CASE REPORTS

Echocardiographic Identification of Right-sided Cardiac Intracavitary Thromboembolus in Massive Pulmonary Embolism

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SUMMARY In a patient with clinical features of massive pulmonary embolism, two-dimensional echocardiography demonstrated an echo-dense mass moving freely across the tricuspid valve between the right atrium and right ventricle. This mass was also visualized at angiography, which in addition confirmed extensive pulmonary embolism. At autopsy, the mass was found to be a coiled thrombus, one end of which had become wedged in a probe-patent foramen ovale. In another patient, bedside echocardiographic demonstration of thrombus in the right ventricular outflow tract was followed by immediate and successful embolectomy.

MASSIVE PULMONARY thromboembolism is a common clinical problem. Pulmonary angiography is an accurate method of diagnosis, but when performed to outline the pulmonary vasculature in the most detailed and complete way (by injection of dye into the main pulmonary artery), does not outline the right-sided cardiac chambers. In this report, we describe a patient in whom two-dimensional echocardiography demonstrated the presence, subsequently confirmed by angiography, of coiled thrombus in the right atrium and the right ventricle. Echocardiography has not been extensively used to assess patients with pulmonary embolism, but we believe that it may have a useful role.

Case Report

A 65-year-old woman presented to the Royal Infirmary of Edinburgh with the sudden onset of dyspnea, which persisted for 8 days. She was initially found to have signs of right-sided heart failure and her physician recommended bedrest and diuretics. On the day of admission to the hospital, she had become more dyspneic and light-headed. The patient had no history of significant previous illness and was a nonsmoker.

Physical examination revealed an obese woman, clearly distressed by dyspnea. She had central cyanosis with a regular tachycardia of 120 beats/min and a blood pressure of 95/60 mm Hg (by sphygmomanometer). The jugular venous pressure was grossly elevated, but there was no detectable hepatomegaly or peripheral edema. There was no clinical evidence of deep vein thrombosis. On auscultation, there was a marked gallop rhythm; the third and fourth heart sounds were heard maximally at the lower left sternal edge. There were no cardiac murmurs. Examination of the lungs was normal and general examination was otherwise unremarkable.

A 12-lead ECG showed sinus tachycardia with nonspecific ST-T-wave changes. The chest radiograph showed enlargement of the cardiac shadow (cardiothoracic ratio 150/270), slight prominence of the right hilum and clear lung fields.

Breathing 60% oxygen by mask, the patient had an arterial P02 of 6.7 kPa (50 mm Hg), Paco2 of 4.0 kPa (30 mm Hg) and hydrogen ion concentration of 36 nmol/l (pH 7.44).

Echocardiography

Because the patient was obese and had dyspnea, we could not obtain an M-mode echocardiogram of diagnostic quality. Two-dimensional echocardiography was also difficult, but the right-sided cardiac chambers were visualized from an apical approach. An echo-dense mass was consistently seen moving back and forth across the tricuspid valve between right atrium and right ventricle. There was no obvious point of attachment of the mass to the atrial or the ventricular wall (fig. 1).

Cardiac Catheterization

The differential diagnosis included intracavitary thrombus, a right atrial myxoma and a right ventricular tumor. Approximately 18 hours after admission, right-heart catheterization was performed. The right-heart pressures were elevated (mean right atrial pressure 18 mm Hg; right ventricle 65/15 mm Hg) and there was moderate pulmonary hypertension (main pulmonary artery pressure 65/20 mm Hg). Right atrial angiography showed a large, apparently coiled mass moving freely across the tricuspid valve between the right atrium and the right ventricle (fig. 2); pulmonary angiography showed occlusion of several major pulmonary arterial branches.

Clinical Course

After cardiac catheterization, a continuous i.v. infusion of streptokinase was started, but despite adequate dosage (according to the results of hematologic monitoring) and initial clinical improvement, she suddenly collapsed and died 30 hours after the commencement of this therapy.
Autopsy Findings

There was no evidence of venous thrombosis in the leg or pelvic veins or in the inferior vena cava. Most of the major pulmonary arterial branches were occluded by thromboemboli, and there was early pulmonary infarction in much of the left lung. A coiled thrombus, whose appearance suggested a venous rather than a primary intracardiac origin, was found in the right-sided cardiac chambers, straddling the tricuspid valve; one end of the thrombus was wedged in a probe-patent foramen ovale (fig. 3). Except for hepatic venous congestion and papillary necrosis in the right kidney, no abnormality was found.

Discussion

The occurrence of right-sided intracavitary thromboembolism has long been recognized, although very few cases have been described.5-11 In a series of 2000 consecutive autopsies,9 four cases of embolism into the right ventricle, thought to be the cause of death, were described.

Antemortem diagnosis of this condition is rare. The sudden development of a systolic murmur or position-related cyanosis, in the presence of known deep vein thrombosis, has been said to be suggestive of this complication,12 but deep vein thrombosis is often clinically silent.14-17 The condition is commonly associated with pulmonary embolism.6, 7, 10, 12, 13

Covarrubias et al.18 described the M-mode echocardiographic appearances of multiple shaggy echoes adjacent to the tricuspid valve in a patient with sudden hypotension, multiple systolic clicks and a systolic murmur at the lower left sternal edge. The patient died approximately 2 days after echocardiography, and autopsy revealed a saddle embolus in the main pulmonary artery. The authors postulated that the clinical and echocardiographic findings were due to an embolus that became temporarily entrapped at or near the tricuspid valve and subsequently migrated to the main pulmonary artery.

