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ECHOCARDIOGRAPHY MEASUREMENT OF PULMONARY HYPERTENSION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASES

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Abstract
Some people with COPD develop pulmonary hypertension (PHT), which is a prevalent condition in and of itself. Its presence is linked to poorer clinical progression and shorter survival times. The purpose of this study was to use echocardiography to evaluate pulmonary hypertension (PHT) and right ventricular size in patients with chronic obstructive pulmonary disease (COPD) and to determine whether or not these variables were associated with disease severity. A total of sixty COPD patients were chosen from the medical ward of Al-Yarmouk Teaching Hospital. They were categorized according to the severity of their condition using the GOLD classification system. After that, they had echocardiography to assess the size of their right ventricle and the estimated systolic pressure of their pulmonary arteries. The results showed that out of the total number of patients, 20 (33.3%) had no PHT, 10 (16.7%) had mild PHT (ePPASP 36–45 mmHg), 21 (35.0%) had moderate PHT (ePPASP 46–60 mmHg), and 9 (15.0%) had severe PHT (ePPASP >60 mmHg). In moderate COPI, the frequency of PHT was 57.1%; in severe COPI, it was 65%; and in very severe COPI, it was 91.7%. Among the patients with IL, 18.3% had enlarged right ventricles. Hemodynamic alterations on the right side of the heart, including RV enlargement and PHT, are strongly associated with the severity of chronic obstructive pulmonary disease (COPD) as classified by GOLD. It is worthwhile to search for PHT in this cohort because it is frequently found in COPD patients.

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Aim of the study The research aims to evaluate the relationship between the severity of chronic obstructive pulmonary disease (COPD) and echocardiographic measurements of the right ventricle size and pulmonary arterial pressure.

Keywords: Chronic Obstructive Pulmonary Diseases, Pulmonary Hypertension, Echocardiography.

INTRODUCTION

When airflow restriction is present and cannot be completely reversed, the condition is known as chronic obstructive pulmonary disease (COPD). In cases of chronic obstructive pulmonary disease (COPD), the airflow restriction is typically severe and accompanied by an aberrant inflammatory reaction of the lungs to harmful substances. Chronic obstructive pulmonary disease (COPD) is a catch-all term for two conditions: emphysema and chronic bronchitis. After other potential causes of chronic cough (such as bronchiectasis) have been ruled out, a patient may be diagnosed with chronic bronchitis if they experience a productive cough for at least three months twice in a row for at least two years. This condition may occur before or after the onset of airflow limitation. and The chronic obstructive pulmonary disease known as emphysema is characterized by the progressive narrowing and eventual collapse of the airways that lie beyond the terminal bronchioles. People without airflow blockage can still develop emphysema, although those with moderate to severe airflow obstruction are more likely to have it [3].

• Medical research One global health concern that is gaining prominence is chronic obstructive pulmonary disease (COPD). Worldwide, chronic obstructive pulmonary disease (COPD) is expected to jump from sixth to third place in terms of mortality rate by several factors, such as the time it takes to diagnose COPD, the difficulty in agreeing on a single definition, and the absence of age-adjusted figures, suggest that the global prevalence is probably underreported. The global prevalence of chronic obstructive pulmonary disease (COPD) (stage I or above) was 1.8% in men and 8.5% in women, according to the Burden of Obstructive Lung Disease (BOLD) research. Site and sex differences in smoking prevalence account for the observed differences in PMT. The disparity has shrunk as a result of the women's rising smoking rate. Covid-19 may be more common among women, according to some research. PI Potential cause Tobacco use is the leading cause of chronic obstructive pulmonary disease (COPD) globally. A person's lifetime exposure to inhaled particles is inversely proportional to their risk of chronic obstructive pulmonary disease (COPD): • Cigarette smoking: There is no better-known risk factor for chronic bronchitis and emphysema than smoking cigarettes. A smoking history is present in 80-90% of COPD patients, and one out of every five smokers will acquire COPD [3]. Generalized exposure to dust and fumes while working is known as occupational exposure. Coal mining, gold mining, cadmium exposure, and cotton textile dust are some of the specific occupational exposures that have been linked to chronic airflow blockage. Prolonged exposure to smoke from biomass combustion, a prevalent cooking method in some countries, also seems to be a substantial risk factor for chronic obstructive pulmonary disease (COPD) among women living in those nations. • The total burden of inhaled particles in the lungs is also increased by outdoor air pollution, but it seems to have a very limited

