Echocardiographic Manifestation of Acute Pulmonary Thromboembolism

A Case Report

Akira SHINA, M.D., Kunio KONDO, M.D., Nobuyoshi KAWAI, M.D.,* and Saichi HOSODA, M.D.

SUMMARY

We report a case of recurrent pulmonary thromboembolism with echocardiographic findings, documenting the occurrence, resolution and recurrence of pulmonary hypertension with right ventricular pressure and volume overload.

Additional Indexing Words:
Pulmonary hypertension Right ventricular overload Pulmonary infarction

It has been proven that echocardiography was useful in evaluating not only changes in cardiac chamber size but also changes in intracardiac pressure. In this communication, we report on the efficacy of echocardiography in evaluating acute right ventricular pressure and volume overload and pulmonary hypertension produced by pulmonary thromboembolism.

CASE REPORT

A 60-year-old female was admitted to Jichi Medical School on November 25, 1976 because of the sudden onset of high fever and chill. Examination revealed hepatomegaly with significant elevation of lactic dehydrogenase and alkaline phosphatase. A diagnosis of carcinoma of the gallbladder with liver metastasis was made by angiography and laparoscopy.

On December 16, the patient suddenly developed severe right pleuritic chest pain associated with dyspnea, palpitation, and spiky fever. There was no hemoptysis. Physical examination revealed moderate cyanosis, marked neck vein engorgement, and bilateral pitting edema. Her blood pressure was 100/76 mmHg, and the heart rate was 110/min, regular. Tachypnea (36/min) was noted. There

* Present address is Division of Cardiology, Seikeikai Hospital, Sakai City, Osaka.
Address for reprint: Akira Shiina, M.D., Department of Cardiology and Internal Medicine, Jichi Medical School, 3311-1, Minamikawachi-machi, Kawachi-gun, Tochigi-ken, 329-04, Japan.
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were an accentuated pulmonic second sound (II P), a high-pitched pansystolic murmur at the lower sternum which increased during inspiration, and a dull percussion sound with decreased breath sound at the base of the right lung. No pulmonary rales were heard.

Chest X-rays revealed increased translucency in the right pulmonary field associated with pleural effusion and elevation of the right side of the diaphragm. Electrocardiogram revealed sinus tachycardia (H.R. 110/min), low-voltage QRS waves in the limb leads, prominent p waves in leads II, III, and aVF, and a recently developed rSR' pattern in lead V1.

Echocardiograms (UCG) were recorded on an Ekoline 20A or an Aloka SSD-110 instrument using a 2.25 mHz ultrasonic transducer with a half-inch diameter. The transducer was placed on the left third intercostal space with the patient lying in a 30° semi-Fowler position.

UCG on December 18 revealed significant enlargement of the right ventricular cavity, reduction of the interventricular septal movement, abnormal motion of the pulmonic valve, including a decreased e-f slope, disappearance of the "a" wave and systolic semi-closure (Fig. 1 left).

![Fig. 1. Echocardiograms taken on December 18 and 26, 1976. On December 18, enlargement of the right ventricular cavity and reduction of the interventricular septal motion were observed (upper left). Abnormal pulmonic valve motion (lower left) suggested pulmonary hypertension. On December 26, the size of the right ventricular cavity and interventricular septal motion were almost normal (upper right). The pulmonic valve echo motion was normal (lower right).](image-url)
Based on these findings, a diagnosis of acute pulmonary hypertension and right ventricular pressure and volume overload due to pulmonary thromboembolism was made and this was confirmed by pulmonary arteriography (Fig. 2).

UCG taken on December 26 revealed that, although mild pericardial effusion still persisted, all other abnormalities described above had been reduced by the intravenous administration of sodium Heparin (10,000 U/day) and oxygen inhalation (Fig. 1 right). Phonocardiography indicated that the pulmonic second sound had become normal.

One month later, pulmonary thromboembolism recurred and UCG findings again indicated right ventricular overload and pulmonary hypertension (Fig. 3). Pulmonary scintigraphy confirmed an extension of the pulmonary thromboembolism to both lungs. While drug therapy improved the symptoms, the patient expired on March 15, 1977 due to progression of the gallbladder carcinoma, bleeding tendency, and hepatic dysfunction.

Autopsy revealed primary carcinoma of the gallbladder with metastatic foci in the liver and marked obstruction of the biliary tracts. An index-finger-sized yellowish-red thrombus was detected in the main branch of the pulmonary artery of the right lower lobe, and multiple thromboemboli were noted in the pulmonary arterial branches of the left upper and lower lobes. There was moderate bilateral pleural effusion and multiple segmental atelectasis, although no pleuritic changes were observed.

