Cystic Tumors of the Right Atrium Caused by Nonbacterial Thrombotic Endocarditis in a Patient with Large Cell Carcinoma

Norikazu Sakamoto¹, Takashi Ueda², Katsufumi Mizushige³ and Mitsuo Maeda⁴

Key words: echocardiography, cystic tumor, large cell tumor, endocarditis

(Inter Med 48: 733-734, 2009)
(DOI: 10.2169/internalmedicine.48.2020)

Picture 1. The cystic tumor had a pediculus attached to the right atrium wall above the anterior tricuspid valve which moved from the right atrium cavity at the diastolic phase (Picture 1-a, b) to the right ventricular cavity at the systolic phase (Picture 1-c, d). The morphology of the tumor changed from cycle shape at pre-systolic (Picture 1-a) and early-diastolic periods (Picture 1-c) to a pressed shape at systolic (Picture 1-b) and end-diastolic periods (Picture 1-d).
Three cystic tumors were observed and had the same pediculus attached to the right atrium wall (Picture 2-a). A histological study of most of the small cyst wall (Picture 2-b) showed fibrin, thrombus, macrophage infiltration, and malignant cells, without evidence of bacterial infection.

We present an 80-year-old man who consulted our hospital because of general fatigue and intermittent high fever. His systemic blood pressure was 140/80 mmHg and he had a pulse rate of 84 beats/min with a regular rhythm. A chest X-ray and chest CT images revealed bilateral pleural effusion without cardiomegaly and a thick lesion of pulmonary pleura at the left lung. Pleuralcentesis was performed and pleural fluid cytology was positive for malignant cells. An echocardiography examination showed a vegetation-like lesion of the aortic valve without regurgitation and a giant cystic tumor at the right atrium cavity. The cystic tumor had a pediculus attached to the right atrium wall cranial to the anterior tricuspid valve and it moved from the right atrium cavity at diastolic phase to the right ventricular cavity at systolic phase. The morphology of the tumor changed to a cycle shape at pre-systolic and early-diastolic period and a pressed shape at systolic and end-diastolic period, suggesting that the tumor wall and content were soft with a high compliance (Picture 1). In his blood tests, the white blood cell count increased from 13,100 mm$^3$ (80% neutrophils and 5% eosinophils) on admission to 31,700 mm$^3$ (75% neutrophils and 20% eosinophils) on the 37th hospital day. Repeated blood cultures were not significant positive for bacterial infection. He died on the 52nd hospital day from multi-organic disorder with disseminated intravascular coagulation, but there was no clinical evidence of systemic embolization. An autopsy revealed three cystic tumors and they had the same pediculus attached to the right atrium wall (Picture 2). The contents of two big cystic tumors were yellow fluid like serum and the most small cyst content was only thrombus. A histological study showed that malignant cells with thrombus invaded at the right atrium and ventricular endocardium. Fibrin, thrombus, macrophage infiltration, and malignant cells were observed at all cystic tumor walls and the vegetation-like lesion of the aortic valve, but there was no evidence of bacterial infection. Large cell carcinoma was revealed at the thick lesion of pleura as observed by CT images. In echocardiographic studies, nonbacterial thrombotic endocarditis usually occurs in left heart valves and produces bacteria-free verrucae in 19% and 63% of patients with solid malignant tumors and myeloproliferative disorders, respectively (1-3). However, there have been no reports regarding a cystic tumor caused by nonbacterial thrombotic endocarditis. Nonbacterial thrombotic endocarditis lesions have been previously reported on high-flow areas of the distal portion on heart valves or in an impaired endocardium lesion (4, 5). In the present case, the right atrium endocardium was impaired by the invasion of malignant cells, and thrombi formation occurred on the injured endocardium in the condition of platelet aggregation disorder and coagulopathy, which are frequently present in patients with cancer or myeloproliferative disease. The cystic formation might be produced by the necrotic change of the thrombus core, after the thrombotic mass developed from the thrombi formation and the malignant cell invasion.

References


© 2009 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html