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Acute Pulmonary Embolism Caused by Subacute Infective Endocarditis of the Tricuspid Valve

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Acute pulmonary embolism is usually caused by deep vein thrombosis of the lower extremities. However, vegetations of the tricuspid valve (TV) can cause embolic occlusion of the pulmonary vasculature and hence pulmonary embolism. We experienced a case of acute pulmonary embolism caused by subacute infective endocarditis of the TV. The computerized tomographic scans showed huge vegetations on the patient's TV and obstruction of the bilateral pulmonary arteries. He was successfully treated with intravenous antibiotics and TV replacement.

KEY WORDS: Acute pulmonary embolism · Subacute infective endocarditis · Tricuspid valve.

Introduction

Acute pulmonary thromboembolism (PE) is a common problem, with at least 600,000 estimated cases of newly diagnosed PE and more than 50,000 related deaths occurring in the United States each year.[1)2)] PE is usually caused by deep vein thrombosis of the lower extremities, but vegetations of the tricuspid valve (TV) can cause embolic occlusion of the pulmonary vasculature and pulmonary embolism, especially in the case of large emboli.

Here we present a case of acute pulmonary embolism caused by subacute infective endocarditis of the TV. The computerized tomographic scans showed huge vegetations on the patient's TV and obstruction on the bilateral pulmonary arteries. He was successfully treated with intravenous antibiotics and TV replacement.

Case

A 17-year-old man presented with dyspnea and fever for five days. He had had several episodes of acupuncture treatment for myalgia approximately two months before admission. On physical examination, his body temperature was 39.0℃, pulse rate 130 beats/min, respiratory rate 21 times/min, and blood pressure 100/72 mmHg. Grade 2-3/6 pansystolic murmurs were heard at the level of the TV. There were multiple variable-sized petechiae on the lower extremities without evidences of pustule or abscess formation. The initial oxygen saturation measured by pulse oxymetry was 89% with room air. A complete blood count revealed leukocytes 18,300 cells/mm^3, hemoglobin 11.0 g/dL, and platelets 1,280,000/mm^3. Urinalysis showed prominent hematuria (4+), leukocyturia (3+), and bacteriuria. A chest radiograph showed cardiomegaly with pulmonary edema and nodular lesions in the right lower lung field. Transthoracic echocardiogram performed in the emergency room showed increased right ventricular (RV) size and decreased RV systolic function associated with a D-shaped left ventricle. Multiple masses were noted on the TV and moderate tricuspid regurgitation (TR) was found. The measured maximal velocity of TR jet was 3.9 m/s (estimated systolic pulmonary arterial pressure was 71 mmHg) suggesting severe resting pulmonary hypertension (Fig. 1). Spiral computerized tomography of the chest demonstrated multifocal intraluminal filling defects with cavitary nodules adjacent to the segmental branches of the pulmonary arteries associated with proximal vascular dilatation (Fig. 2). Viral tests for human immunodeficiency virus and hepatitis virus B and C were all negative.

The patient was diagnosed with subacute infective endocarditis and treated with intravenous antibiotics including

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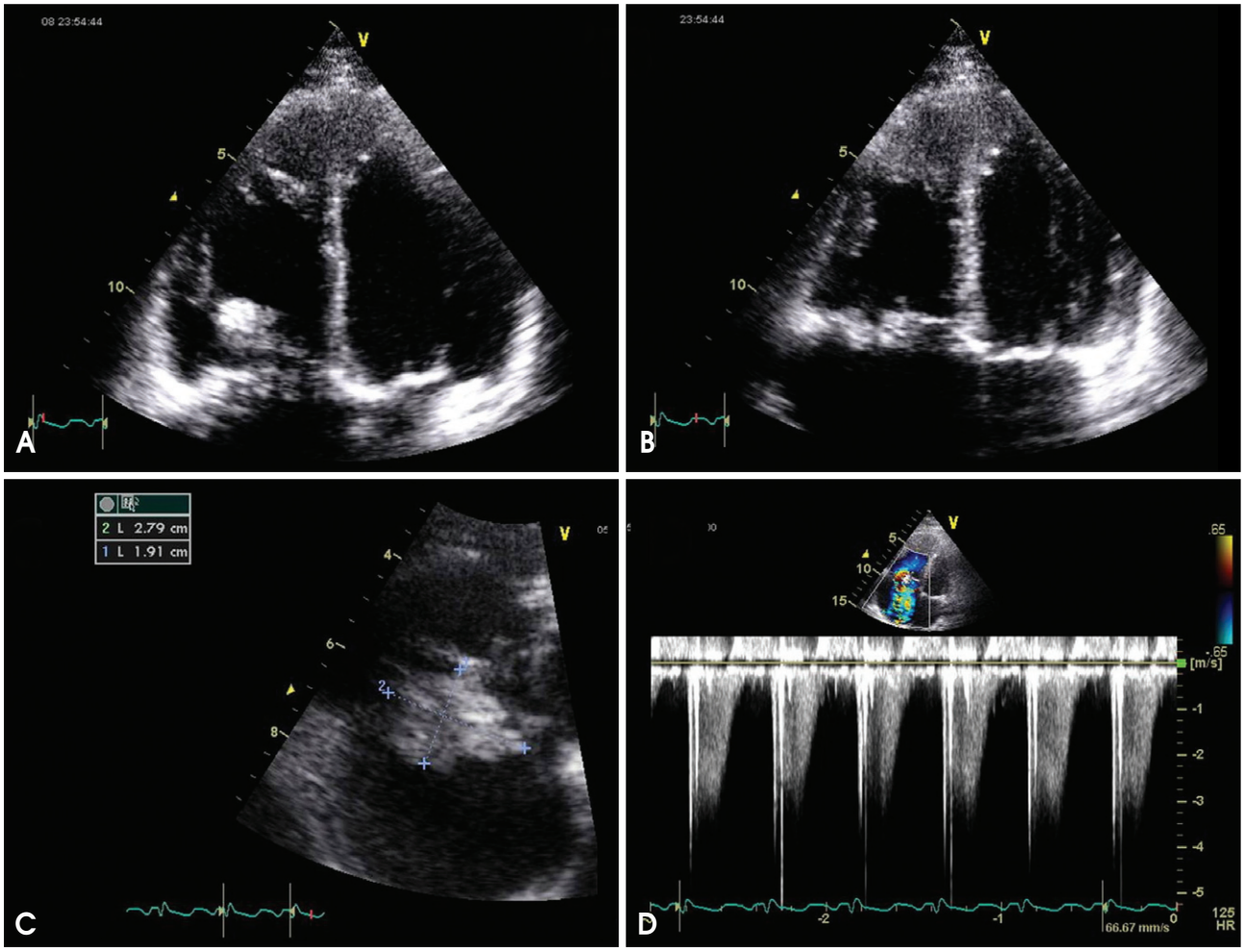