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influence in producing chronic obstructive pulmonary disease (COPD) [21]. • Approximately 1% of COPD patients are discovered to have severe (Il AT deficiency) as a contributory cause of the disease. This illness causes an imbalance of proteases and antiproteases, as well as the unopposed action of neutrophil elastases. As a result of its symptoms, COPD is Pulmonary hypertension is the leading cause of lung disease. The term "pulmonary hypertension" (PMT) refers to a condition in which the right cardiac catheter measures a rise in resting mean pulmonary arterial pressure (PAP) of more than 225 mm Hg. A worse prognosis and shorter survival time are linked to pulmonary hypertension (PH T). Mild to moderate pulmonary hypertension develops gradually in chronic obstructive pulmonary disease (COPD). Nevertheless, pulmonary arterial pressure might temporarily rise during exacerbations, physical activity, and sleep. While maintaining cardiac output, right ventricular function is only slightly affected [14, 15, 16]. Chronic obstructive pulmonary disease (COPD) worsens dramatically when secondary pulmonary hypertension develops. In addition to increasing healthcare expenditures, PHT has been linked to a higher risk of severe acute exacerbation [171]. PHT as a result of chronic obstructive pulmonary disease (COPD) is a mortality predictor that is unrelated to the severity of pulmonary obstruction (114'151). One study found that patients admitted with COPD exacerbation who had PHT had an increased risk of 1-year mortality [151], while another found that patients with COPD who had PHT had a 37% 5-year survival rate compared to 63% without PHT [161]. The risk of chronic obstructive pulmonary disease (COPD) worsening as a result of secondary pulmonary hypertension (PHT) is dose-dependent. Consequently, the 5-year survival rate is 15% for patients with severe PHT, compared to 55% for those with mild to moderate PHT [181]. The administration of COPD medication may potentially be affected by PH. When there is a lot of PHT, people usually start thinking about getting a lung transplant. Lung volume reduction surgery for advanced emphysema is contraindicated in patients with severe PHT [191]. Optimal treatment for these individuals may be possible with PHT assessment in light of the aforementioned research on PHT's effect on COPD progression and prognosis. Physiology of disease: Pulmonary hypertension develops as a result of several processes, including endothelial dysfunction, vascular remodeling, and inflammation. It has just recently been discovered that heredity could be a factor: I. Respiratory Blood Clots: The protective mechanism of hypoxic constriction of the small muscular pulmonary arteries is to redirect blood flow from poorly ventilated alveoli to those that are better supplied with oxygen. This helps to decrease the ventilation-perfusion mismatch [202 1 1]. In severe chronic obstructive pulmonary disease (COPD), for example, widespread alveolar hypoxia leads to pulmonary vasoconstriction and an increase in PVR. Pulmonary vascular remodeling, which is a result of chronic hypoxia, raises the PVR1221. Redesigning the Vasculature of the Lungs Transient intimal fibrosis, proliferation of longitudinal smooth muscle in the muscular pulmonary arteries and arterioles, and neomuscularization of pulmonary arterioles are hallmarks of vascular remodeling in chronic obstructive pulmonary disease (COPD) patients [2324]. Smokers without airway blockage and individuals with moderate chronic obstructive pulmonary disease (COPD) who do not have hypoxia also have these pulmonary vascular alterations. This provides more evidence that hypoxia is not the only significant factor in the pathophysiology of vascular remodeling. A pulmonary hyperinflation score of 5–7.5 cm (1120–1261), accompanied by air trapping and hyperinflation, is indicative of severe emphysema. During breathing, positive

