

Pulmonary Vascular Disease, continued

21 and 40 mm Hg and > 40 mm Hg are consistent with Mild-moderate and severe pulmonary hypertension respectively.

METHODS: Retrospective review of 2D Echo and CT Scan results of 30 patients referred to pulmonary service over one year period for evaluation of parenchymal lung disease who were found to have pulmonary hypertension based on 2D Echo results. We measured the Mean Pulmonary Artery Diameter (MPAD) on the CT Scan at the widest portion of the main pulmonary artery within 3 cm of the bifurcation.

RESULTS: In our study we found that patients with 2D Echo PAP between 25-40 mm Hg graded as mild-moderate PH have CT determined mean MPAD 34 mm(SD =6.57) and those with PAP >40 mm Hg graded as severe PH have mean MPAD 48.5 mm(SD =7.45). There is statistically significant difference between the above two categories based on the Two-Sample T-Test (P-Value 0.001). Also we noted a linear correlation between the MPAD on CT Scan and PAP as measured by 2D Echo (P-Value 0.002).

CONCLUSION: CT Scan, a non invasive and readily available modality, is frequently used to evaluate chest pathology. Our study demonstrates that CT scan measurement of main pulmonary artery diameter (MPAD) can be a useful tool for detection and grading the severity of pulmonary hypertension.

CLINICAL IMPLICATIONS: Utility of CT scan for detection and grading the severity of pulmonary hypertension in patients with parenchymal lung disease.

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BODY MASS INDEX AND RISK FOR PULMONARY HYPERTENSION (PH)

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PURPOSE: PH has a multifactorial etiology and is a major cause of morbidity and mortality among patients. Obstructive sleep apnea (OSA) is a possible contributor for PH, but there have been no conclusive studies on BMI alone causing PH. We performed a retrospective study on the relationship of BMI and PH.

METHODS: Medical records of patients who had sleep studies in our institution over the past one year were reviewed. Patients were divided into two groups based on BMI (patients who are morbidly obese and patients who are not morbidly obese). Two-dimensional Echocardiography reports of mean pulmonary artery pressure (PAP) were obtained, together with other data like age, sex, race, co morbidities, respiratory disturbance index (RDI), Epworth sleepiness scale (ESS), and neck circumference.

RESULTS: The mean BMI of patients belonging to the morbidly obese group is 49.72 (SD 4.83), with mean PAP of 30.62 (SD 11.79), and mean RDI of 21.39 (SD 27.17). For the other group the mean BMI is 33.46 (SD 5.07), with mean PAP of 28.2 (SD 4.71), and mean RDI of 63.52 (SD 44.06). Using one-way ANOVA, there is no statistically significant difference in the MPAP among the two groups, with a p-value of 0.663. However, there is a statistically significant correlation between BMI and RDI of our patients with p-value of 0.008. There is also a statistically significant correlation between neck circumference and RDI among our patients with p-value of 0.037.

CONCLUSION: Our study indicates that there is no statistically significant correlation between BMI and MPAP. Further investigation, in the form of a prospective, case-control study, is needed to determine if BMI alone has a causative effect on PH. Our study also shows that there is linear correlation between RDI and BMI, and neck circumference and BMI.

CLINICAL IMPLICATIONS: Body mass index (BMI) and risk for pulmonary hypertension (PH).

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SCREENING FOR PULMONARY ARTERIAL HYPERTENSION WITH EXERCISE-STRESS-ECHOCARDIOGRAPHY AND CARDIOPULMONARY EXERCISE TESTING

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PURPOSE: In the early stages of Pulmonary Arterial Hypertension (PAH), the pulmonary arterial pressure may be normal at rest, but elevated during exercise. Exercise-Stress-Echocardiography (EE) may detect these abnormal reactions.

METHODS: We examined patients with a risk factor for PAH with EE and Cardiopulmonary Exercise Testing (CPET), and we determined the systolic PAP at rest (rSPAP) and during exercise (exSPAP). Right Heart Catheterisation (RHC) and the measurement of the mean PAP (mPAP) was recommended for patients with an abnormal EE result (exSPAP>40mmHg) and for patients with a VO₂ peak<75% predicted. The results of the EE and RHC examinations were compared. Patients with known PAH were excluded.

RESULTS: 60 patients with Systemic Sclerosis, Systemic Lupus Erythematoses and liver cirrhosis were examined. rSPAP was elevated in 1 patient (54mmHg) (RHC mPAP: 27mmHg), 27 patients had normal rSPAP (27±5mmHg), and abnormal exSPAP (54±9mmHg). 18 of these patients were examined by RHC. 1 patient had PAH at rest (mPAP: 27mmHg), 10 had an exercise-induced PAH (mPAP at rest: 17±3mmHg, ex mPAP: 36±4mmHg). 6 patients had an exercise induced PH with pulmonary capillary wedge pressure (PAWP) elevation (PAWP at rest: 7±2mmHg, ex PAWP: 32±10mmHg), and 1 patient had normal SPAP at rest and during exercise. EE showed normal values in 32 patients at rest and during exercise (rSPAP: 23±3mmHg, exSPAP: 29±7mmHg). Out of these, 6 patients underwent RHC because of limited exercise capacity. 4 of them had an exercise-induced PAH (mPAP at rest: 18±4mmHg, ex mPAP: 39±7mmHg), 2 of them had normal values.

