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A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]



Right and Left Ventricular Function and Pulmonary Artery Pressure in Patients With Bronchiectasis*

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Background: Bronchiectasis may have deleterious effects on cardiac function secondary to pulmonary hypertension (PH). This study was designed to assess cardiac function and determine the prevalence of PH in patients with cystic and cylindrical bronchiectasis.

Methods: A cross-sectional study of patients with bronchiectasis diagnosed by CT scan was conducted at King Khalid University Hospital, Riyadh, Saudi Arabia between December 2005 and January 2007. Pulmonary function tests were performed, arterial blood gas measurements were made, and cardiac function and systolic pulmonary artery pressure (SPAP) were assessed by echocardiography.

Results: Of 94 patients (31% men, $n = 29$), 62 patients (66%) had cystic bronchiectasis and 32 patients (34%) had cylindrical bronchiectasis. Right ventricular (RV) systolic dysfunction was observed in 12 patients (12.8%), left ventricular (LV) systolic dysfunction was observed in 3 patients (3.3%), and LV diastolic dysfunction was observed in 11 patients (11.7%); all had cystic bronchiectasis. RV dimensions were significantly greater in the cystic bronchiectasis group, and were positively correlated with SPAP ($p < 0.0001$) and negatively correlated with PaO_2 ($p < 0.016$). Other hemodynamic variables were not different between groups. PH in 31 patients (32.9%) was significantly greater in patients with cystic bronchiectasis compared with cylindrical bronchiectasis ($p = 0.04$). In cystic bronchiectasis, SPAP was positively correlated with PaCO_2 ($p = 0.001$), and inversely correlated with PaO_2 ($p = 0.03$), diffusion capacity of the lung for carbon monoxide percentage ($p = 0.02$), and FEV_1 ($p = 0.02$).

Conclusions: RV systolic dysfunction and PH were more common than LV systolic dysfunction in bronchiectatic patients. LV diastolic dysfunction was mainly seen in severe PH. We recommend detailed assessment of cardiac function, particularly LV diastolic function, in patients with bronchiectasis. (CHEST 2008; 133:468–473)

Key words: bronchiectasis; cardiac function; pulmonary hypertension

Abbreviations: CO = cardiac output; DLCO = diffusion capacity of the lung for carbon monoxide; EDD = end-diastolic diameter; FS = fractional shortening; LV = left ventricular; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; LVESD = left ventricular end-systolic diameter; PH = pulmonary hypertension; RA = right atrial; RV = right ventricular/ventricle; SPAP = systolic pulmonary artery pressure; SV = stroke volume; TLC = total lung capacity

Bronchiectasis is dilatation of the bronchial walls resulting from chronic airway infection, which leads to structural lung tissue damage. It is manifested by repetitive productive cough and is occasionally associated with hemoptysis.^{1–5}

Hemodynamic alterations due to bronchiectasis have been described.^{5–6} Based on these changes, Ashour⁷ proposed two types of bronchiectasis: (1) perfused

type, mainly seen in cylindrical bronchiectasis; and (2) nonperfused type, which is seen in cystic bronchiectasis. He proposed that in nonperfused disease, more capillary bed destruction and left-to-right shunt can occur, which can eventually have a deleterious effect on cardiac function and pulmonary gas exchange.⁷

Koelling et al⁸ demonstrated left ventricular (LV) diastolic dysfunction in 40 patients with cystic fibrosis,

while LV diastolic function was preserved in 9 patients with bronchiectasis. Right ventricular (RV) systolic function was impaired in both groups. The small number of patients with bronchiectasis in this study precluded reaching definitive conclusions about ventricular function in these patients.⁸

There are no systematic studies on cardiac function or pulmonary hypertension (PH) in patients with bronchiectasis. Accordingly, we designed this study to assess RV and LV function in cystic bronchiectasis and cylindrical bronchiectasis, as well as to determine the prevalence of PH in these patients and correlate these findings with pulmonary gas exchange.

