Juvenile Ischemic Stroke Secondary to Cardiogenic Embolism: A Rare Case Report

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ABSTRACT

Myxomas, the most common primary cardiac tumors, are known as a source of cardiogenic emboli. The possibility of their early detection has made them of great importance for emergency medicines. Detection of the disease is probable at early stages using echocardiography and associate complications such as syncope, cerebral embolic ischemic strokes, and sudden death. We report experience of a rare case of juvenile acute stroke in a patient with cardiac myxoma affecting all cardiac chambers presenting to the emergency department. In young stroke patients with signs and symptoms compatible with cardiovascular involvement, cardiogenic emboli should be taken into consideration; early echocardiographic studies are highly recommended. Prompt myxoma resection is required in both asymptomatic and stroke patients in whom intravenous thrombolysis course has not been implemented due to any limitations.

Keywords: Cerebral embolic ischemic stroke, emergency department, emergency medicines, myxomas

Case Report

INTRODUCTION

Atrial myxomas account for nearly half of all cardiac tumors,[1] Mostly seen in the 3rd-6th decades of life, myxomas commonly originate from subendocardial mesenchymal cells of the left atrium. Signs of heart failure, including dyspnea and pulmonary edema, could accompany left atrial myxomas in addition to syncope, sudden death, or signs of systemic embolism in some cases. Additionally, in almost 50% of the cases in which cerebral arteries are affected by embolization of tumor particles or thrombotic material covered with tumor cells embolic ischemic stroke is dreaded,[2] In this report, we are describing a patient presented with a right-sided hemiparesis due to cerebral infarction. Further investigations including transthoracic echocardiography (TTE) detected a four chamber myxoma as a source of cardiogenic embolism.

CASE REPORT

Our patient was a 24-year-old male presented to the emergency department with the chief complaint of right-sided weakness

without any loss of consciousness. He reported sudden onset of weakness within the previous 24 h having occurred after exercise and followed by difficulty in speech. No further specific symptoms including nausea, vomiting, vertigo, chest pain, or headache were noted prior to the emergence of sudden weakness except for a complaint of recent dyspnea on exertion (DOE). In his medical history, we detected a period of posttraumatic seizure that had been controlled with carbamazepine; he had stopped the course of medication 3 months ago. Also, a suspicious history of rheumatic fever in childhood without any documented therapy or diagnosis was present.

In physical examination, he was alert and oriented with obvious aphasia as he was able to obey the commands yet unable to speak. His vital signs were as follows: BP = 110/70 mm Hg, PR = 70/min, RR = 16/min, BT = 37°C. In his respiratory and cardiovascular examination, there was no specific abnormal finding. His peripheral pulses were symmetric and normal. In central nervous system examination, he had a right-sided hemiplegia with muscle force of 2/5 in upper and lower extremities; right plantar reflex was extensor, while the left plantar reflex was flexor and his deep tendon reflexes were increased up to 3 +. Due to his lateralized signs, the spiral brain computed tomography (CT) scan was recommended and routine lab tests were checked simultaneously. In spiral brain CT, a hypodense lesion could be observed in the left middle cerebral artery (MCA) [Figure 1].

The lab tests results were as follows:

Erythrocyte sedimentation rate (ESR) = 47 mm/h, PT = 14.4 s, International normalized ratio (INR) = 1.33, PTT = 30.9 s, K = 4.5 mmol/L, Na = 141 mmol/L, Blood sugar (BS) = 94mg/dL, Blood urea nitrogen (BUN) = 13 mEq/dL, Cr = 1.06mg/dL, Creatine phosphokinase (CPK) = 116 U/L, Creatine phosphokinase-MB (CPK_{MB}) = 27U/L, lactate dehydrogenase (LDH) = 358 U/L, White blood cells (WBC) = 6700/mm^3 (Neut = 57%, Lymph = 40%, Mono = 3%, Eos = 0%, BASO = 0%, BAND = 0%.), Red blood cells (RBC) = 4.32 million/mm^3, hemoglobin (Hb) = 11.5g/dL, Hematocrit (Hct) = 35.5 g%, Platelet (PLT) = 221000/ mm^3.

The NIHSS of the patient was 14; however, no intravenous thrombolysis therapy was considered due to the delayed referral to our center (more than 3-4.5 h).