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alveolar pressure raises PAWP and PAP127•28 1, which in turn causes a high PVR. Patients with severe emphysema who are not hypoxemic and during exercise: this mechanism may play a more significant role in the development of PCI. The malfunctioning of endothelial cells and inflammation: Restraining vascular tone, regulating hypoxic vasoconstriction, and directing the mechanism of vessel adaptation under situations of increased blood flow are the three primary roles of endothelial cells in pulmonary vessels [291]. Exhaled nitric oxide (NO) is lower in chronic obstructive pulmonary disease (COPD) patients with pulmonary hypertension (PH) compared to those without PH because of endothelial dysfunction, which is defined by diminished NO synthesis and/or release [301]. Vasodilatation due to endothelial-produced chemicals is decreased in chronic obstructive pulmonary disease (COPD) patients compared to healthy controls [311]. There is evidence that low-grade systemic inflammation plays a part in the development of pulmonary hypertension in people with chronic obstructive pulmonary disease (COPD). This is because high mPAP levels are linked to high levels of C-reactive protein and tumor necrosis factor-u in the blood [321]. Five, polycythemia A lack of NO in the area, which could be because hemoglobin 351 removes too much NO from the pulmonary circulation, makes hypoxic pulmonary vasoconstriction worse, and polycythemia makes the blood thicker and harder to flow through the pulmonary circulation [33]. 6. Genetic predisposition: The link between a change in the serotonin transporter gene and pulmonary hypertension in people with chronic obstructive pulmonary disease (COPD) showed that genetic predisposition plays a role in the development of PHT [36]. Hyperplasia of smooth muscle fiber in the pulmonary arteries is one way in which the serotonin transporter contributes to the development of PHT [361]. Echocardiography is the initial screening tool for pulmonary hypertension (PMT) in chronic obstructive pulmonary disease (COPD) patients because it can also detect the right ventricular effects of PH on the heart. With a specificity of 720/0 and a sensitivity of 83%, echocardiography had a modest diagnostic accuracy when it came to pulmonary hypertension. The absence of a high pulmonary systolic pressure (PASP) on echocardiography rules out potentially life-threatening pulmonary hypertension (PHT) and the need for additional intrusive testing. [36] Chronic obstructive pulmonary disease (COPD) often causes moderate pulmonary hypertension (PHT), although a small percentage People with pulmonary hypertension are put into three groups based on their estimated peak pulmonary arterial systolic pressure (ePPASP): mild (366–45 mmHg), moderate (46–60 mmHg), and severe (mmHg) [38, 39]. Patients with severe PH T have unique symptoms, a worse prognosis, and require a tailored treatment plan. Despite this, cardiac catheterization is still considered the "gold standard" for measuring pulmonary arterial pressures; however, there are major risks [40], financial concerns, and the fact that it would not be practical to conduct the procedure on every patient with moderate to severe chronic obstructive pulmonary disease (COPD). The method also isn't ideal for the kind of close-quarters monitoring that's necessary for clinical trials of treatments for secondary PHT. Therefore, it is evident that there would be significant therapeutic and research utility in developing a noninvasive method of assessing pulmonary arterial pressure that is dependable and reproducible.Table 1. Key Indicators for Considering a Diagnosis of COP D

Over 40-year-olds are more likely to have chronic obstructive pulmonary disease (COPD) if they experience any of these symptoms. Having said that, these signs alone cannot diagnose anything. Spirometry is a diagnostic tool for chronic obstructive pulmonary disease (COPD). Progressive

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dyspnea is characterized by progressively worsening symptoms, which are at their worst during physical activity. • A dry, hacking cough that flares up and goes away at random intervals. Persistent coughing or sputum production is a sign of chronic obstructive pulmonary disease (COPD). • There is a history of exposure to potentially dangerous airborne particles, such as cigarette smoke, smoke from home appliances, industrial chemicals, and dust. One or more family members have a history of COPD. Spirometry is an essential diagnostic tool for chronic obstructive pulmonary disease (COPD). An FEVI/FVC value below 0.70 after bronchodilator administration indicates persistent airflow limitation, which translates to chronic obstructive pulmonary disease (COPD). According to the GOLD staging approach developed by the Joint International Program on Chronic Obstructive Pulmonary Disease, individuals with chronic obstructive pulmonary disease are grouped according to the degree of their airway limitation (Table 2). Classification of COPD Severity (Table 2) Bronchodilator aftereffects The following people have FEVI/FVC ratios lower than 0.70: the five GOLD: In GOLD 2, we anticipate a modest FEVI of 50%. For GOLD 3, we anticipate an extremely high FEVI of 30%. Very In GOLD, severe FEVI is anticipated to be below 30%. GOLD is a worldwide group addressing chronic obstructive pulmonary disease.