The present case appears to be unique in that the right-sided intracavitary thromboembolism was recognized by two-dimensional echocardiographic exami-
nation soon after admission to hospital, was present the next day when angiography was performed, and remained in situ until the patient died, presumably from further pulmonary embolism. Although emboli have generally been described as entrapped in the tricuspid valve apparatus, in this case passage of the embolus through the right side of the heart may have been prevented by wedging within a probe-patent foramen ovale, a common anatomic abnormality also present in the case described by Covarrubias et al.18

The use of echocardiography to assess pulmonary embolism has not been widely reported. Kasper et al.19 described an increased dimension of the right pulmonary artery, as assessed by suprasternal M-mode echocardiography, in patients with an elevated mean pulmonary arterial pressure after pulmonary embolism; 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, provided there was no history of previous cardiopulmonary disease. Kasper and colleagues20 also described the echocardiographic demonstration of thrombus in the right pulmonary artery from a suprasternal approach. We have seen another patient in whom bedside M-mode echocardiography revealed an echo-dense mass in the right ventricular outflow tract; subsequent immediate pulmonary embolectomy resulted in the removal of a large thrombus and the patient made a good recovery.

We believe that echocardiography can be useful for assessing patients with suspected pulmonary embolism. Confident identification of thrombus in the pulmonary artery in the presence of other clinical and echocardiographic features of massive pulmonary embolism might permit urgent embolectomy without the delays or hazards inherent in angiography.

When surgical therapy is thought to be indicated for pulmonary embolism, the preoperative identification of intracavitary thromboembolus calls for exploration of the right atrium and right ventricle (a departure from standard surgical treatment21–23) to remove it. If left in situ, the embolus might jeopardize the success of the operation by obstructing the tricuspid valve or by acting as a source for further pulmonary emboli.

Whether the identification of right ventricular or right atrial thrombus should be regarded as an indication for surgical rather than medical treatment is conjectural. Left ventricular thrombus sometimes disappears rapidly with conventional anticoagulant treatment,24 but its structure and natural history may well be different from thrombus that embolizes to the right ventricle. In retrospect, the death of our patient after an initially satisfactory response to streptokinase may even have been due to further pulmonary embolism after partial dissolution of the right atrial and ventricular thrombus, a possibility already recognized with streptokinase treatment of peripheral venous thrombosis.25 In patients who already have a compromised pulmonary vascular bed, the presence of intracavitary thrombus should probably be considered an indication for surgery.

References


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\section*{Acute Coronary Artery Occlusion During Percutaneous Transluminal Coronary Angioplasty: Reopening by Intracoronary Streptokinase Before Emergency Coronary Artery Surgery to Prevent Myocardial Infarction}

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\textbf{SUMMARY} Percutaneous transluminal coronary angioplasty (PTCA) was complicated by acute coronary artery occlusion associated with ST elevation and severe chest pain in three patients. Within 10 minutes, the occluded artery was reopened by an intracoronary (i.c.) infusion of streptokinase, resulting in the disappearance of chest pain and normalization of ST segments. To keep the artery patent, i.c. streptokinase had to be continued until emergency bypass surgery was performed. In two patients, no myocardial infarction occurred, as shown by a normal postoperative left ventricular angiogram, ECG and thallium-201 scintigram. In the other patient, who was admitted with an inferior infarction and underwent PTCA after i.e. lysis, no infarct extension was observed. These results show that i.e. streptokinase rapidly opens an acute coronary artery occlusion complicating PTCA, preventing myocardial infarction.

\textbf{SINCE} its introduction by Gr"untzig et al.\textsuperscript{1} in 1978, percutaneous transluminal angioplasty (PTCA) has been performed in more than 1200 patients. Acute coronary artery occlusion is a well known complication of PTCA, and requires emergency coronary artery bypass graft surgery (CABG).\textsuperscript{2} In many cases, however, the interval between the occlusion and CABG is too long to prevent myocardial infarction.\textsuperscript{3} To reestablish flow early after occlusion, we infused streptokinase into the occluded coronary artery in three cases, and then performed emergency CABG.

\textbf{Case Reports}

\textbf{Case 1}

A 64-year-old female patient had severe angina pectoris due to an isolated, proximal LAD stenosis (fig. 1A). Left ventricular wall motion was normal (fig. 2). She had no electrocardiographic evidence of prior myocardial infarction. On the day before PTCA, she received acetylsalicylic acid, 1 g twice a day. During the procedure, she received 10,000 U of heparin i.v. and 10 mg of nifedipine sublingually. PTCA was attempted using a Gr"untzig dilatation catheter (type DG 20-30). The stenosis was passed. Contrast injection through the dilatation catheter revealed a slightly delayed filling of the left anterior descending coronary artery (LAD) compared with the diagonal branch (fig. 1B). However, this was recognized retrospectively and was interpreted as a dissection induced by the catheter tip. In this position, the balloon was inflated. Immediately after inflation, the patient complained of severe chest pain with monophasic ST elevation. A control injection showed a complete occlusion of the LAD at the bifurcation (fig. 1C). After sublingual and intracoronary nitroglycerin (5 mg and 0.2 mg, respectively) and nifedipine (10 mg and 0.2 mg, respectively), the artery remained occluded. Streptokinase was then infused into the ostium of the left main coronary artery at a rate of 2000 U/min.\textsuperscript{4} Within a few minutes, the chest pain disappeared and the ST segments returned to normal. Contrast injection revealed a patent LAD with a high-grade remaining stenosis (fig. 1D). However, occlusion and chest pain recurred when intracoronary streptokinase was discontinued. It was started again and resulted in immediate reopening of the LAD. The intracoronary streptokinase infusion was continued until CABG. The LAD was grafted.

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