Due to his history of DOE and occurrence of his signs and symptoms exactly following exercise, echocardiography was suggested. Interestingly, echocardiography revealed multiple masses in all cardiac chambers [left atrium (LA) and Left ventricular (LV) (two separated), right ventricle (RV) and right atrium (RA) in order of largest to smallest, respectively] [Figure 2]. Findings were in favor of a multiple myxoma. On the basis of cardiology consultations, association of a multiple cardiac myxoma and probable cerebral emboli originating from it forced anticoagulant therapy because of high risk of hemorrhagia of

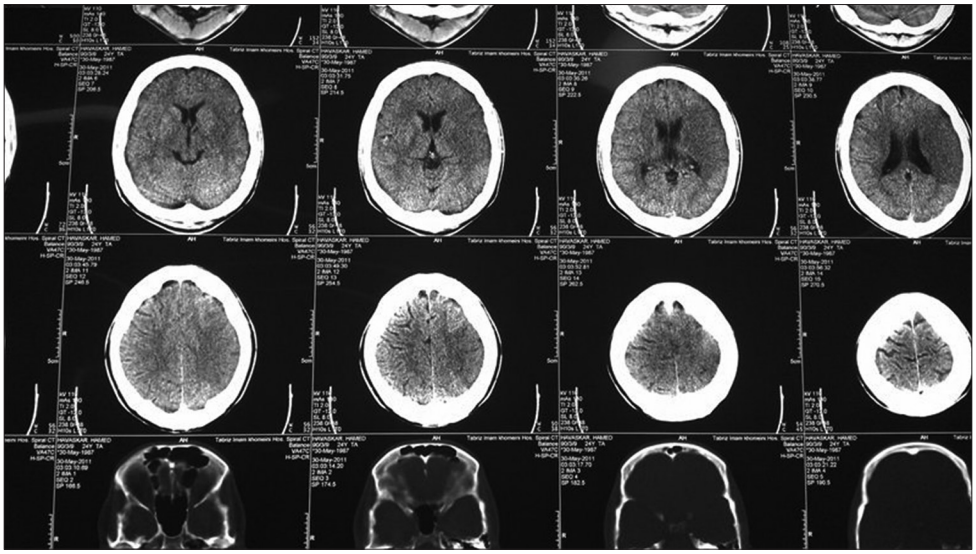

Figure 1: Patient's computed tomography scan in admission

lesion did not start. He was admitted at cardiac surgery intensive care unit and scheduled to undergo cardiac myxoma resection 2 days after his emergency department admission. The day following cardiac surgery, his hemiplegia resolved and he was ambulated at the time of discharge. Furthermore, muscle forces of the upper and lower limbs improved from 2/5 and 2/5 to 3/5 and 4/5 before and after operation, respectively. Postoperation imaging of the patient including brain CT scans and magnetic resonance imagings are presented in Figures 3-5. The follow-up CT scan revealed evidences of remained ischemic injury in the left MCA 2 months after operation [Figure 4].

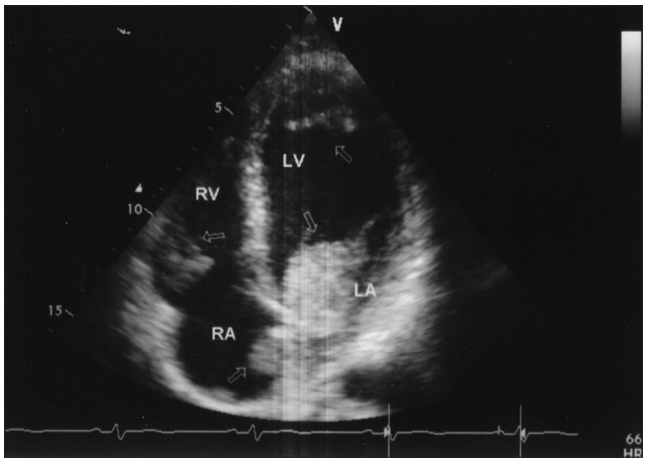

Figure 2: Patient's echocardiography in admission

DISCUSSION

Myxomas commonly affect the left atrium; however, right atrium, ventricles, or mitral valve could also be involved contributing to clinical manifestations including embolism, obstruction of the mitral valve, and general findings (i.e., fever, anemia, and elevated erythrocyte sedimentation rate). A set of diagnostic criteria have been recommended; differential diagnosis is difficult due to the fact that myxomas typically remain uninfected.[3] Cerebral infarction prompted by cardiogenic embolism is seen in almost 20% of stroke patients of whom atrial fibrillation accounts for over 50% of the cardiogenic emboli, while myxomas have been reported in only 0.5% of emboli and their consequent ischemic strokes.[4] Owing to the ability of producing instantaneous emboli, myxomas are able to become fatal despite being commonly asymptomatic. A median delay of 36 months has been reported between commencement of symptoms and diagnosis in