Patients and Methods

As part of their research plan, Al Yarmouk Teaching Hospital did a descriptive analytics study from January to July 2015. Choices made by patients: Al-Yarmouk Teaching Hospital is home to sixty individuals suffering from chronic obstructive pulmonary disease. Choosing factors A diagnosis of chronic obstructive pulmonary disease is required for participation in this study: Firstly, the patient's medical history and physical examination provide the following information: People who smoke and are over the age of 40 often experience chest pain with reduced or nonexistent expansion of the chest, liver and heart dullness, a greater distance between the front and back of the chest, or a barrel shape to the chest. In a chest X-ray, I see obvious lung fields; in a second, a space lower than the sternum that exceeds 4.5 cm; in a third, diaphragms that are flat or low below the seventh rib in the front; and in a fourth, border narrowing of blood vessels. A positive result for both FEVI and FVC can lead to the diagnosis of chronic obstructive pulmonary disease (COPD) through pulmonary function testing. When diagnosing emphysema, high-resolution chest computed tomography (CT) scans and positron emission tomography (PET) are very helpful. A subset of patients underwent the high-resolution computed tomography (HRCT) scan primarily to detect emphysematous alterations. All of the chosen patients had conventional testing, which included complete blood counts, lipid panels, glucose, urea, serum creatinine, electrocardiograms, and echocardiographies. Inclusion requirements Participants in the study were not eligible if they met any of the following exclusion criteria: Blockage My health is inconsistent; I struggle with left-sided heart failure, asthma, and bronchiectasis. cardiac issues. This patient has a narrow echo window due to their chronic obstructive lung illness. Researchers found individuals with chronic obstructive pulmonary disease who were unable to undergo spirometry. The severity of airflow limitation is used to classify COPD patients as mild, moderate, severe, or very severe according to the GOLD staging technique (Table 2). Resting two-dimensional transthoracic Doppler echocardiography was used to check on all patients at the cardiology department of AL-

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Yarmouk teaching hospital. An instrument called a VIVID E9 XDcIear with a multifrequency probe was used, which was capable of 2D, M-Mode, and color flow modes. We conducted experiments using Doppler. To rule out pulmonary hypertension and right-sided heart illness, we utilized echocardiography. We determined the maximum jet velocity using color flow and confirmed it with continuous wave Doppler. We observed the tricuspid regurgitation flow using Doppler. Assuming unimpeded right ventricular outflow, the modified Bernoulli equation can be used to compute the right ventricular systolic pressure: One way to find the pressure is to add 4v2 to RVP, where v is the peak trivelocity of cuspid regurgitation in m/s and PPASP is millimeters of mercury. Look at the inferior vena cava diameters and how they changed with inspiration to see how RAP baseline values were calculated: 5–15 mmHg. With no collapse whatsoever, the RAP is 15 mmHg, a partial collapse is 10 mmHg, and a full collapse is 5 mmHg. Researchers in this study used PPASP estimates of 36 mmHg (38•39) to define pulmonary hypertension (PHT). When the PPASP value was between 36 and 45 mmHg, 46 to 60 mmHg, or more than 60 mmHg, it was used to classify PHT as mild, moderate, or severe [38, 39]. In this investigation, we used M-mode echo to measure the right ventricle's size. For the right ventricle to be present, the mid-cavity diameter needed to exceed 35 mm.

Analyzing statistical data:

We used SPSS (Statistical Packages for the Social Sciences), version 22, to examine the data. Standard deviation, range (from lowest to highest), percentage, mean, and frequency were essential metrics used for data visualization. We used the analysis of variance (ANOVA) test to make sure that there were significant variations between more than two quantitative means. We used the Pearson Chi-square test (12-test) with Yate's correction and the Fisher exact test to see if there was statistical significance in the qualitative data. We went ahead and assumed statistical significance if the P-value was less than 0.05.

Results

Six patients (10%) were younger than 60 years old, forty-nine patients (65%) were between the ages of 60 and 69, and fifteen patients (25%) were older than 70. Of the sixty COPD patients included in our study, forty-nine (81.7%) were male and one patient (18.3%) was female.

According to the data in table (3), 12 patients, or 20% of the total, are current smokers, whereas 48 patients, or 80%, were previously smokers. Table 3 displays the sample demographics and socioeconomic status. What on earth? The frequency of PH was 57.1% in moderate COPD, 65% in severe COPD, and 91.7% in very severe COPD,

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Table 3: Demographic and social characteristics of study sample:

		No	%
Age (years)	<60	6	10.0
	60---64	24	40.0
	65---69	15	25.0
	=>70	15	25.0
Gender	Male	49	81.7
	Female	11	18.3
Smoking status	Current smoker	12	20.0
	X-Smoker	48	80.0

Based upon Global Initiative for Chronic Obstmctive Lung Disease (GOLD) classification , patients were classified as having moderate (50% FEVI < 80% predicted was seen in 28 patients (46.7%), severe (30% FEVI < 50% predicted was seen in 20 and very severe (FEVI< 30% predicted was seen in 12 patients(20%) of COPD as shown in the table (4).

Table 4: Categorization of study sample according to the PFT.

