A Case of Stroke due to Tumor Emboli Associated with Metastatic Cardiac Liposarcoma

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Abstract

Cardioembolic stroke due to tumor emboli is a rare complication of neoplasm. A patient with metastatic cardiac liposarcoma who suffered from embolic stroke is reported. Autopsy confirmed that the cardiac tumor was a metastatic liposarcoma from the retroperitoneum, and the cerebral vessel was occluded by tumor cells and fibrin clot.

Key words: tumor emboli, metastatic cardiac liposarcoma

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Introduction

The prevalence of metastatic heart and pericardial tumors was 14% in serial autopsy cases with cancer (1); malignant melanoma was the most frequent tumor, and sarcoma was relatively uncommon. In most of the cases, autopsy incidentally revealed the cardiac involvement, and clinical symptoms, such as dyspnea and palpitation, were noted in 15% of the patients with metastatic cardiac tumor (2); stroke did not occur frequently. Cerebral infarction and vessel occlusion causing the corresponding ischemia have been rarely confirmed (3). In the present case of metastatic cardiac liposarcoma, histopathological examination revealed that the cerebral vessel was occluded by tumor cells and fibrin clot.

Case Report

A previously well, 71-year-old man presented with a retroperitoneal mass in 2008. Excision of the mass along with the left kidney and spleen was performed, and histology confirmed dedifferentiated liposarcoma (Fig. 1A). Unfortunately, during the 10-month period after excision, he suffered recurrence locally in the left thigh and left shoulder, for which he received irradiation and chemotherapy. In September 2009, computed tomography incidentally showed a mass in the whole left ventricle that was completely asymptomatic (Fig. 1B).

Two months later in November, he was admitted to the emergency room with sudden onset of dysarthria and left hemiparesis. His blood pressure was 155/92 mmHg, and the pulse was regular with no pulsus paradoxus. Cardiac examination was normal except for a systolic ejection murmur. There was no marked jugular venous distention or edema of the extremities. Radial and dorsal pedis arteries were symmetrically palpable. We detected a painful mass in his left shoulder and left thigh. Neurological examination revealed disturbance of consciousness, dysarthria, and left hemiparesis. His National Institutes of Health Stroke scale was 6. Premature ventricular contraction on electrocardiography and cardiomegaly (cardiothoracic ratio: 63%) on chest X-ray were found. His laboratory data showed decreased platelet count of 5.5×10⁴ μL (normal range; 15-35×10⁴ μL) and elevated serum creatinine of 1.56 mg/dL (normal range; 0.6-1.2 mg/dL). D-dimer elevated into 20.8 μg/mL (normal range; <1.0 μg/mL). Head MRI using diffusion-weighted sequence revealed multiple hyper-intense lesions on bilateral cerebral hemisphere and cerebellum (Fig. 1C). An echocardiogram detected a huge movable mass occupying the left ventricle, collapse of the right ventricle, and pericardial effusion.
Twelve hours after admission, cardiac tamponade rapidly reached a severe state. On day 1, acute arterial obstruction of his left lower extremity occurred. Although peri-cardiac effusion was drained, he died of heart failure on day 3.

Autopsy revealed that the cardiac mass was metastatic dedifferentiated liposarcoma (Fig. 1D, 1E). There was tumor embolus to the brain of right precentral gyrus. The cardiac weight was 370 g and tumor size was 3×3×4 cm (Fig. 1D). The mass occupied the left ventricle and presented invasion into the papillary muscle. On the surface of the mass, a combination of fibrin and tumor was detected by microscopic observation. The microscopic findings on the tumor in the left ventricular cavity were quite similar to the with histological findings on previous resection of dedifferentiated liposarcoma, suggesting metastasis (Fig. 1A, 1E). Concerning recent infarct at right cerebral gyrus, the embolus occluded the cerebral artery supplying blood flow to the territory corresponding with the infarct region (Fig. 1F). Fibrin, inflammatory cells, and tumor cells were composed of the embolus. Patent foramen ovale was not detected. There was no tumor embolus in any organ including lungs.

**Figure 1.** Imaging and histological findings in a patient with multifocal stroke from tumor emboli. A, Dedifferentiated part of the primary region of retroperitoneum liposarcoma (Hematoxylin and Eosin staining, original magnification ×10). B, Chest computed tomography demonstrating a mass in the left ventricle. C, Head MRI demonstrating multiple hyper-intense lesions on bilateral cerebral hemisphere. D, Metastatic dedifferentiated liposarcoma (3×3×4 cm) in the left ventricular cavity. E, Histological findings of cardiac mass liposarcoma were in line with the primary region at retroperitoneum (Hematoxylin and Eosin staining, original magnification ×20). F, Cerebral artery occluded by tumor cells (white arrow), fibrin, and inflammatory cells (Hematoxylin and Eosin staining, original magnification ×40).

**Discussion**

We reported a case with metastatic cardiac liposarcoma who suffered from embolic stroke. Microscopic histology revealed that cerebral vessel was occluded by fibrin and tumor cells.

Cardioembolic stroke accompanied with a metastatic cardiac tumor seems to be quite rare; only 8 cases were previously described (4-9). The primary organ of metastatic tumor was lung in 2 cases and soft tissue in 3 cases. Regarding microscopic histology for cardioembolism due to metastatic tumor, we were able to find only 1 autopsy case reported by Navi et al (3). The histological findings of the present case were not in line with those of Navi’s case in that moderately differentiated squamous cell carcinoma but not fibrin clot occluded the cerebral artery (3). To the best of our knowledge, the present case is the first report that not only tumor cells but also a fibrin clot contributed to the stroke mechanism.

We were able to refer 17 cases with metastatic cardiac
liposarcoma confirmed by microscopic histology (10). Metastatic tumor was found at the right atrium and/or ventricle in 8 cases and on the left side in 8 cases. Only one case presented metastatic region at the interventricular septum. Among those reports, heart failure was the most frequently observed as a clinical symptom. There was no case with cardioembolic stroke after diagnosis as metastatic cardiac liposarcoma, except for the present case. The most common lesion for metastasis of liposarcoma was the lung (2). The present case did not have metastasis or tumor emboli in the lung.

We could elucidate novel information of stroke mechanism in a case with tumor brain emboli caused by metastatic cardiac liposarcoma. There has been little evidence for hyper-acute management for tumor emboli such as in our case. Our case had a fibrin clot in the occluded artery. Therefore thrombolysis as t-PA may be effective for recanalization of such an occluded artery.

The authors state that they have no Conflict of Interest (COI).

References


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