Central Pulmonary Artery Lesions in Chronic Obstructive Pulmonary Disease

A Transesophageal Echocardiography Study

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Background—In patients with acute pulmonary embolism, transesophageal echocardiography (TEE) often reveals presumably thrombotic lesions within the central pulmonary arteries (CPAs). These CPA lesions, when found in patients with primary pulmonary hypertension, have been attributed to in situ thrombosis or atherosclerosis. We hypothesized that similar CPA lesions may also develop in patients with chronic obstructive pulmonary disease (COPD) in the absence of pulmonary embolism.

Methods and Results—We examined by TEE 25 patients with COPD and 27 control patients with left heart disease. None of the patients had previous pulmonary embolism or ileofemoral and popliteal vein thrombosis. By use of TEE, CPA lesions were found in 12 COPD patients (48%) and 2 control patients (7.4%) ($P<0.01$). When CPA lesions were subdivided into types 1 (protruding and mobile) and 2 (wall-adherent), type 1 lesions proved to be uncommon, being found within the pulmonary trunk in 12% and 3.7% of COPD and control patients, respectively ($P=NS$). Conversely, type 2 lesions, which were always localized in the right pulmonary artery, were frequent in COPD patients (36%) and rare in control patients (3.7%) ($P<0.01$). When available, helical CT and MR angiography confirmed TEE findings, supporting an atherosclerotic origin of type 2 lesions, which were different from typical thrombotic lesions. FEV$_1$/FVC ratio, RV/TLC ratio, PaO$_2$, hematocrit value, and pulmonary artery systolic pressure were not significantly different in COPD patients with and without CPA lesions. At TEE, however, COPD patients with CPA lesions showed a larger size of the main and right pulmonary arteries.

Conclusions—TEE often reveals CPA lesions in stable patients with COPD even in the absence of significant pulmonary hypertension and not in close relation with the severity of pulmonary dysfunction. (Circulation. 1999;100:1808-1815.)

Key Words: echocardiography ▪ thrombus ▪ pulmonary heart disease

In patients with acute pulmonary embolism, transesophageal echocardiography (TEE) frequently shows extensive (presumably thrombotic) lesions within the central pulmonary arteries.$^{1-12}$ In most patients, the source of these central pulmonary artery (CPA) lesions is probably embolization from peripheral veins.$^{13,14}$ However, CPA lesions with similar morphology were also found in some patients with pulmonary hypertension but no previous pulmonary thromboembolism.$^{15}$ In these patients, CPA lesions were attributed to development of in situ thrombosis, although an atherosclerotic origin could not be excluded.$^{15}$ CPA lesions found at TEE may be unsuspected clinically, as demonstrated in critically ill patients.$^{16}$ The prevalence of these CPA lesions in other clinical settings, and in particular in patients in stable condition, remains unknown.

In an old autopsy study, 28% of patients with chronic pulmonary disease and no previous pulmonary thromboembolism had CPA lesions.$^{17}$ This high rate is not surprising, because chronic pulmonary diseases are associated with pulmonary hypertension$^{18,19}$ and hypercoagulability,$^{20}$ which may promote either in situ thrombosis or atherosclerosis within the great pulmonary arteries. No data exist, however, on the prevalence in vivo of CPA lesions in patients with chronic pulmonary disease. Accordingly, we used TEE to determine whether vascular lesions compatible with in situ thrombosis or atherosclerosis are present at the level of the great pulmonary arteries in patients with chronic obstructive pulmonary disease (COPD) and no previous or recent pulmonary embolism. As a control, we examined a group of patients with left heart disease of various origins.

Methods

Patients

Between January 1995 and January 1997, we examined by TEE 27 consecutive outpatients with chronic pulmonary disease (obstructive...
pattern) and, as a control group, 27 consecutive patients with left heart disease of various origins. All patients were in stable clinical condition. In particular, patients did not have a history of pulmonary embolism or recent symptoms suggesting a diagnosis of pulmonary embolic events, such as sudden onset of dyspnea or pleuritic chest pain. Moreover, all patients were ≤75 years old, and none of them had acquired conditions often associated with pulmonary embolism, such as recent surgery, trauma, immobilization, cancer, obesity, or localized or unilateral swelling of an extremity. All patients in both the study and the control groups underwent color Doppler ultrasonography of the lower extremities, and neither ileofemoral nor popliteal vein thrombosis was detected in any of them.

COPD was diagnosed on the basis of clinical history, physical examination, chest radiograph, arterial blood gas analysis, and pulmonary-function tests (FEV₁/FVC <70% of the predicted value). As an index of lung hyperinflation, the residual volume/total lung capacity ratio (RV/TLC) was calculated. Two patients with COPD were excluded from analysis because of incomplete echocardiographic examination owing to severe discomfort during the procedure. Thus, the study group consisted of 25 patients (22 men and 3 women, mean age ± SD 65 ± 6 years, ranging from 53 to 74 years). In the control group, there were 15 men and 12 women (mean age ± SD 61 ± 14 years, ranging from 39 to 72 years). Ten patients had ischemic heart disease, 11 had mitral valve disease (6 with pure mitral valve stenosis), and 6 had primary dilated cardiomyopathy.