MATERIALS AND METHODS

Ninety-four stable patients with bronchiectasis comprised the study group. We recruited patients from the outpatient clinic of King Khalid University Hospital, a large tertiary medical center in Riyadh, Saudi Arabia, between December 2005 and January 2007. All consecutive patients with a diagnosis of bronchiectasis proven by high-resolution CT scan of the chest were included. The local research ethics committee approved the study. Consent was obtained from each patient involved. Patients who were ex-smokers, had a history of hypertension, valvular, or rheumatic heart disease, ischemic heart disease, or cardiomyopathy; had previous resectional lung surgery; or had findings of mixed cystic and cylindrical disease on CT scan or positive sweat chloride test results were excluded.

Demographic and clinical data obtained included patient age, sex, respiratory symptoms, oxygen supplementation, and signs such as cough, sputum, hemoptysis, shortness of breath, wheezes, cyanosis, clubbing of fingers, and crackles. All patients were previously screened for acid-fast bacilli (stain and culture), Igs, and α_1 -antitrypsin level, and underwent ECG and chest radiography.

Pulmonary function assessment was measured as FEV₁ and FVC using a spirometer. Total lung capacity (TLC) and residual volume were measured by plethysmography. Diffusion capacity of the lung for carbon monoxide (DLCO) was measured using the carbon monoxide single-breath technique (Master Screen PFT, 671178; Erich Jaeger, GmbH; Hoechberg, Germany). Data were expressed as percentages of predicted values using the standard protocol from the American Thoracic Society.⁹ Arterial blood gases were measured while patients were breathing room air: pH, PaCO₂, and PaO₂ were recorded.

All patients underwent high-resolution CT of the chest (Light Speed L52002; GE Medical Systems; Milwaukee, WI). Two chest

radiologists who were blinded to clinical severity of disease, spirometry, and echocardiographic findings differentiated between cystic bronchiectasis and cylindrical disease. Cylindrical bronchiectasis was diagnosed based on dilatation and thickening of the bronchial wall with a broncho/arterial ratio > 1.¹⁰ Cystic bronchiectasis was diagnosed by noticing thin-walled cystic spaces that may contain fluids and were seen in subsequent axial cuts either in conglomerate fashion or in branching order.¹¹

Doppler echocardiography was reviewed and read by a certified cardiologist who did not know about the aim of the study (model 5500; Phillips; Andover, MA). LV systolic function was assessed by estimating the LV ejection fraction (LVEF) using end-diastolic diameter (EDD) in the Teicholz formula.¹²

$$\text{Volume} = \text{EDD}^3 \times 7/2.4 + \text{EDD}$$

LV fractional shortening (FS) was measured using the following formula: FS = ((LVEDD - LVESD)/LVEDD) × 100%, where LVEDD is LV EDD and LVESD is LV end-systolic diameter. RV systolic function was assessed by measuring the systolic excursion of the tricuspid annulus as follows: normal (≥ 2 cm); mild (< 2 cm); moderate (< 1 cm); or severely impaired (hypokinetic) [< 0.5 cm].¹³ The thickness of the RV free wall and the pattern of ventricular septal motion were noted. The simplified Bernoulli equation was used to calculate the systolic pulmonary artery pressure (SPAP) from the peak velocity of tricuspid regurgitant jet, and the right atrial (RA) pressure was added based on inferior vena cava collapsibility. In those patients for whom the tricuspid regurgitant jet signal was not adequate, SPAP was calculated using the Meehan formula. PH was defined in this study as SPAP ≥ 40 mm Hg based on criteria established by the World Health Organization Symposium on Primary Pulmonary Hypertension (1998). Further, those with SPAP of 40 to 70 mm Hg had mild-to-moderate PH, and those with SPAP > 70 mm Hg were considered to have severe PH based on the criteria modified from Mayo Clinic.¹⁴

Stroke volume (SV) was derived as follows: SV = CSA × VTI; where CSA is cross-sectional area and VTI is velocity time integral. Cardiac output (CO) was derived by multiplying SV by heart rate.¹⁵

Diastolic function was assessed by evaluation of mitral inflow, E-wave velocity, A-wave velocity, E/A ratio, and deceleration time. Tissue Doppler imaging of mitral annulus was also performed. The E' velocity, the A' velocity, and the E'/A' ratio were analyzed.¹⁶

Data were analyzed using statistical software (SPSS Pc+; SPSS; Chicago, IL). Continuous variables that followed a normal distribution were summarized as mean ± SD. Comparisons between two groups were done using Student *t* test (two tailed), and rates were compared using χ^2 test. Pearson correlation coefficient was calculated to quantify the relationship of two continuous variables. Simple regression analysis was performed to determine which echo finding would best explain the variability of lung function; *p* < 0.05 was considered statistically significant.

