Microemboli monitoring by trans-cranial doppler in patient with acute cardioembolic stroke due to atrial myxoma

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Abstract

This is the first reported attempt to examine the emboliogenic potential of cardiac myxoma in patients with acute stroke through the monitoring of microembolic signals (MES) by transcranial doppler. A 43-year old woman was brought to the emergency department because of acute onset of generalized tonic-clonic seizures and left hemiplegia. A CT scan of the brain demonstrated a large acute infraction in the territory of the right middle cerebral artery (MCA) and another smaller one in the territory of the posterior cerebral artery on the same side. Trans-cranial doppler (TCD) microemboli monitoring did not reveal MES. Transesophagial echocardiography (TEE) identified a 5 cm left atrial mass, which was highly suspected to be an atrial myxoma attached to the interatrial septum and prolapsed through the mitral valve. After the TEE results were obtained, another TCD monitoring was performed. Again, there were no MES found in either of the MCAs.

Our findings showed the absence of MES on two consecutive TCD examinations, suggesting a spontaneous occurrence, rather than the permanent presence, of embolization, even in the most acute phase of stroke. Thus, the tendency of myxomas to spontaneously produce multiple emboli emphasizes the need for the surgical excision of myxomas.

Introduction

Left atrial myxoma is a benign primary cardiac tumor and a cause of embolic stroke. Primary tumors of the heart are rare and 75% of those tumors are benign. Nearly half of the benign heart tumors are myxomas, making it the most common heart tumor. Although classified as benign tumors, myxomas seem to grow and spread, but there is still debate as to whether they metastase or fragments simply embolize.[1] There is a 2:1 female preponderance and the age at onset is usually between 30-60 years. Although atrial myxoma is largely sporadic, at least 7% of cases are familial. Atrial myxomas have been estimated to cause up to 0.5% of ischemic stroke.[2] Stroke appears to be responsible for 80% of the neurological presentations of myxomas, and only 40% of these have a typical pattern of cardioembolism involving several vascular territories. Myxoma usually causes ischemic stroke by embolism of tumor or thrombus, but aneurismal dilations at sites of earlier embolic vascular occlusion can cause intracerebral or subarachnoid hemorrhage.[3]

Cerebral imaging often demonstrates multiple infracts suggestive of an embolic cause, but in some cases it may show only small subcortical ischemic lesions, as seen in lacunar infracts.[4] Transthoracic echocardiography has approximately 90% sensitivity in the detection of left atrial myxoma, while the sensitivity of transesophageal examination is 100%. Transesophageal examination is also preferred because of its ability to detect other cardioemoblic sources, such as intracardiac thrombus, vegetations, or aortic arch plaque.[5] General or constitutional manifestations, such as fatigue, fever, erythematous rash, arthralgia, myalgia, and weight loss, as well as laboratory abnormalities, such as anemia and elevated ESR and serum C reactive protein and globulin levels, have been observed in many patients irrespective of the site and size of the myxoma. The risk of recurrent myxoma is 1-3% for sporadic cases, and it is usually attributed to multifocal myxoma embolization or incomplete resection. Follow-up by echocardiography is recommended.[1]

It has not yet been established as to whether transcranial Doppler (TCD) can be used as a means to examine the emboliogenic potential of cardiac myxoma in patients with acute stroke.

Case Report

A 43-year old woman from the Philippines was brought to the emergency department of Rambam Medical Center in Haifa, Israel because of acute onset of generalized tonic-clonic seizures and left hemiplegia. The patient was generally healthy without any kind of treatment or risk factors. No history of previous transient neurological deficit was reported. On physical examination, she was found to be in a stupor. Her vital signs were normal, and cardiac exam revealed sinus rhythm without heart murmurs. Neurological examination showed left central facial palsy, eye deviation to the right, left hemiplegia, brisk deep tendon reflexes, and extensor plantar reflex on the left. A CT scan of the brain on admission revealed a small old right parietal lobe infraction. Another CT was performed 24 hours later and revealed a large acute infraction in the territory of the right middle cerebral artery (MCA) and another smaller one in the territory of the posterior cerebral artery on the same side. The erythrocyte sedimentation rate was 72 mm in the first hour with no anemia. Blood coagulation, lipid and homocysteine levels were normal. Treatment with phenytoin and plavix was started. Carotid duplex, including common, internal and external carotid arteries and vertebral arteries, was normal.

TCD (Pioneer, TC 8080, Viasys™, Nicolet) microemboli monitoring was performed for 30 minutes, with both MCAs monitored. No microembolic signals (MES) were revealed. Examination of cerebral blood flow velocities was normal. Cardiac holter showed a sinus rhythm average of 74 (62-119) without tachy or brady-arrhythmia or conduction disturbances. Transesophagial echocardiography (TEE) identified a 5 cm left atrial mass, which was highly suspected to be an atrial myxoma (Figure 1) attached to the interatrial septum and prolapsed through the mitral valve (without significant LV inflow obstruction in the supine position). After the TEE results were obtained, another TCD monitoring was performed and continued this time for one hour. Again, there were no MES found in either of the MCAs. The myxoma was resected 23 days after admission. Transthoracic echocardiography was performed three days after surgery and showed no evidence of residual myxoma or atrial septal defect. The patient was referred for further rehabilitation because of severe left hemisyndrome.

Discussion

One of the widely used applications of TCD

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Key words: myxoma, stroke, transcranial doppler.

Received for publication: 8 June 2009.
Revision received: 9 December 2009.
Accepted for publication: 20 January 2010.