Fig. 2. Pulmonary arteriogram performed on December 27, 1976. The pulmonary arterial branch of the right lower lobe was totally obstructed.
Fig. 3. Echocardiograms on January 26, 1977, showed the right ventricle to be enlarged again (upper). The pulmonic valve motion revealed abnormal mid-systolic semi-closure, decreased e-f slope and disappearance of the "a" wave (lower).

Gross examination of the heart revealed no abnormality, except for a small amount of serous pericardial effusion.

DISCUSSION

The early diagnosis of acute pulmonary thromboembolism is clinically very important, but there are many attendant medical problems. In diagnosing this disease, electrocardiography, chest X-ray and pulmonary scintigram are useful, but they are not always specific. Pulmonary arteriography is the best diagnostic, but this invasive procedure involves some risk and cannot be made when the patient is severely ill.

Echocardiography is easy and safe to perform and is very useful in eval-
ulating the changes in chamber size and valve motion, especially in the follow-up of their instantaneous changes. Utilizing these properties, there have been some papers on the echocardiographic findings of pulmonary thromboembolism. However, their findings are not always obtained in all cases of this disease. Alpert et al reported a case in whom enlarged right ventricular cavity and unusual bifid pattern of left ventricular posterior wall was observed by echocardiogram when the patient had massive thromboembolic occlusion of his pulmonary vascular bed.

Kasper et al reported that a dilated pulmonary artery, delineated by suprasternal echocardiography, indicated an increase in pulmonary arterial pressure and that the ratio of right to left ventricular end-diastolic diameter correlated well with the angiographic index of severity of embolic obstruction in patients with acute pulmonary embolism. On the basis of the observations of Kasper et al and some other investigations, it can be assumed that the increase in right ventricular dimension and the abnormal septal motion in the acute stage of pulmonary thromboembolism are due to right ventricular pressure and/or volume overload. Steckley et al reported a case with acute pulmonary thromboembolism in whom interval development of right ventricular dilatation and paradoxical septal motion coincided with a clinical event which was proven angiographically to represent pulmonary thromboembolism. This observation is very similar to what was found in our case, but we recognized recurrent development of right ventricular dilatation associated with abnormal septal motion and recovery to normal.

Our observation suggests that transient dilatation of the right ventricle could be a very sensitive indicator in the assessment of acute right ventricular pressure and/or volume overload in the acute onset of pulmonary thromboembolism. Furthermore, abnormal pulmonic valve motion which coincided with the above-mentioned abnormal findings, was obtained during recurrent clinical events of acute pulmonary thromboembolism.

A decreased e-f slope, loss of the “a” wave and systolic semi-closure of the pulmonic valve echo are considered to be UCG abnormalities in pulmonary hypertension. All of these abnormal patterns were detected in our patient who presented with clinical evidence of pulmonary thromboembolism.

The recurrence of pulmonary thromboembolism was thought to be caused by disseminated intravascular coagulation (DIC). In the present case, UCG was useful in demonstrating the transient and reversible abnormalities of right ventricular dilatation and pulmonary hypertension.

Weyman et al reported mid-systolic closure or notching of the systolic segment of the pulmonic valve echogram in 18 of 20 subjects with pulmonary hypertension; this finding was not obtained in normal subjects in his study.
We made similar observations on 15 patients (unpublished results).

Although pulmonary arterial pressure could not be determined during the most serious stage of the disorder, periodic echocardiography at the patient’s bedside revealed abnormal motion of the pulmonic valve echo and enlargement of the right ventricle, suggestive of a significant elevation of pulmonary arterial pressure associated with right ventricular overload.

On the first day of the pulmonary thromboembolism, only slight pericardial effusion was recognized, but during the month following, pericardial effusion apparently increased in volume and persisted (Fig. 1 upper), and finally disappeared on Jan. 19, 1977. In our experience, the appearance of pericardial effusion during the acute stage of pulmonary thromboembolism has been noticed in 4 other cases. Therefore, this observation will be of interest that the genesis of pericardial effusion in cases with acute pulmonary thromboembolism may be due to acute right ventricular pressure and/or volume overload.

Our findings suggest that UCG is extremely useful for the bedside diagnosis of acute pulmonary hypertension associated with right ventricular pressure and volume overload in patients with pulmonary thromboembolisms, especially during episodes of recurrence.

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