Echocardiography

Transthoracic echocardiography (TTE) and TEE were performed with an Aloka SSD-870 or an Acuson 128 XP5 system using 2.25- or 3.5-MHz transducers for TTE and 5.0-MHz multiplane transducers for TEE. All studies were stored on a 1/2-inch video recorder for subsequent review. Two observers unaware of the clinical findings reviewed each study.

TTE was performed with the patient lying in the left recumbent position. Cranio-caudal and lateral dimensions of the right atrium and ventricle were measured in the 4-chamber apical view at end systole and end diastole, respectively. Fixed or mobile lesions within the right atrium or ventricle were noted. Tricuspid regurgitation was determined by color Doppler. Pulmonary artery systolic pressure (PASP) was estimated by continuous-wave Doppler analysis of tricuspid regurgitation jet.

TEE was performed in the fasting state and after the patients’ histories had been assessed for esophageal disorders. We did not administer sedatives or analgesics intravenously to any of the patients. The transducer was introduced with the patient lying in the left recumbent position. The base of the heart was first imaged in the short-axis view. Then the transducer was tilted and withdrawn superiorly to visualize the main and proximal right and left pulmonary arteries. The left pulmonary artery could be imaged on the transverse plane only in its proximal segment because of interposition of the left main bronchus. The right pulmonary artery was imaged on the transverse plane along its entire course up to the division into its major lobar branches. For longitudinal plane imaging, the transducer was rotated slightly to the right to visualize the pulmonary valve and the main pulmonary artery up to its bifurcation. With further rightward rotation, the right pulmonary artery could be imaged in the short-axis view. Similarly, the left pulmonary artery was imaged by rotating the transducer laterally. After imaging of the main pulmonary artery, the diameter of the main, right, and left pulmonary arteries was measured at the origin of the vessels whenever possible.

We noted any echolucent lesions present in both transverse and longitudinal scanning in any segment of the great pulmonary arteries. On the basis of their pattern and mobility, CPA lesions were classified as type 1 when they were in part protruding and mobile, suggesting thromboembolism, and as type 2 when they were totally wall-adherent (either laminated or only in part protruding). No attempt was made to subdivide type 2 lesions into thrombotic and atherosclerotic on the basis of their TEE morphology. Artifact images within the pulmonary arteries were excluded through fine adjustments of the transducer rotation in each plane, by modifying the gain regulation settings, and with the aid of color Doppler flow imaging. Location and mobility patterns of structures adjacent to the transducer were also considered to exclude a reverberation phenomenon. In particular, fine linear echoes within the right pulmonary artery were considered to be probable reverberation from the right pulmonary posterior wall. A spontaneous contrastographic effect inside the pulmonary artery was also noted.

Other Imaging Techniques

Helical CT, MR angiography after Gd-DTPA injection, and conventional pulmonary angiography were available in 6, 4, and 1 COPD patients, respectively.

Statistical Analysis

Comparisons of continuous and discrete variables were made by the test for unpaired data and either χ² or Fisher’s exact test as appropriate. A value of P<0.05 was considered to be significant.

Results

Patients With COPD Versus Control Patients

Atrial fibrillation was present in 4 COPD patients (16%) and 11 control patients (40.7%) (P=0.09) (Tables 1 and 2). On TTE, no COPD or control patient had right atrial or ventricular thrombus. There was no difference in the size of the right atrium and ventricle or prevalence of tricuspid regurgitation between COPD and control patients. PASP tended to be greater in COPD patients than in control patients, considering only individuals in whom PASP could be assessed (n=14 and 17, respectively), but the difference was not significant. At TEE, the main and right pulmonary arteries were visualized along their entire course in transverse and longitudinal planes in all COPD and control patients. In contrast, the left pulmonary artery was visualized exclusively in its proximal portion (transverse plane) and in only 19 of 25 COPD patients (76%) and 19 of 27 control patients (70%) (P=NS). The diameter of the CPA was not different between COPD and control patients.

CPA lesions were found in 12 COPD patients (48%) and 2 control patients (7.4%) (P<0.01) (Table 3). Examples of type 1 and type 2 CPA lesions are shown in Figures 1 and 2, respectively. The 2 control patients with CPA lesions (1 of type 1 and 1 of type 2) had mitral valve stenosis with mild pulmonary hypertension. Type 1 and type 2 CPA lesions were found in 3 (12%) and 9 (36%) COPD patients, respectively (P=NS and P=NS, respectively, versus control group [both: 3.7%]). One patient with COPD had 2 type 2 CPA lesions in the right pulmonary artery.