Neurology International 2010; 2:e5
doi:10.4081/ni.2010.e5

is MES monitoring. This technique provides useful information about possible sources of embolism, as well as their activity. Acute ischemic stroke, carotid stenosis, and cardiac diseases are the most studied areas in the field of MES monitoring. The significance of MES in patients with cerebrovascular diseases has been the subject of many studies. The short Pubmed literature search by combination of MES and stroke retrieved 122 related items.

The first line of research is devoted to revealing the prevalence and timing of MES appearance in patients with acute ischemic stroke. It is now well established that MES is a frequent phenomenon in patients with ischemic stroke and that the amount of MES is related to stroke onset, with the peak occurring in the first hours and days after stroke.[6-10] Many other important studies have been devoted to the key issue of using the presence and amount of MES as predictors of the short- and long-term clinical outcomes during the index event, as well as possible predictors of recurrent ischemic stroke.[11-17] Studies exploring the correlation between prevalence and amount of MES and the etiology of ischemic stroke can also be found in the literature.[18-23]

Another line of research estimates the impact of MES in both symptomatic and asymptomatic carotid disease. In patients suffering from carotid stenosis, the presence of MES can be indicative of an unstable structure of carotid plaque that is prone to be a source of emboli. The existence of MES in patients with carotid disease is also an important indicator of the substantial danger of the first and recurrent cerebrovascular event.[24-29] Another area in which the presence of MES determines the increased risk of peri- and post-procedural complications is that of carotid surgery and stenting. In both cases, the appearance of a significant amount of MES reflects damage of the plaque, resulting in multiple emboli with neurological and cognitive impairment.[30-42]

In cardiology, TCD is a reliable technique for the detection of MES from cardiac embolic sources, such as prosthetic cardiac valves,[43-45] aortic plaques,[18,46-48] and atrial fibrillation.[20,23,49] It is also used as an especially sensitive technique for detecting right-to-left cardiac or pulmonary shunts.[50-56] The microembolic signals obtained from patients with artificial heart valves and during examinations for PFO detection have also been used as a model, alongside other methods, in an attempt to differentiate between gaseous and solid microemboli.[57-59] The results of MES monitoring may also be found in some other clinical situations, where their presence[60-65] or absence[66-67] helps us to better understand the role of embolism in the development of the disease. Our case emphasizes that MES monitoring may be useful not only in detecting the embolic nature of the disease, but also in exploring the precise timing of embolization (i.e., either permanent, as in the many clinical situations mentioned above, or sporadic, as in our case).

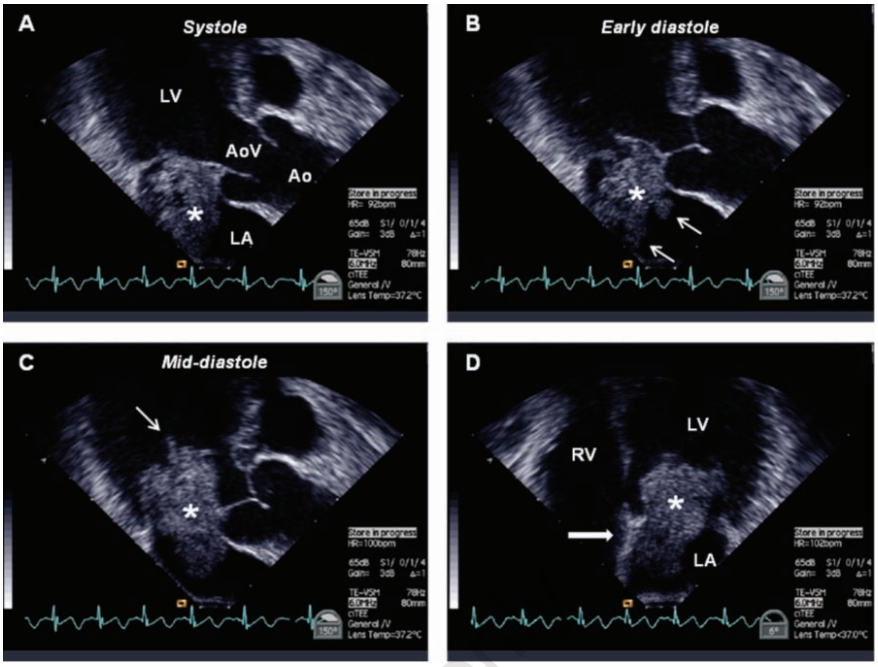

Figure 1. Multiple trans-esophageal images of a large left atrial myxoma (marked by *). Long-axis mid-esophageal views of the heart (A – systole, B – early diastole, C – mid-diastole) demonstrating prolapse of the myxoma from the left atrium into the left ventricle during diastole. Multiple, mobile, "finger-like" projections of the tumor were evident (thin arrows). Four-chamber mid-esophageal view (D) showing the connection of the tumor to the interatrial septum (thick arrow). LA = left atrium, LV = left ventricle, Ao = ascending aorta, AoV = aortic valve, RV = right ventricle.

Although cardiac myxoma is a rare disease, it should be considered in young patients with stroke and elevated ESR, even without systemic symptoms. There are no data in the literature about TCD monitoring in patients with atrial myxomas. Our findings showed the absence of MES on two consecutive TCD examinations, suggesting a spontaneous occurrence, rather than the permanent presence, of embolization, even in the most acute phase of stroke. These data are compatible with literature sources pointing to the simultaneous appearance of multiple emboli at different target sites in patients presenting with cardiac myxomas.[68] Another line of evidence supporting our data is the large number of silent lesions commonly found in patients with stroke and myxomas,[69] as in the case of the patient presented here. Thus, it seems that in many cases, despite silent embolic events, the patient may remain clinically intact for years until a symptomatic event occurs. The tendency of myxomas to spontaneously produce severe and multiple emboli emphasizes the need for the surgical excision of myxomas when possible anticoagulation seems to be doubtful.

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