RESULTS

Of the 94 patients included in the study, 29 patients (30.8%) were men. Mean age was 53.4 years (SD, 17.7 years). Almost two thirds (*n* = 62) of the patients had cystic bronchiectasis, with the remainder (*n* = 32) having cylindrical bronchiectasis. Mean values of FVC, FEV₁, and DLCO percentage of predicted values, PaCO₂, and PaO₂ in cystic bronchiectasis patients were significantly lower than the mean values in patients with cylindrical bronchiec-

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Table 1—Pulmonary Function Test Results of 94 Patients With Bronchiectasis*

Variables	All	Cystic Bronchiectasis	Cylindrical Bronchiectasis	Cystic vs Cylindrical Bronchiectasis	
				t Value	p Value
Vital capacity, % predicted	65.8 ± 21.4	58.9 ± 19.6	79 ± 18.4	4.7	< 0.0001†
FEV ₁ , % predicted	59.7 ± 23.2	53.4 ± 21.6	71.3 ± 22.0	3.8	< 0.0001†
TLC, % predicted	91.1 ± 22.4	87.9 ± 18.6	97.3 ± 27.8	1.6	0.11
Residual volume, % predicted	134.5 ± 57.5	131.1 ± 51.9	140.3 ± 71.5	0.57	0.57
DLCO, % predicted	66.7 ± 25.9	61.1 ± 25.5	76.9 ± 24.0	2.7	0.01†
pH	7.4 ± 0.04	7.4 ± 0.05	7.4 ± 0.03	0.56	0.57
PCO ₂ , mm Hg	44.2 ± 11.4	47.6 ± 12.3	37.8 ± 5.0	- 4.3	< 0.0001†
PO ₂ , mm Hg	66.2 ± 14.1	62.3 ± 13.6	73.8 ± 11.8	4.0	< 0.0001†
Hemoglobin, g/dL	13.9 ± 4.9	13.7 ± 3.5	14.3 ± 7.1	0.52	0.6

*Data are presented as mean ± SD.

†Statistically significant.

tasis. No statistically significant differences were found in the mean values of TLC percentage of predicted, residual volume percentage of predicted, pH, and hemoglobin between groups (Table 1). Twenty-one patients (22.3%) used home oxygen; of these, 20 patients had cystic bronchiectasis.

RV dysfunction occurred in 12 patients (12.8%) and was severe in 3 patients, moderate in 6 patients, and mild in 3 patients; all had cystic bronchiectasis. The mean RV dimension was significantly greater in cystic bronchiectasis patients (31.9 mm) compared with cylindrical bronchiectasis patients (26.7 mm; $p < 0.02$); otherwise, hemodynamic variables were similar in the two groups (Table 2).

PH (SPAP ≥ 40 mm Hg) was found in 31 patients (32.9%), affecting 26 of 62 cystic bronchiectasis patients (41.9%) and 5 of 32 patients (15.6%) with cylindrical bronchiectasis ($\chi^2 = 5.47$, $p = 0.019$). SPAP > 70 mm Hg was found in 12 patients (19.3%) with cystic bronchiectasis and in none of the patients with cylindrical bronchiectasis ($\chi^2 = 5.46$, $p = 0.019$) [Table 3].

