repeat CT scan showed no evidence of further intracerebral bleeding.

DISCUSSION

Pathogenesis

The pathogenesis of intracerebral hemorrhage in myxoma patients is not fully understood; however, all theories support the opinion that cardiac myxoma cells metastasize to the brain's vessels with subsequent cell growth in situ and destruction of the arterial wall with aneurysm formation and further bleeding. Several mechanisms have been described for the formation of an arterial aneurysm. One of the most likely is that particulates from the tumor mass embolize into the vasa vasorum of cerebral arteries with subsequent weakening of the subintimal tissue of the vessel wall [Suzuki 1994; Furuya 1995; Sabolek 2005]. Other authors believe that perivascular damage to cerebral vessels may be due to their occlusion by tumor emboli and to endothelial scarring [Sabolek 2005]; however, this hypothesis contradicts the fact that most reported aneurysms occur in the absence of a history of cerebrovascular embolization [Herbst 2005]. Another proposed mechanism is the direct transendothelial invasion of tumor cells into the arterial wall, causing destruction of its architecture and subsequent aneurysm dilatation [Furuya 1995; Herbst 2005]. Evidence for this process can be seen in the pathologic report for a biopsied aneurysm, which revealed myxoid tissue occupying the lumen of the vessel, with invasion of the endothelium and disruption of the internal elastic lamina [Furuya 1995]. Because of the slow growth of myxomatous tissue in situ and the slow destruction of the arterial wall tissue, there is a delay between embolism of the tumor material and the destruction of the vessel wall with aneurysm formation [Sabolek 2005]. This growth can proceed even after the intracardiac mass has been removed [Suzuki 1994].

Surgical Resection of Myxoma in Acute Hemorrhagic Cerebral Infarction

To our knowledge, this report is the second of a successful surgically resected left atrial myxoma in the acute phase of hemorrhagic cerebral infarction [Yamamoto 2007]. It is clear that when a diagnosis of left atrial myxoma with intracranial

aneurysms and cerebral hemorrhage is made, surgery should be performed without delay to prevent further tumor embolization. On the other hand, systemic anticoagulation treatment during cardiac surgery with cardiopulmonary bypass is not recommended after a hemorrhagic brain infarction because of the risk of extending the infarct's size with a possible secondary intracranial hemorrhage [Zisbrod 1987; Bays 2004].

The appropriate timing of a surgical intervention in patients with recent hemorrhagic stroke is a difficult problem and has not been established in the literature. Eishi et al [1995] reported that the rate of exacerbation of cerebral complications decreased to 10% in patients who underwent surgical treatment more than 15 days after they experienced a cerebral infarction. Some authors who have encountered similar situations have recommended the use of a heparin-bonded circuit with low-dose heparinization [Bays 2004].

The potential causes of further neurologic deterioration after a recent infarction are multifactorial and include the following: progression of the cerebral infarction and edema; loss of autoregulation of the intracranial blood flow, resulting in a "steal" phenomenon; and risk of a secondary cerebral hemorrhage [Zisbrod 1987]. A biatrial operative approach was performed to excise the tumor as nontraumatically and completely as possible, to limit manipulation, and to visualize all 4 heart chambers. To avoid spilling fragmented tumor material into the pulmonary artery or the mitral valve orifice, we covered these structures with sponges before excising the tumor and flushed the left heart cavities multiple times. The surgical margin of the excision included a wide base of atrial septum to prevent the tumor's recurrence.

CONCLUSION

Multiple hemorrhagic cerebral infarctions may be an initial manifestation of cardiac myxomas. Further neurologic deterioration can successfully be prevented by surgical intervention carried out early after a recent hemorrhagic stroke.

REFERENCES

Bays S, Nicholson E, Humphreys N, Parry A. 2004. Aortic root replacement after recent intracerebral hemorrhage. Eur J Cardiothorac Surg 26:863-5.

Burke AP, Virmani R. 1993. Cardiac myxoma: a clinicopathologic study. Am J Clin Pathol 100:671-80.

Eishi K, Kawazoe K, Kuriyama Y, Kitoh Y, Kawashima Y, Omae T. 1995. Surgical management of infective endocarditis associated with cerebral complications. J Thorac Cardiovasc Surg 110:1745-55.

Furuya K, Sasaki T, Yoshimoto Y, Okada Y, Fujimaki T, Kirino T. 1995. Histologically verified cerebral aneurysm formation secondary to embolism from cardiac myxoma: case report. J Neurosurg 83:170-3.

Herbst M, Wattjes MP, Urbach H, et al. 2005. Cerebral embolism from left atrial myxoma leading to cerebral and retinal aneurysms: a case report. AJNR Am J Neuroradiol 26:666-9.

Sabolek M, Bachus-Banaschak K, Bachus R, Arnold G, Storch A. 2005. Multiple cerebral aneurysms as delayed complication of left cardiac myxoma: a case report and review. Acta Neurol Scand 111:345-50.

Suzuki T, Nagai R, Yamazaki T, et al. 1994. Rapid growth of intracranial aneurysms secondary to cardiac myxoma. Neurology 44:570-1.

Swartz MF, Lutz CJ, Chandan VS, Chandan VS, Landas S, Fink GW. 2006. Atrial myxomas: pathologic types, tumor location, and presenting symptoms. J Card Surg 21:435-40.

Yamamoto S, Shichijo T, Yamane K, Terasaka K. 2007. Left atrial myxoma surgically resected in acute phase of hemorrhagic cerebral infarction: report of a case [in Japanese]. Kyobu Geka 60:237-41.

Zisbrod Z, Rose DM, Jacobowitz IJ, Kramer M, Acinapura AJ, Cunningham JN. 1987. Results of open heart surgery in patients with recent cardiogenic embolic stroke and central nervous system dysfunction. Circulation 76:V109-12.