	% predicted	No	%
Severity of COPD according to GOLD classification	Moderate	28	46.7
	Severe	20	33.3
	Very severe	12	20.0

On echocardiography PHT defined as estimated PPASP > 35 mmHg.,It has been found that 20 patients (33.3%) had no PHT, 10 patients (16.7%) had mild PHT(ePPASP 36-45 mmHg), 21 patients (35.0%) had moderate PHT (ePPASP 46—60 mmHg) and 9 patients(15.0%) had severe PHT (ePPASP >60 mmHg).

Right ventricular (RV) enlargement was observed in I I patients (18.3%) as shown in the table (5).

Table 5: Classification of the patients according to estimated PASP, RV size and severity of PIIT.

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		No	%
Echo RV size	Enlarged	11	18.3
	Normal	49	81.7
Severity of PHT	No	20	33.3
	Mild	10	16.7
	Moderate	21	35.0
	Severe	9	15.0

Table (6) shows that I l patients (18.3%) had enlarged right ventricular size, out of those I l patients 2 patients (18. I had severe COPD and 9 patients (81.9%) had very severe COP D, thus there is significant correlation between severity of COPD and RV size (p value -0.0001).

Table 6: correlation between severity of COPD and right ventricular size by echocardiography.

		Gold classification						P value
		Moderate		Severe		Very severe		
		No	%	No	%	No	%	
Echo RV size findings	Enlarged	-	-	2	18.1	9	81.9	0.0001*
	Normal	28	57.1	18	36.8	3	6.1	

*Significant difference between proportions using Pearson Chi-square test at 0.05

Table (7) shows that frequencies of PH in moderate, severe, and very severe COPD were 57.1%, 65% and 91 .7%, respectively; thus we can see that there is a good co-relation between the frequency of PH and severity of COPD. Table 7: Frequency of PHT with severity of COPD

Number of patients according to Severity of COPD	Number and percentage % of patients with PHT	P value
Moderate (28)	16 = 57.1%	0.039*
Severe (20)	13 = 65.0%	
Very severe (12)	11 = 91.7%	

*Significant difference between proportions using Pearson Chi-square test at 0.05

In table (8) which show mean and standard deviation of (PHT) in relation with severity of COPD according to GOLD classification, there is a significant correlation between severity of (PI-IT) and severity of COPD (P

Table (8): Mean of estimated PASP by echocardiography and severity of COPD according to GOLD classification.

	Gold classification			P value
	Moderate Mean+SD (Range)	Severe Mean+SD (Range)	Very severe Mean+SD (Range)	
FEV1% Predicted value	54.11±3.61 (51.0-63.0)	40.55±4.70 (31.0-49.0)	28.00±1.58 (24.0-29.0)	0.0001 *
estimated PPASP by echocardiography	29.33±2.89 (24.0-34.0)	42.76±5.86 (34.0-54.0)	59.89±3.26 (55.0-65.0)	0.0001 *

*Significant difference among three independent means using ANOVA test at 0.05 level.

Discussion

We categorized the chronic obstructive pulmonary disease (COPD) patients in this study into four severity levels: mild, moderate, severe, and very severe. None of the patients had mild COPD since we only included patients from the medical ward; we don't think patients with mild COPD should be hospitalized. All of the patients had a bronchodilator therapy (nebulized salbutamol) ratio of less than 70%, and none of them showed any signs of reversibility after following a predetermined protocol. There were 28 patients with moderate COPD (46.7%), 20 with severe COPD (33.3%),