LV diastolic dysfunction was present in 11 patients (11.7%), of whom 10 had SPAP > 70 mm

Hg. Concomitant LV systolic dysfunction was noticed in three patients. Eight patients (72.7%) had a flat interventricular septum. All had concomitant RV dysfunction. The mean left atrial diameter in patients with LV diastolic dysfunction was 33.72 ± 1.79 mm, compared to 35.96 ± 4.71 mm in those with PH ($t = 1.53$, $p > 0.05$). There was no relationship between CO and SPAP in all patients ($r = 0.079$) or in patients with cystic bronchiectasis ($r = -0.04$).

There was a significant positive correlation between RV dimensions and SPAP in all patients ($r = 0.74$), while RV dimensions were inversely correlated with PaO₂ values ($r = -0.37$). The variability in RV dimension explained by SPAP was 55%, and by PaO₂ was 14%. The multivariate regression equation for RV function of all patients was as follows: $RV = 22.38 + 0.298 (SPAP) - 0.056 (PaO_2)$ [$R^2 = 0.564$, $p < 0.0001$].

Similarly, a significant positive correlation between RV dimensions and SPAP ($r = 0.74$), as well as a significant negative correlation between RV dimensions and PaO₂ values ($r = -0.32$) were observed in cystic bronchiectasis patients. A proportion

Table 2—Hemodynamic Data of All Bronchiectatic Patients*

Variables	All	Cystic Bronchiectasis	Cylindrical Bronchiectasis	Cystic vs Cylindrical Bronchiectasis	
				t Value	p Value
RA dimension, mm	38.9 ± 11.6	39.8 ± 13.1	37.3 ± 7.4	- 0.09	0.07
RV dimension, mm	30.3 ± 9.5	31.9 ± 10.6	26.7 ± 4.6	- 2.35	0.02†
LV SV, mL	72.4 ± 9.1	72.3 ± 8.7	72.7 ± 9.9	0.15	0.88
LVESD, mm	28.5 ± 6.0	28.8 ± 6.3	27.5 ± 5.0	- 0.83	0.41
LVEDD, mm	47.4 ± 0.04	47.6 ± 6.2	46.8 ± 4.7	- 0.57	0.57
FS, %	1.14 ± 5.9	1.408 ± 6.9	0.418 ± 0.57	- 0.585	0.56
LVEF, %	60 ± 2	59.8 ± 2	60.5 ± 1	- 0.59	0.61

*Data are presented as mean ± SD.

†Statistically significant.

Table 3—Prevalence of PH in All Patients, Cystic Bronchiectasis, and Cylindrical Bronchiectasis*

SPAP, mm Hg	All Patients (n = 94)	Cystic Bronchiectasis (n = 62)	Cylindrical Bronchiectasis (n = 32)
< 40	63 (67.0)	36 (58.1)	27 (84.4)
40–70	19 (20.2)	14 (22.6)	5 (15.6)
> 70	12 (12.8)	12 (19.3)	
Mean ± SD	38.2 ± 21.5	42.8 ± 24.0	29.3 ± 11.3

*Data are presented as No. (%) unless otherwise indicated.

of 54.6% variability in RV values was explained by SPAP and 10% of variability by PaO₂ values (Table 4). The multivariate regression equation for RV function of cystic bronchiectasis patients was as follows: RV = 24.27 + 0.302 (SPAP) – 0.0889 (PaO₂) [R² = 0.559, p < 0.0001].

There was a high statistically significant positive correlation between SPAP values and PaCO₂ (r = 0.42) in cystic bronchiectasis, and the change in SPAP values explained by PaCO₂ values was 18%, which is statistically significant (Table 4). A negative significant correlation was observed between SPAP values and Po₂, percentage of predicted DLCO, and percentage of predicted FEV₁ (r = – 0.28, r = – 0.32, and r = – 0.30, respectively; Table 4). The variability in pulmonary artery pressure values explained by these three variables was 8%, 10%, and 9%, respectively, all of which were statistically significant. The multiple regression equation for SPAP was as follows: pulmonary artery pressure = – 10.91 + 1.109 (PaCO₂) + 0.112 (PaO₂) – 0.098 (percentage of predicted DLCO) + 0.011 (percentage of predicted FEV₁) [R² = 0.34, p < 0.0001]. In contrast, no statistically significant correlation was found between the above parameters in patients with cylindrical bronchiectasis.