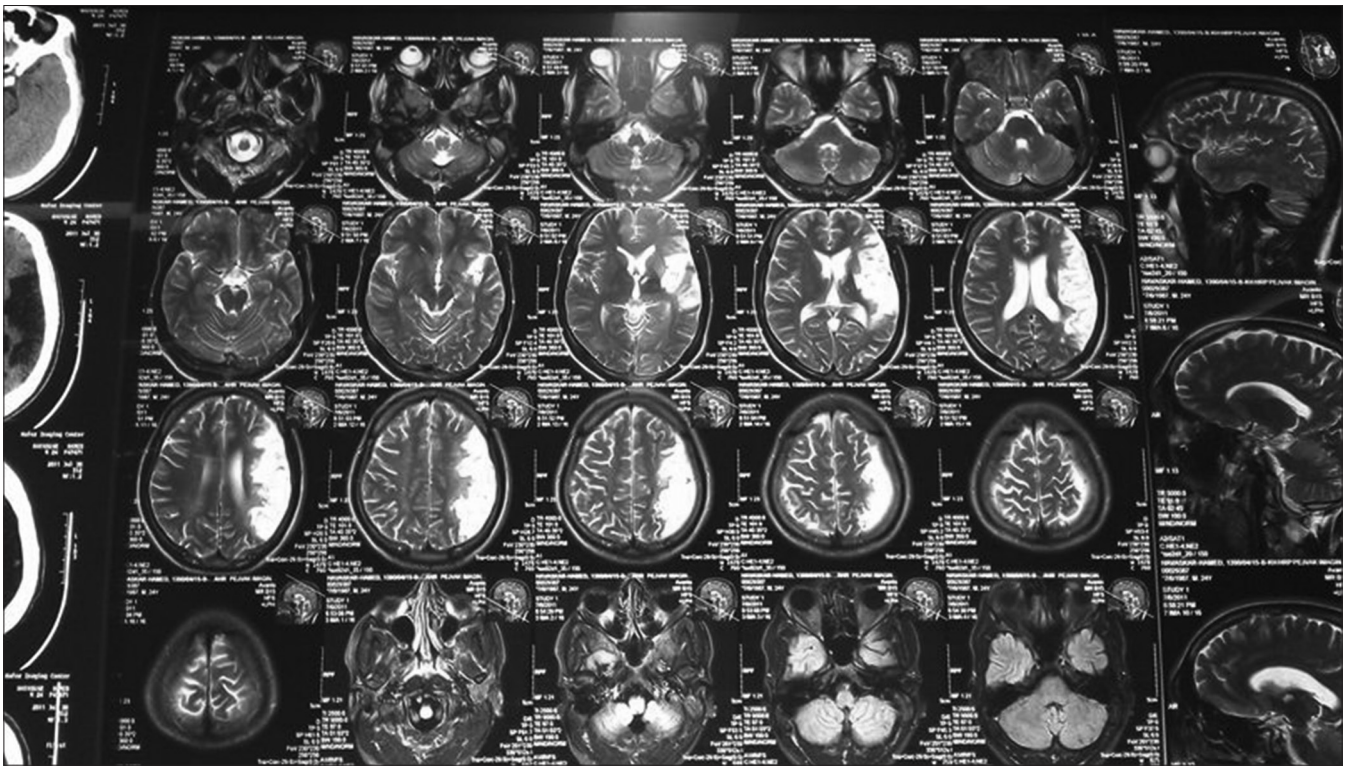

Figure 3: Patient's postoperation magnetic resonance imaging

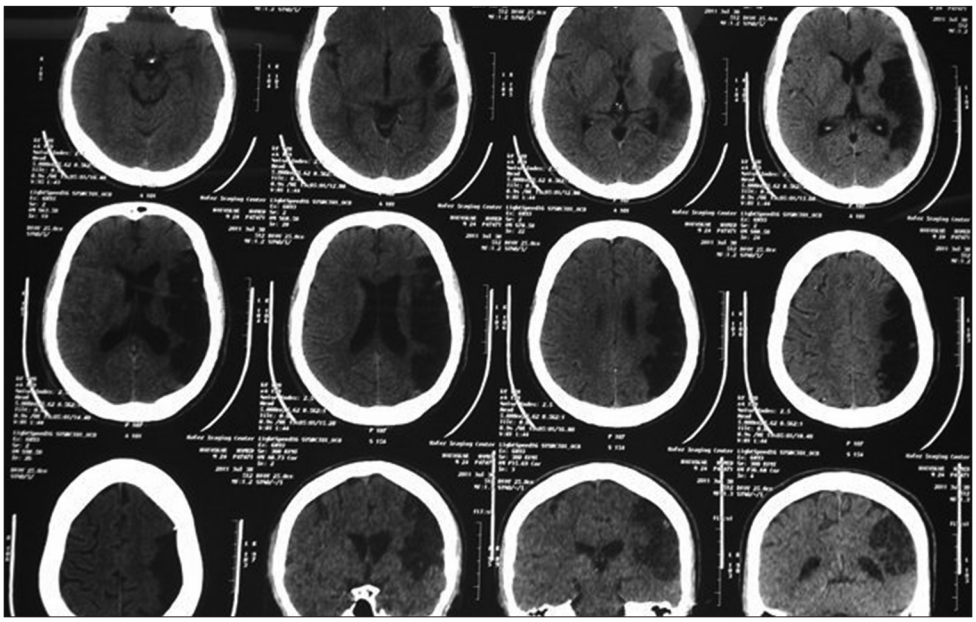

Figure 4: Patient's computed tomography scan in 2 months after admission

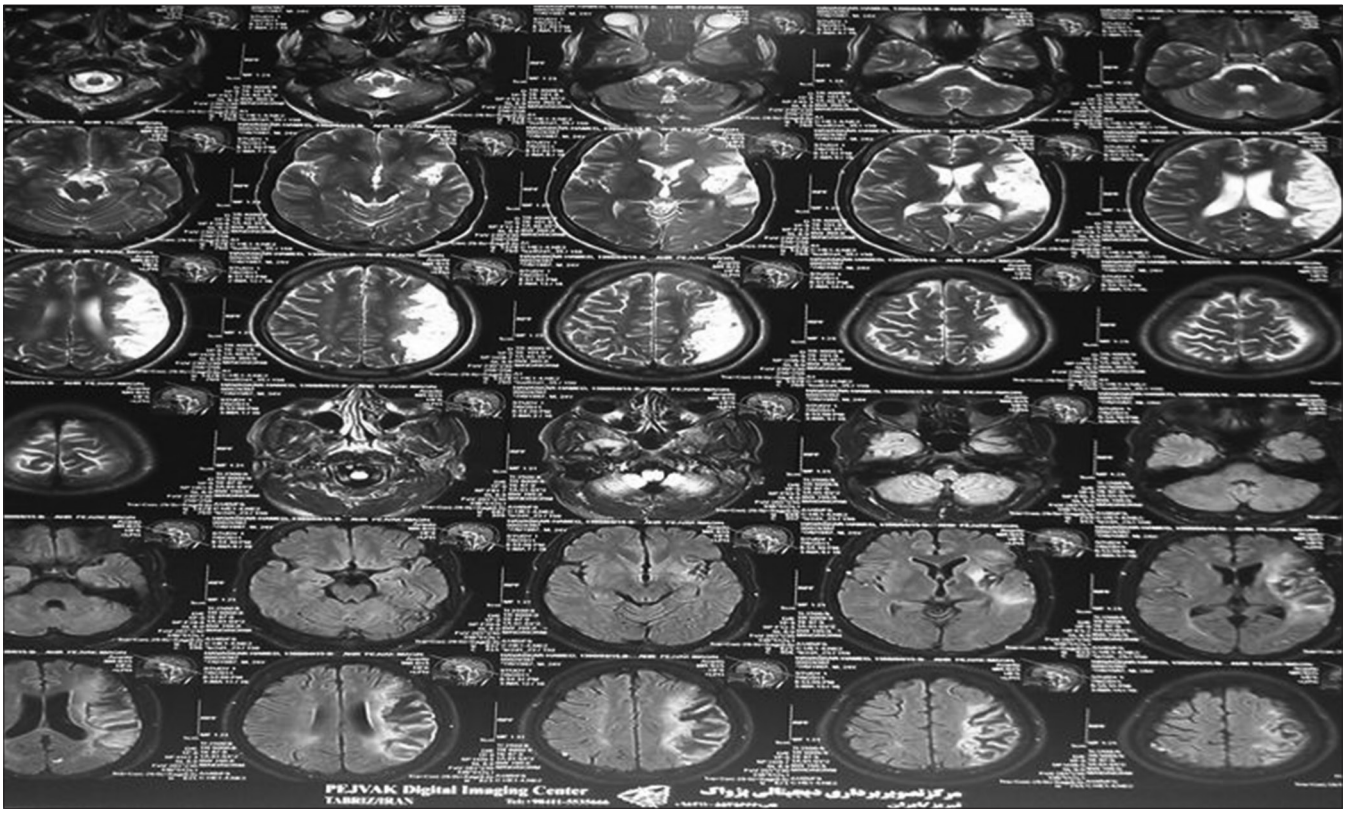

Figure 5: Patient's magnetic resonance imaging 12 months after admission

myxoma patients with neurologic manifestations.[5] The findings provided by cerebral imaging often include multiple infarcts being suggestive of a cause with an embolic origin; however, small subcortical ischemic lesions mimicking lacunar disease is occasionally reported as well.[6] Although, the sensitivity of transesophageal examination is higher, TTE with a sensitivity of almost 90% would serve as an excellent diagnostic tool in the detection of left atrial myxoma.[7] Myxomas might

be asymptomatic until they are bulky enough to either produces emboli or obstruct the mitral or tricuspid valves. Due to their intravascular and fragile nature, myxomas are known as the major cause of tumor emboli which occurs in almost 30%-40% of patients. The location of the myxoma (left or right atrium) and the presence of an intracardiac shunt are the main factors affecting the site of embolism.[3,5,8]

The emboli that occur are either a tumor fragment that is released from the myxoma or a blood clot that is formed on the surface of the myxoma. These subsequent emboli can result in infarction, as occurred in our patient. On the, the cerebral embolus was due to mural thrombosis mounted on the myxoma in our patient, fragmentation of the embolus