All type 1 CPA lesions were found at the level of the pulmonary trunk, in contrast with those of type 2, which were found exclusively at the level of the proximal right pulmonary artery. No patient had lesions in the left pulmonary artery. No CPA lesion was occluding or obstructive, as assessed by color and pulsed-wave Doppler. All type 1 CPA lesions were attached to the vessel wall. Their size ranged from 2 to 5 cm in length and from 1×3 to 2×5 cm in cross section. Type 2 CPA lesions measured 3 to 7 cm in length and 2×2 to 3×5 cm in cross section. In no patient was a spontaneous echo-contrast phenomenon noted.

Helical CT was available in 4 COPD patients with CPA lesions (1 of type 1 and 3 of type 2) and in 2 without. In all these patients, CT scanning confirmed TEE findings. How-
ever, CPA lesions appeared to be smaller by helical CT scanning than by TEE. An example of a type 2 CPA lesion at helical CT scanning is shown in Figure 3. MR angiography after Gd-DTPA injection was available in 4 COPD patients (2 of type 1 and 2 of type 2). In 3 of them (2 with type 1 and 1 with type 2 lesions), MR angiography confirmed the presence of some abnormality within the CPA. Also with MR angiography, type 1 lesions were similar to

### TABLE 1. Clinical and Echocardiographic Data in COPD Patients

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*Patients with tricuspid regurgitation at continuous-wave Doppler.

TABLE 2. TTE and TEE Data in COPD and Control Patients

|                      | COPD (n=25) | Control (n=27) | P  
|----------------------|-------------|----------------|-----
| **TTE**              |             |                |     
| Right ventricle superoinferior diameter, mm | 49±9        | 46±14          | NS  
| Right ventricle laterolateral diameter, mm  | 29±7        | 33±7           | NS  
| Right atrium superoinferior diameter, mm   | 40±7        | 46±10          | NS  
| Right atrium laterolateral diameter, mm     | 37±6        | 37±6           | NS  
| Tricuspid regurgitation, %                  | 56          | 63             | NS  
| PASP,* mm Hg                                  | 45±18       | 40±9           | NS  
| **TEE**                                         |             |                |     
| Main pulmonary artery diameter, mm          | 25±6        | 25±3           | NS  
| Right pulmonary artery diameter, mm         | 19±7        | 20±4           | NS  
| Left pulmonary artery diameter,† mm         | 19±7        | 18±4           | NS  

*Only patients with tricuspid regurgitation at continuous-wave Doppler.

†The left pulmonary artery was visualized in 19 COPD (76%) and 19 control patients (70%).
those often visible in pulmonary thromboembolism. The type 2 lesion was stratified on the right pulmonary artery wall and was well evident in either the coronal or sagittal plane (Figure 4). In the other patient with a type 2 lesion by TEE, no CPA abnormality was found by MR angiography. Another patient with a type 2 lesion underwent conventional pulmonary angiography, which confirmed the presence of a lesion within the right pulmonary artery.

COPD Patients With CPA Lesions Versus Those Without
COPD patients with and without CPA lesions were similar in age, prevalence of atrial fibrillation, hematocrit value, $PaO_2$, $FEV_1/FVC$ ratio, and $RV/TLC$ ratio (Table 4). The right atrial and ventricular sizes and prevalence of tricuspid regurgitation were similar in COPD patients with and without CPA lesions. Among patients in whom it could be assessed (75% and 38% of those with and without CPA lesions, respectively), PASP tended to be greater in those with CPA lesions than in those without (49±18 versus 38±18 mm Hg, respectively; $P=NS$). PASP could be assessed in 7 of 9 patients with type 2 CPA

| Table 3. Pulmonary Artery Lesions at TEE in COPD and Control Patients |
|---------------------------------|-----------------|-------------|
|                                  | COPD (n=25)     | Control (n=27) | $P$   |
| Type 1 lesions, n (%)           | 3 (12.0)        | 1 (3.7)      | NS    |
| Type 2 lesions, n (%)           | 9 (36.0)        | 1 (3.7)      | <0.01 |
| Type 1 + type 2 lesions, n (%)  | 12* (48.0)      | 2 (7.4)      | <0.01 |

*In 1 patient with COPD, 2 type 2 lesions were present.

Figure 1. TEE section intermediate between longitudinal and transverse planes in a patient with COPD: a protruding lesion (arrow) is present within main pulmonary artery (MP) before origin of right pulmonary artery (RP). In real time, this lesion appeared to be attached to artery wall through a fixed base and slightly motile. This type 1 lesion had some features similar to thromboembolism.
lesions and was ≥40 mm Hg in 4 patients and <40 mm Hg in 3. The diameters of the pulmonary trunk and right pulmonary artery were significantly greater in patients with CPA lesions than in those without (P<0.05 for both).