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and 12 with extremely severe COPD (20%). This investigation detected pulmonary hypertension (PHT) in 40 patients, accounting for 66.7%. In contrast, N. K. Gupta, Ritesh Kumar Agrawal, A. B. Srivastav, and M. L. Ved conducted a study that reported 42.5% of COPD patients with varying degrees of severity had pulmonary hypertension. Researchers Thabut, Dauriat, Stern, Logeart, Levy, and Marrash-Chahla Mal H. found PHT in a group of patients undergoing evaluation for lung volume reduction surgery or a lung transplant who had severe chronic obstructive pulmonary disease (COPD) and a high level of pulmonary hyperinflation. When people with severe chronic obstructive pulmonary disease (COPD) who needed a lung transplant were checked out by Andersen KH, Iversen M, Kjaergaard J, Mortensen J, Nielsen-Kudsk JE, Bendstrup E et al., 36% of them had PHT [161]. Researchers Oswald-Mammosser, Apppill, Bachez, Ehrhart, and Weitzenblum investigated this very topic. Prominent emphysema teratogenesis (PHT) was detected in 21% of chronic obstructive pulmonary disease (COPD) patients [50]. Participants in the "National Emphysema Treatment Trial" who suffered from severe emphysema had PHT found in 91% of their cases. In this study, 20 patients (16.7% of the total) were found to have mild PHT (ePPASP 36–45 mmHg). The other 21 patients (35.0%) were found to have moderate PHT, and 9 patients (15.0%) were found to have severe PHT (ePPASP 46–60 mmHg). ' Moderate COP D had a frequency of 57.1%, severe COP D had a frequency of 65%, and very severe COP D had a frequency of 91.7%; the p-value for this finding is 0.039*. This trend is in line with the findings of a study conducted by N. K. Gupta, Ritesh Kumar Agrawal, A. B. Srivastav, and M. L. Ved (491), which indicated that the prevalence of PH was 54.55% in mild COPD, 60.00% in moderate COPD, and 83.4% in very severe COPD. Higham MA, Dawson D, Joshi J, Nihoyannopoulos P, and Morrell NW [51] observed that 43% of patients with mild COPD, 68% of patients with moderate COPD, and 79% of patients with severe COPD experienced PH. Therefore, it is clear that the frequency of PH is positively correlated with the severity of chronic obstructive pulmonary disease (COPD). In contrast, a study conducted by Thbut et al. found that the severity was only weakly related to the underlying lung disease severity. Out of 11 patients with chronic obstructive pulmonary disease (COPD), 2 (18.1%) had severe COPD, and 9 (81.9%) had very severe COPD; this finding is in agreement with that of the study by N. K. Gupta, Ritesh Kumar Agrawal, A. B. Srivastav, and M. L. Ved, as there is a statistically significant correlation between the severity of COPD and RV size.

Conclusion

Hemodynamic alterations on the right side of the heart, such as RV enlargement and PI-IT, are strongly associated with the severity of chronic obstructive pulmonary disease (COPD) as classified by GOLD.It is worthwhile to search for PHT in this cohort because it is frequently found in COPD patients.

Recommendations

I. In COPD patients whose symptoms are severe and not proportional to their spirometric value or the degree of hypoxemia, we recommend maintaining a high index of suspicion of PHT.

2. We advise these patients to undergo an echo scan since echocardiography is still the gold standard for screening for PHT; it is easy, inexpensive, readily available, and dependable.

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3. Due to its predictive value, an expanded investigation should be conducted with a greater number of patients and a longer time frame to investigate the prevalence of PI-IT in COPI patients.

References

l. Stcvcnl). Sliapiro, MLD, Gordonl,,. Snider, M.D. Stephen l, Rcnnafd. M.D. .COPD in Murry J.P. and Nada' J.A., •rcxtbook of Respiratory Medicine, 5th edition 2005, W Tj Saunders, chap.39.pnge 947-983.

2.J.A. Innes P.m. Reid; Respiratory disease: (COPI)). Davidson's prtnciples and practice of tncdicinc 21 th Ed.

3.Dennis E, Niewochner ; Chronic obstructive pultnonary diseasc in Cecil Textbook of medicine 24'" Edition 2012 chapter 88 page 537-544.

4.Mun•ay CJ, I.opez AD. Evidence based health policy-lessons from the Global Burden of disease Study. Science 1996; 274:740e3

5.S. Scharf Sm, Iqbal M. Keller C. Criner G, Lee S, Fessler ilE. Hemodynmnic chatälcterization of patients with severe emphysema. Am J Respir Crit Care Mcd 2002;

6.Berj George DerniUiian, MD Fellow, Division of Pulmonary/Critical Care Medicine, Cedars-Sinai Medical Center is a member of the American College of Chest Physicians.

7.Thurlbcck WM. Pathophysiology of chronic obstructive pulmonary disease. Clin Chest Med. sep 1990;

8.Peinado Vi, Santos S, Ramirez J. Roca J, Rodriguez-Roisin R, Barbera JA. Response to hypoxia of pulmonary arteries in chronic obstructive pulmonary disease: an in vitro study. Eur Respir J 2002;

9.Barbera JA, Peinado VI, Santos S. 2003. Pulmonary hypertension in chronic obstructive pulmonary disease. Eur Respir J, 21:892—905.

10.Daniels LB, Krummen DE, Blanchard I)G. Echocardiography in pulmonary vascular disease. Cardiol Clin .