Fig. 1. Echocardiographic examination at admission showed right ventricular dilatation and systolic dysfunction (A: End-diastole. B: End-systole). About 28×19 mm sized masses was noted on the TV (C). The measured maximal tricuspid regurgitation velocity was 3.9 m/s (estimated systolic pulmonary arterial pressure was 71 mmHg) suggesting severe resting pulmonary hypertension (D).

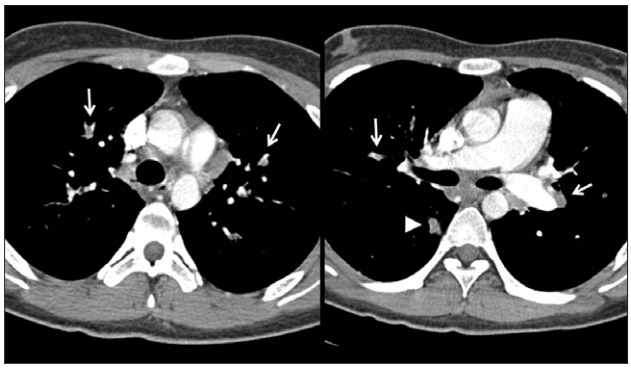

Fig. 2. Spiral computerized tomography of the chest demonstrating multifocal intraluminal filling defects (arrows) with cavitary nodules adjacent to the segmental branches of the pulmonary arteries associated with proximal vascular dilatation. There were many nodular lesions suggesting septic pneumonia (arrowhead).

penicillin and gentamicin. The results of blood culture revealed Streptococcus oralis in two of three sets of culture. After two weeks of antibiotic treatment, the patient underwent TV replacement with prosthetic tissue valve (St. Jude Medical, Minnesota, US) and removal of embolic materials from the pulmonary arteries. After the operation, the patient showed clinical improvement. The petechiae of the lower extremities disappeared after the operation and a six-week treatment of intravenous antibiotics, and the patient was discharged without other complications.

Discussion

Right-sided infective endocarditis (IE) accounts for 10% of all IE in population based surveys and a higher proportion of IE in injection drug users.[3] Right-sided IE may occur in association with congenital heart disease and instrumentation of the right heart.[4][5] Our case had no previous history of congenital heart disease or instrumentation. Acupuncture therapy can be associated with right-sided IE and usually caused by skin flora.[6][7] Patients with valvular heart disease and/or prosthetic valve are more susceptible for right-sided

IE.[6)7)] The only probable route of entry was acupuncture in our case. However, there was no history of valvular heart disease or congenital heart disease in our case.

The dominant infective organism in right-sided IE in most series is Staphylococcus aureus accounting for around 70% of infections, with the remainder being caused mainly by streptococcal species or, less frequently, by Gram-negative organisms, fungi or diphtheroids.[8)9)] The causative bacterium in our patient was Streptococcus oralis that was successfully treated with surgery and antibiotic treatment in our case.

The usual manifestations of right sided IE are persistent fever, bacteremia, and multiple pulmonary emboli.[10)] Symptoms and complications usually arise from the presence of multiple septic pulmonary emboli in the pulmonary vasculature, and hence chest discomfort, dyspnea, cough, and hemoptysis may be presenting features in these patients. Peripheral embolic manifestations are rarely present, especially in cases with either left-sided endocarditis or paradoxical embolism through abnormal shunt. The conjunction of bacteremia, fever, and multiple pulmonary infiltrates on chest radiography should suggest the possibility of a right-sided IE.[10)]

Complications of right-sided IE include pulmonary infarction, pulmonary abscesses, bilateral pneumothoraces, pleural effusions, and empyema. Multiple pulmonary emboli may result in right-sided chamber dilatation, right heart failure, and a worsening of tricuspid regurgitation. The embolization of vegetations large enough to result in obstruction of a significant portion of the pulmonary arterial circulation has been described, but is very unusual.[10)] In our case, the typical features of acute PE were caused by the obstruction of the proximal portion of the pulmonary arteries by large vegetations of the TV. Besides the presence of fever, clinical clues suggesting right-sided IE were obtained from the transthoracic echocardiographic examination showing multiple mobile masses on the TV and multiple nodular lesions on the chest radiograph. Patients with acute PE are usually treated with intravenous anticoagulant and/or thrombolytic therapy, because this patient was suspected of having right-sided IE, he was treated with intravenous antibiotics only. After stabilization with two weeks of antibiotic therapy, the patient was successfully treated with TV replacement.

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