**Discussion**

We have hypothesized that patients with chronic pulmonary disease and no pulmonary embolism may show CPA lesions at TEE. In COPD patients, in fact, reversible or fixed pulmonary hypertension, prothrombotic state, and hypoxemia might promote metabolic and structural changes in the artery wall resulting in in situ thrombosis or atherosclerosis. Confirming our hypothesis, CPA lesions were detected by TEE in 48% of our COPD patients, who were clinically stable and did not have previous pulmonary embolism. Specifically, 12% of COPD patients had slightly motile masses that were protruding into the pulmonary trunk (type 1 lesions), and 36% of them had flat or rounded lesions that

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**Figure 2.** TEE transverse plane section in a patient with COPD: a round lesion (arrows) is present, totally adherent to right pulmonary artery (RPA) wall. In real time, this lesion was not motile. This type 2 lesion was considered to be possible in situ thrombosis or atheroma.

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**Figure 3.** Helical CT scanning in COPD patient with type 2 lesion by TEE presented in Figure 2. A round lesion (*) is evident totally adherent to right pulmonary artery (RPA) wall. Ao indicates aortic root.
were totally adherent to the right pulmonary artery wall (type 2 lesions). CPA lesions were remarkably frequent in COPD patients in comparison with the low prevalence (7.4%) in cardiac patients.

These findings in COPD patients can be explained by silent thromboembolism, in situ thrombosis, or pulmonary atherosclerosis. The first mechanism is convincing only for type 1 CPA lesions. These lesions, in fact, were morphologically similar to typical thromboembolism except for their modest motility and absence of obstruction. The localization of type 2 CPA lesions in the right pulmonary artery may be in favor of thromboembolism, because the right branch is prone to receive emboli for flow continuity with the pulmonary trunk. Conversely, they were morphologically different from thromboembolism. Moreover, silent thromboembolism is rare, being described only in patients with prolonged immobilization from bed rest, recent major surgery, severe congestive heart failure, intubation, etc., a clinical context different from that of our study patients, who did not have recent or worsening dyspnea or peripheral vein thrombosis. Thus, although we did not perform pulmonary scintigraphy or lung and abdominal CT scans as a protocol, we judge silent thromboembolism to be an unlikely pathogenetic mechanism because of the high prevalence of CPA lesions and their peculiar occurrence in stable COPD patients.

Type 2 CPA lesions may be more appropriately explained by in situ thrombosis or, alternatively, by wall thickening with superimposed atherosclerotic debris. In fact, pulmonary arteries often develop thrombosis or wall hypertrophy in response to injury. Thrombotic lesions are common in the peripheral nonelastic pulmonary arteries of patients with pulmonary hypertension. Similar lesions have also been detected in the central elastic pulmonary arteries of a few patients with primary pulmonary hypertension and were considered to be engrafted on intimal injury caused by prolonged exposure to high blood pressure. However, atherosclerosis also may develop in the presence of increased pulmonary flow or pressure. A definitive distinction

**Figure 4.** MR angiography after Gd-DTPA bolus intravenous injection in a COPD patient with type 2 lesion by TEE. An abnormality (arrow) stratified on inferior wall of main branch of right pulmonary artery (RPA) is visible in both coronal (top) and sagittal (bottom) planes. LA indicates left atrium; LPA, left pulmonary artery.
Lesions were more frequent in COPD patients than found in this study. However, more data are necessary to determine their exact origins. Consequently, even in the absence of firm anatomic confirmation, we suggest that TEE type 2 lesions may actually represent CPA atherosclerosis.

We never found lesions within the left pulmonary artery. However, having imaged this artery only proximally and only available in some of our COPD patients with CPA lesions. The presence of some CPA abnormality was in most cases confirmed by both CT and MR angiography, but the lesions were less evident by these techniques than by TEE. Most importantly, also at CT and at MR angiography, CPA type 2 lesions appeared to be different from typical pulmonary thrombotic lesions. Rather, they showed some morphological similarities with the atherosclerotic lesions of the aorta. Consequently, even in the absence of firm anatomic confirmation, we suggest that TEE type 2 lesions may actually represent CPA atherosclerosis.

Conclusions

Our findings may have some clinical implications. In the absence of thromboembolism arising from peripheral veins, unexplained acute respiratory failure might be attributed in some COPD patients to distal migration of atherothrombotic material from the great pulmonary arteries. Moreover, the progressive growth of CPA lesions might contribute to further increase in pulmonary artery pressure, with possible therapeutic implications, such as long-term anticoagulation. Further studies in large populations are necessary, however, to confirm our TEE findings in obstructive and nonobstructive forms of chronic pulmonary disease and to understand their mechanisms.

References


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