CONCLUSION: In patients with a risk factor for PAH, EE showed an abnormal reaction in 47%. At the given indication, the positive predictive value of EE for exercise-induced PAH is 95%, the negative predictive value 33%.

CLINICAL IMPLICATIONS: EE combined with CPET might be a suitable method to diagnose PAH in the very early stage of the disease.

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TREATMENT OF PAH: DATA FROM THE QUALITY ENHANCEMENT RESEARCH INITIATIVE

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PURPOSE: Implementation of the 2004 ACCP Recommendations for the Management of Pulmonary Arterial Hypertension (PAH) was studied in quality enhancement research initiative (QuERI) in PAH 517 patients among 52 US specialists.

METHODS: Physicians were asked to enroll PAH patients (known or newly diagnosed) and provide data on their medical management.

RESULTS: PAH was idiopathic in 37%, familial in 3%, had associated conditions in 50%: CTD in 28%, drug exposure in 9%, shunt in 7%, portal hypertension in 4%, HIV in 3% and venous/capillary involvement in <1%. WHO class was available in 471 patients: 9% were class I, 39% class II, 47% class III, and 5% class IV. Data on disease-specific treatments was available in 450 patients (87%). Epoprostenol/inhaled iloprost/treprostinil (PGI) was used in 33.7% overall and alone in 8.0%. Sildenafil (SID) was used in 37.3% overall and alone in 8.8%. Bosentan (BOS) was used in 53.1% and alone in 26.0%. Combination therapy was PGI + SID in 16.9%; BOS + SID in 18.2%; PGI + BOS in 15.6%; and PGI + BOS + SID in 6.7%. 19.8% were on no therapy. All of these therapies were more frequently used as symptom class worsened. Among 96 patients on CCB, only 27 had acute vasoreactivity testing performed; only 6 of these were reported to have ACCP-defined vasoreactivity (22% of those tested for vasoreactivity and only 6.3% of the total population). Recommended PAH therapy of anticoagulants (warfarin) was used in 38% of class II, 52% of class III and 44% of class IV (44% overall). Diuretics were used in 40%, 53%, and 72% of class II, III, and IV, respectively. Modulators of renin system were used in 21% of patients and digoxin in 16%.

Pulmonary Vascular Disease, continued

CONCLUSION: Medical management of PAH is complex with frequent use of multiple therapies. Disease specific therapies and diuretics are used more frequently as symptoms worsened. Vasoreactivity testing to direct CCB treatment was not performed in the majority of patients.

CLINICAL IMPLICATIONS: Greater adherence to ACCP guidelines for vasoreactivity testing is recommended.

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PULMONARY HYPERTENSION AND VALVULAR HEART DISEASE: INCIDENCE AND IMMEDIATE IMPACT OF SURGICAL REPAIR

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PURPOSE: Mitral and aortic valve diseases can cause secondary pulmonary hypertension (PH). Initially, PH is due to increased left atrial pressure but with time pulmonary vascular remodeling can occur. The former should rapidly improve with surgery; the latter would be more likely persist. We initiated this study to assess the frequency of and changes in PH in patients with aortic (AVR) versus mitral valve (MVR)-repair both immediately before and 24 hours after surgery.

METHODS: Data were retrospectively collected from both preop cardiac cath or from perioperative right heart cath. Three patients with AVR, one with MVR and one with combined AVR/MVR were excluded from analysis for lack of hemodynamic data. PH was defined as a mean PA pressure (mPAP) of 25 mmHg or higher.

RESULTS: Of 76 charts studied, 41 had AVR (51% F; mean age 76 yo), 30 MVR (47% F; 75 yo) and 5 had repair of both valves. In the AVR group, 61% had preop PH; PA pressures preop averaged 45/19 (29) mmHg with CI=2.1 L/min/m² and LVEDP 22 mmHg. 24h after AVR mPAP fell 28%, CI rose 33% but 15% had persistent PH. In the MVR group 80% had preop PH; preop PA pressure were 53/22 (34) mmHg with CI=2.1 and LVEDP = 19 mmHg. By 24h after MVR, mPAP fell by 18% and CI rose 29% but 66% had persistent PH. In the combined AVR/MVR group 80% had PH; average PA pressures were 52/23 (34) mmHg and CI = 2.4.

CONCLUSION: PH is a common complication of valvular heart disease, being slightly more frequent with mitral than with aortic disease. The fall in PA pressures within 24 h of surgery was more dramatic after AVR, perhaps because of higher left ventricular pressures and less pulmonary vascular remodeling.

CLINICAL IMPLICATIONS: Although elevated PA pressures often fall following valve repair, patients with PH and aortic or mitral valve surgery should be evaluated at regular intervals to determine if fixed remodeling has occurred.