12.Sin DD, Anthonisen NR, Soriano 113, Agusti AG. Mortality in COPD: Role of comorbidities. Eur Respir J

13.Simonneau G, Gatzoulis MA, Adatia l, Celermajcr D, Denton C, Ghofrani A, Gomez Sanchez MA, Krishna Kumar R, Landzberg M, Machado RF, Olschewski VI, Robbins 1M, Souza R. Updated clinical classification of pulmonaty hypertension. .1 Am Coll Cardiol

14. Weitzenblum. Hirth , A.Mirhom R, Rasaholinjanahary J, Ehrhart M. Pmgnostic value of pulmonary artery pressure in chronic obstructive pulmonary disease. Thorax 1981;36: 752—758.

15.Stone AC, Machan JT, Mazer J, Casserly B, Klinger JR. Echocardiographic evidence of pulmonary hypertension is associated with increased I-year mortality in patients admitted with chronic obstructive pulmonaty disease. Lung. 201 1 Jun; 189(3):207—12.

16.Andersen KH, Iversen M, Kjaergaard J, Mortensen J, Nielsen-Kudsk JE, Bendstrup E et al. Prevalence, predictors and survival in pulmonary hypertension related to end-stage chronic obstructive pulmonary disease. J Heart Lung Transplant. 2012 Apr;31 (4):373—80.

17.Kessler R, Faller M, Fourgut G, Mennecier B, Weitzenblum E. Predictive factors of hospitalization for acute exacerbation in a series of 64 patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1999; 159:158-164.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

18. Chaouat A, Bugnet AS, Kadaout N, Schott R, Enache I, Ducolone A et al. Severe Pulmonary Hypertension and Chronic Obstructive Pulmonary Disease.Am J Respir Crit Care Med 2005; 172: 189—194. 10

19.Arcasoy SM, Christie JD, Ferrari VA, Sutton MS, Zisman DA, Blumenthal NP et al. Echocardiographic assessment of pulmonary hypertension in patients with advanced lung disease.Am J Respir Crit care Med. 2003 Mar l ; Epub 2002 Dec 12,A. Hales, site and mechanism of' oxygen sensing for the pulmonary vessels." Chest. vol. 88, no. 4, pp. 235S—240S. 1985.

21 .U. Von Euler and G. l.i!ielstrand. "0bServations on the pulmonary,arterial blood pressutv in the Physiologica Scandinavica, vol. 12, pp. 301-320, 1946.

22..K. R. Stenmatk, K. A. l;agati. and M. G. Frid. "Ivlypoxia-induced pultnonaty vascular temodeling: cel lular and molecular mecllanistns," Circulation Research, vol. 99, no. 7, pp. 675—691, 2006.

23.1M. Wilkinson, C. A. Langhorne, D. [Icath, G. R. Barer, and P. Howard, "A pathophysiological study of 10 cases of hypoxic cor pulmonale," Quarterly Journal of Medicine, vol. 66, no. 249, pp. 65—85, 1988.

24.1. L. Wright, T. Petty, and W. M. Thurlbcck, "Analysis ofthe structure of the muscular pulmonary atteries in patients with pulmonary hypertension and COPD: national Institutes of Health nocturnal oxygen therapy trial," Lung, vol. 170, no. 2, pp. 109—124, 1992.

25.S. Santos, V. I. Peinado, J. Ramirez et al., "Characterization of pulmonary vascular remodel ling in smokers and patients with mild CORD," European Respiratory Journal, vol. 19, no. 4, pp. 632—638, 2002.

26.E. M. Tschcrnko, E. M. Gruber, P. Jaksch et al., "Vcntilatory mechanics and gas exchange during exercise before and after lung volume reduction surgery," American Journal of Respiratory and Critical Care Medicine, vol. 158, no. 5, pp. 1424-1431, 1998.

27.J. L. Wright, L. Lawson, P. D. Pare, et al., "The structure and tunction Of the pulmonary vasculature in mild chronic obstructive pulmonary disease. The efféct of oxygen and exercise," The American Review of Respiratory Disease, vol. 128, no. 4, pp. 702-707, 1983.

28.J. Butler, F. Schr(jen, A. Henriquez, J, M. Polu, and R. K. Albert, "Cause of the raised wedge pressure on exercise in chronic obstructive pulmonary

29.Peinado VI, Pizarro S, Barbera JA. Pulntonaty Vascular Involvement in COPD. Chest 2008; 134:808-814.