DISCUSSION

In this study, patients with intrinsic LV disease were rigorously excluded. We demonstrated that RV systolic dysfunction and PH were more common than LV systolic dysfunction in patients with bronchiectasis. LV

diastolic dysfunction was seen mainly in severe disease. Moreover, PH was more prevalent in cystic bronchiectasis than cylindrical bronchiectasis patients and may indicate deteriorating pulmonary function.

RV systolic dysfunction was observed in 12.8% of the patients and was restricted to those with cystic bronchiectasis. RV systolic function has not been systematically investigated in patients with bronchiectasis. Tomlin et al¹⁷ described chronic cor pulmonale as a complication of fibrocystic disease of the pancreas in 1952. Vizza et al¹⁸ examined RV and LV function in patients with severe airway, parenchymal, and pulmonary vessel disease; the presence of RV dysfunction varied between 59% and 94%. Two other studies^{19,20} of patients with cystic fibrosis showed disparate RV dysfunction rates of 0% and 72%. Koelling et al⁸ observed that RV systolic function at rest and after peak exercise was similarly compromised in 40 patients with advanced cystic fibrosis, as well as in 9 patients with moderately severe bronchiectasis. They also noticed abnormal diastolic function in patients with cystic fibrosis compared with control subjects or patients with bronchiectasis.⁸ In their study, however, patients with cystic fibrosis had greater impairment of pulmonary function than those with bronchiectasis. Although none of our patients was subjected to exercise, we expect more patients in our cohort would exhibit impairment of RV function if they were stressed by exercise.

We also found that RV dimensions were significantly greater in patients with cystic bronchiectasis compared with cylindrical disease (Table 2). More-

Table 4—Simple Regression Analysis and Correlation Coefficients for Cystic Patients

Dependent Variables	Independent Variables	Regression Coefficient	Correlation Coefficient (r)	p Value
RV, mm	SPAP	0.316	0.74	< 0.0001
	Po ₂	– 0.243	– 0.32	0.016
	PCO ₂	0.84	0.42	0.001
SPAP, mm Hg	Po ₂	– 0.49	– 0.28	0.03
	DLCO, %	– 0.29	– 0.32	0.02
	FEV ₁ , %	– 0.331	– 0.3	0.02
CO, L/min	SPAP	– 0.0016	– 0.04	0.77

over, hypoxemia and hypercapnia were seen more frequently in this group of patients (Table 1). Linear regression analysis revealed a close correlation between RV dimensions in cystic disease and both PaO₂, and pulmonary artery pressure (Table 4). These findings suggest the important role of poor gas exchange and PH in the pathophysiology of RV dysfunction, which has been described before in other pulmonary diseases.^{18,21} In our study, RV function was assessed qualitatively; thus, we were unable to calculate its correlation with SPAP. Nevertheless, our study suggests that RV dysfunction is seen in patients with cystic bronchiectasis.

LV systolic function was preserved in all except 3 patients (3.2%), all of whom had LVEF < 45%, while diastolic dysfunction was observed in 11 patients (11.7%). Previous studies in patients with other chronic lung diseases regarded LV dysfunction as rare.¹⁸ Vizza et al¹⁸ reported that LV dysfunction was present in 6% of patients with advanced stage of different pulmonary diseases, with a higher prevalence in patients with PH.

In this study, 11 patients (11.7%) with significant PH had LV diastolic dysfunction, of whom eight also had a flat interventricular septum. Although statistically insignificant, the mean left atrial dimension in patients with LV diastolic dysfunction was lower than in patients with PH. Nevertheless, these findings suggest that abnormal LV performance may be due to ventricular interdependence, in which RV dilatation leads to bulging of the septum into the LV, which in turn increases LVEDD, decreases LV performance, and alters left atrium mechanics. This is in agreement with previous reports^{18,22} on the cause of LV diastolic dysfunction in patients with PH.