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PULMONARY HYPERTENSION: A MANITOBA PERSPECTIVE

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PURPOSE: To report on the patient population, classification, treatment options, and prognosis of pulmonary hypertension in Manitoba.

METHODS: Prospective study following a group of patients in Manitoba diagnosed with pulmonary artery hypertension from December 2000 to December 2005. Diagnosis of pulmonary artery hypertension was determined by right heart catheterization as defined by the National Institute of Health as mean pulmonary artery pressure of greater than 25 mmHg at rest or greater than 30 mmHg with exercise. Patients were followed with pulmonary function testing and six-minute walk tests; all medications were recorded. Clinical severity was graded according to the World Health Organization functional classification.

RESULTS: In December 2000 there were 60 live patients enrolled, in December 2005 there were 48 live patients with pulmonary hypertension in the registry. Ten were male with a mean age of 59 years +/- 9.86 and 38 were females with a mean age of 52.3 +/- 16.8 years. Sub-diagnosis was as follows: 17 cases were idiopathic pulmonary hypertension, 20 cases were related to collagen vascular disease, 3 cases were related to congenital pulmonary shunts, and 4 were related to chronic thromboembolic disease. Fourteen patients were classified as WHO functional class III, and 7 were WHO functional class IV. Thirty-four patients were being treated with Bosentan, and 3 patients were treated with Flolan, 5 patients were on combination therapy.

CONCLUSION: Pulmonary artery hypertension is a serious disease with significant burden of illness and mortality in Manitoba. Currently there are 70 patients in the Manitoba database.

CLINICAL IMPLICATIONS: The results from this study will provide the necessary framework upon which further studies can be launched. These include the effectiveness of specific and combination therapies in treatment of idiopathic disease and other subtypes.

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CORRELATION OF PULMONARY FUNCTION VARIABLES WITH HEMODYNAMIC MEASUREMENTS IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION

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PURPOSE: A reduced diffusing capacity (DLCO) measured during a pulmonary function test (PFT) can suggest pulmonary arterial hypertension (PAH). The DLCO has been reported to weakly correlate with pulmonary pressures. We hypothesized that the DLCO "normalized" to spirometric variances in patients would correlate more strongly (than DLCO alone) with pulmonary pressures.

METHODS: DESIGN: Retrospective chart review. SUBJECTS: Patients who underwent pulmonary arterial catheterization (PAC) for the evaluation of suspected PAH. SETTING: Tertiary referral academic center (Mayo Clinic, Jacksonville) between 1991-2006. DATAPOINTS: Invasive pulmonary arterial hemodynamic measurements, clinical diagnoses, and pulmonary function test variables. STATISTICS: Nonparametric correlation (Spearman), tests of 2-sided statistical significance, and correlation plots were generated using GraphPad Prism.

RESULTS: 241 patients who under PAC for the evaluation of suspected PAH were identified. Of these, 208 patients also had PFT performed. No significant correlation was identified between the mean pulmonary artery pressure (mPAP) or the pulmonary vascular resistance (PVR), against any of the PFT variables; including the DLCO alone or the DLCO normalized to the forced expiratory volume in 1 second, the forced vital capacity, and the total lung capacity (i.e. DLCO%/FEV1%, DLCO%/FVC%, DLCO%/TLC%). Subgroups were separated further into standard definitions of restrictive (TLC<80%), obstructive (FEV1/FVC<70%), or mixed obstructive-restrictive ventilatory defects. Again, no significant correlation could be identified between PFT variables and PAC hemodynamic measurements. Finally, the subjects were subgrouped based on the clinical diagnosis for the cause of the patients PAH (including collagen-vascular disease, idiopathic PAH, chronic thromboembolic disease, cardiac disease not due to congenital defects, portopulmonary hypertension, and intrinsic lung disease). Within each clinical categorization, no significant correlation was identified between any PFT variable and hemodynamic measurements of pulmonary pressures.

CONCLUSION: In patients with suspected PAH, invasive hemodynamic measurements of PAH do not correlate with PFT variables, regardless of the subgroup of ventilatory physiology or clinical diagnoses.

CLINICAL IMPLICATIONS: Despite known effects of pulmonary vascular disease on the diffusing capacity, PFT variables should not be used clinically to exclude or suggest the presence of PAH.

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WORSENING SYMPTOMS OF PULMONARY HYPERTENSION AFTER CLOSURE OF AN INTERATRIAL SHUNT

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PURPOSE: Systemic to pulmonary shunts such as patent foramen ovales (PFO) and atrial septal defects (ASD) are associated with cryptogenic stroke, migraine headaches, and arrhythmias. Increasingly percutaneous closure of these lesions with an atrial septal closure device (ASCD) is being performed. We present two cases where closure of an interatrial defect was performed in patients with pulmonary arterial hypertension (PAH), resulting in increased morbidity.

METHODS: Retrospective chart review of 183 cases of PAH followed at our institution. Two cases with a history of ASCD were identified and reported.

RESULTS: Case 1: AB is a 38 year-old female with hereditary hemorrhagic telangiectasia who presented with cerebrovascular accident. A PFO was discovered and percutaneously closed. Since, she reported