30. E, Cretnona G, Campana M, Scotti C, Pagani M, Bianchi L, ct al. production of endogenous nitric oxide in chronic obstructive pulmonary disease and patients with cor pulmonale. Correlates with echo-Doppler assessment, Am J Respir Crit Carc Med. 2000 Aug;

31.Jyothula S, Safdar Z. Update on pulmonary hypertension cornplicating chronic obstructive pulmonary disease. International Journal of COPD

32.Joppa P, Petrasova D, Stancak B, Tkacova R. Systemic Inflammation in Patients With COPD and Pulmonary Hypertension. Chest 20()6;130:326— 333.

33.A. Nakamura, N. Kasamatsu, l. Hashizume et al., "Effects ofhcmoglobin on pulmonary arterial pressure and pulmonary vascular resistance in patients with chronic emphysema," Respiration, vol. 67, no. 5, pp. 502— 506, 2000.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

34.S. Deem, E. R. Swenson, M. K. Albeils, R. G. Hedges, and M. J. Bishop, "Red-blood-cell augmentation of hypoxic pulmonary vasoconstriction: hematocrit dependence and the importance of nitric,Journal of Respiratory and Critical Care Medicine, vol. 157, no. 4, pp. 1 181-1 186, 1998.
35.1. Azarov, K. T. Huang, S. Basu, M. T. Gladwin, N. Hogg, and D. B. Kim-Shapiro, "Nitric oxide scavenging by red blood cells as a function of hematocrit and oxygenation," Journal of Biological Chemistry, vol. 280, no. 47, pp. 39024-39032, 2005.
36.Eddahibi S, Chacuat A, Morrell N, Fadel E, Fuhrman C, Bugnet AS et al. Polymorphism of the serotonin transporter gene and pulmonary
37. Surinder Janda, Neal Shahidi, Kenneth Gin, John Swiston. Diagnostic accuracy of echocardiography for pulmonary hypertension: a systematic review and meta-analysis.
38.European Journal or Interilal Medicine. Echocardiographic estimation of puhnonaty alterial systolic pressure in acute heart failure 2013.
39. Roldan C. The ultinmte echo guide. Philadelphia, PA: Lippincott Williatns and Wilkins; 2005.
40.Groves BN'I, Badesch DB. Cardiac catheterization of patients with pulmonary hypeflension. In: Peacock AJ, Editor. Pulmonary circulation. London: Chapman & Hall, 1996: 51—67.
41. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. Circulation 1984;70:657-62.
42.Currie PJ, Seward JIB, Chan KL, Fyfe DA, Hagler DJ, Mair DD, et al. Continuous wave Doppler estimation of right ventricular pressure: A simultaneous Doppler-catheterization study in 127 patients. J Am Coll Cardiol
43.Chan KL, Currie PJ, Seward 1B, Hagler DJ, Mair IDD, Tajik AJ. Comparison of three Doppler ultrasound methods in the prediction of pulmonary artery pressure. J Am Coll Cardiol 1987;9:549-54.
44.Bredikis AJ, Liebson PR. The echocardiogram in COPD: Estimating right heart pressures. J Respir Dis
45.Braunwald's Heart Disease. 8th Edition. By Libby P, Bonow RO, Zipes DP, Mann DL. Philadelphia: Saunders 2008. p. 251.
46.Galie N, Hoeper MM, Humbert M, Torblcki A,Vachiery JL, Barbera JA. et al. Guidelines for the diagnosis and treatment of pulmonary hypertention ESC/ERS. Eur Respir J 2009; 34:1219-1263.,disease," The American Review Of Respiratory Disease, vol. 138, no. 2, pp. 350-354, 1988.
47.Dow L. asthma versus chronic obstructive pulmonary disease exploring why reversibility versus irreversibility is no longer appropriate approach. Clin. Exp. Allergy.
48.Calverley PMA, Burge PS, Spencer S, ct al. Bronchodilator reversibility testing in chronic obstructive pulmonary disease. "Ihorax. 2003;58:65964.
49.N. K. Gupta, Ritesh Krunar Agrawal, A. B. Srivastav, M. L.Ved. Echocardiographic evaluation of heart in chronic obstructive pulmonary disease patient and its co-relation with the severity of disease.
50.0swald-Mammosser M, Apppill M, Bachez P, Ehrhart M, Weitz.enblum E. Pulmonary hemodynamics in chronic obstructive pulmonary disease of the emphysematous type. Respiration. 1991;

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

51.Higham MA, Dawson D, Joshi J, Nihoyannopoulos P, Morrell NW. Utility of echocardiography in assessment of pulmonary hypertension secondary to COPD. Eur Respir J 2001; 17:350—355.40.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5