We expected a decrease in CO as SPAP increased, particularly in patients with severe PH. However, we found CO was maintained in these patients, and there was no correlation between CO and SPAP. This suggests other factors might be contributing to the increasing venous return in these patients; however, we have no data to support this hypothesis. Similar findings were reported previously in patients with cystic fibrosis and were thought to be related to chronic infection.^{23,24}

Thirty-one of our patients (32.9%) had PH (Table 3). The prevalence of PH has not been determined in patients with bronchiectasis. In this study, 41.9% of patients with cystic disease had PH develop, compared to 15.6% with cylindrical bronchiectasis. The etiology of the observed PH in these patients is potentially multifactorial. Liebow and others⁵ described several pathologic changes in bronchiectasis, including dilatation and hypertrophy of bronchial circulation with extensive bronchopulmonary anastomosis, which can lead to left-to-right shunt. Ashour⁷ expanded this further, with more emphasis on hemo-

dynamic changes. They demonstrated, using right-sided pulmonary angiography, that pulmonary artery flow was absent in the destroyed segment, while there was a retrograde filling of the pulmonary artery through bronchial circulation demonstrated by thoracic aortography. Darke and Lewtas⁶ described this phenomenon as reversal of pulmonary artery flow. These changes were mainly seen in cystic bronchiectasis and were described as *nonperfused segments*.⁷ We believe these hemodynamic alterations increase right-sided afterload due to the contribution of systemic pressure on pulmonary vascular resistance, which further increases the pulmonary artery pressure.

Using multiple regression analysis, we found that increasing SPAP was significantly correlated with declining DLCO percentage of predicted (Table 4). Transfer factor is frequently impaired in patients with interstitial disease and PH,^{25,26} which may suggest destruction of the pulmonary microvessels. This negative correlation was seen in cystic disease, whereas no correlation was found in cylindrical bronchiectasis, suggesting that the latter disease has a more favorable course.

SPAP correlated negatively with FEV₁ in patients with cystic bronchiectasis (Table 4). A previous study²⁷ on bronchiectasis emphasized that obstructive pulmonary insufficiency was related to morphologic changes and bronchial responsiveness. Obliterative bronchiolitis of small and medium airways together with secretions and associated emphysematous changes all contribute to pulmonary function deterioration associated with obstructive disease.^{28,29} Sustained hypoxia has been implicated in the pathogenesis of PH in animals and humans,^{30,31} and was found to be an important stimulus for vascular remodeling resulting in vasoconstriction, which in turn leads to an increase in pressure and wall stress.³² We found a correlation between high SPAP and low PaO₂ and high PaCO₂ (Table 4), suggesting a consequence of poor gas exchange on SPAP. The above findings of deteriorating pulmonary physiology, particularly in patients with cystic bronchiectasis, have an important role in the pathogenesis of PH and suggest that PH is a marker of lung damage in these patients.

Limitations of the Study

The estimation of LVEF was not corroborated by an independent method, such as radionuclide ventriculography. However, the ability of the echo to detect LVEF has been extensively validated before.¹⁶ Accurate assessment of RA and RV dimensions by two-dimensional echocardiography may be limited in those patients with hyperinflated lungs. RV ejection fraction was assessed based on qualitative method and, although a number of techniques exist for accurate quantitation, direct calculation of

RV volumes and ejection fraction remains problematic given the complex geometry of the RV and the lack of standard methods of assessment. CO was measured using continuity of equation method but can sometimes overestimate CO, and optimal results require careful data acquisition. This cross-sectional study does not allow us to determine the outcome in our patients, particularly in those who had PH. Future studies may focus in this point.

In summary, this study systemically assessed cardiac function and SPAP in a large population of adult patients with bronchiectasis. Our findings showed that RV systolic dysfunction and PH were more common than LV systolic dysfunction in adult patients with bronchiectasis. LV diastolic dysfunction was seen mainly in severe disease. PH was seen more frequently in cystic bronchiectasis, and correlated with deteriorating pulmonary physiology; therefore, it can be a marker of lung damage in bronchiectatic patients. In view of the above findings, we recommend detailed cardiac assessment of patients with bronchiectasis, particularly in those with cystic disease and PH, with more emphasis on LV diastolic function.

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