into smaller particles downsizing the ischemic area might have contributed to the partial improvement and hemiparesis reduction. In addition, thrombosis is composed of blood elements primarily affecting larger and consequently smaller arteries following degradation which could result in symptom reduction.[9]

Almost half of the patients with myxomas have been reported to suffer neurologic manifestations having resulted from embolizations (pulmonary, myocardial, mesenteric, retinal artery occlusion, spinal cord ischemia, and stroke). MCA, having a dominant blood flow, is often and in myxoma cases with the suspected involvement of either frontal or parietal lobes, MCA proximity should be carefully probed.[8,10]

Diagnosis is frequently made using two-dimensional echocardiography which is considered the gold standard; however, TEE might be distinctive in equivocal or confusing findings. In addition to the detection of other cardioembolic sources such as a patent foramen ovale, mitral valve calcification, or aortic atherosclerosis; both TTE and TEE are able to clarify the location, size, shape, and movement of myxomas. Cardiac catheterization might be required especially if other concurrent cardiac diseases are assumed or in the presence of equivocal diagnostic findings.[11] In a literature review that we performed on the emergence of the myxoma resection, most authors agreed that early resection of a myxoma leading to stroke is highly recommended. However, the procedure should be postponed in cases which have undergone thrombolytic therapy which could increase the risk of hemorrhage. As in our case, no thrombolytic therapy course had been initiated due to the loss of 24 h golden time, the patient underwent prompt resection. Resection should be performed with negative margins due to the postoperative recurrence rate of 1%~3%.[12-15]

Hence, long-term follow-up with echocardiography is highly recommended in all patients.

TTE is used routinely in stroke patients by physicians in the search for cardiogenic embolic sources. However, myxomas might be missed in case of exclusive use of TTE especially in the obese.[7,11] Therefore, further supplementary diagnostic approaches and modalities should be taken into consideration in these patents.

CONCLUSIONS

The reasons for reporting this case are Rarity of myxoma, asymptomatic nature of the disease until CVA occurrence (except for a recent dyspnea following exercise), lack of any previous syncope history, and involvement of all four cardiac chambers. Emboli and their neurological complications occur frequently in patients with cardiac myxoma. Early and prompt cardiac evaluation using echocardiography in the emergency department seems reasonably necessary in order to detect the source of any probable stroke. As the main involved cerebral site, more accurate exploration of MCA is recommended. Finally, surgical resection of the myxoma in asymptomatic or stroke patients in whom intravenous thrombolysis course has not been implemented due to any limitations should be considered a priority and performed without any delay.

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