Interpretation of Clinical, Radiological, Electrocardiographic and Echocardiographic Findings in Pulmonary Hypertension

Basil N. Saeed

ABSTRACT:

OBJECTIVE:

To make correlation between clinical radiological electrocardiographic and echocardiographic findings in cases of Pulmonary Hypertension.

METHODS:

140 patients refered to the medical departments, Baghdad Teaching Hospital (90 of them admitted in this study were as follow);(52 (47%) cases having Ischemic heart disease,28(25%) cases having C.O.P.D., 10(9%) cases having connective tissue diseases, 9(8%) cases of Rehematoid Arthritis, 1 case S.L.E.). The remaining 50 cases were excluded from the study because they have another chronic disorder like Diabetus mellitus, Renal failure, Malignancy, Skeletal deformity.Fourty normal volunteers were admitted in this study both groups were assessed clinically, radiologically, electrocariograpically, echocardiographically plus the routine blood tests including F.B.S, B.urea,lipid profile.

RESULTS:

90 cases of patients admitted in this study showed variable findings the prominent finding was loud pulmonary sound "p2" in 100% of cases, 19.6% of cases showed cardiomegaly, 33% prominent pulmonary marking, 11.4% percardial effusion, 28% Right Ventricular Hypertrophy.

CONCLUSION:

It is not necessary to find all the clinical, radiological, electrocardiographic and the echocardiographic changes in any patient with pulmonary hypertention>30 mm Hg.

KEY WARDS: pulmonary hypertension medical disorder – Iraq.

INTRODUCTION:

Pulmonary hypertension refer to any elevation of the pulmonary arterial pressure above normal and this may reflect an underlying cardiac, respiratory, connective tissue disorder, familial, pulmonary thromboembolic disorder and parasitic infection.

The world health organization classify pulmonary hypertension from non symptomatic group to sever disability group (4 groups; group 1 non symptomatic to group 4 severe symptoms)[5].

Regarding the primary pulmonary hypertension the cause is unknown but many studies pointed out toward the pulmonary endothelial cells which have reduced no synthesis(essential for production of nitric oxide)[1]. The standard definition of pulmonary hypertension is via right heart catheterization. Normal mean pulmonary artery pressure is 19mm Hg at rest, right heart catheterization is an invasive procedure. So in this study the non ivasive procedure like echocardiogram was very helpful in measuring the pulmonary pressure. The coffecient correlation between catheterization and Doppler measurements is(0.87-0.97), the average standard error of systolic pulmonary artery pressure is 5-9 mm Hg and interoberver variability 3%[(9,10 ref)] pulmonary artery pressure may reach higher figure > 35_ 40 mm Hg as in connective tissue disorders[2].

AIM OF THE STUDY:

To study the association between the clinical,radiological, electerocardiographic and echocardiographic findings in any patient suspected of having pulmonary hypertension.

PATIENTS AND METHODS:

Ninety patients refered to the medical department at Baghdad Teaching Hospital for the period February 2005- March 2006.The questionnaire was including the age, gender, the complaint, the type of the disease and list of investigations which were including routine blood tests, F.B.S., B.urea, lipid profile, chest X ray,E.C.G., pulmonary function tests, and echocardiography. The pulmonary function tests done at the same day of doing the echocardiogram. The pulmonary function tests consisted of spiromety, static lung volume, gas transfer factor, flow loop, and fev1, FVC.The estimation of pulmonary artery systolic pressure by estimating the trans-tricuspid gradient and adding to it the juglar venous pressure[3], the trans tricuspid regurtation is depending on Bernoulli equation(delta p= 4v2) where delta p is the trans – tricuspid gradient and v is the peak velocity measured.Also during the echocardiographic examination the ejection fraction measured using the following equation[4].

Department of Medicine, Baghdad College of Medicine.

$$\frac{\text{Left ventricular diastolic volume – left ventricular systolic volume}}{\text{Left ventricular diastolic volume}} \times 100$$

$$\text{i.e. } \frac{\text{LVDV – LVSV}}{\text{LVDV}} \times 100$$

STASTICAL ANALYSIS:

Stastical package social science(S.P.S.S.) is used including all frequencies of age,Gender,Duration of disease.

RESULTS:

Ninety patients from the 140 patients referred to the medical department/ Baghdad Teaching Hospital went through clinical, Electrocardiographic and Echocardiographic examinations in which the pulmonary artery pressure > 30 mm Hg. All were distributed in the followings tables.

Table I: Distribution of 90 patients according to their primary diseases

Primary disease	NO.	%
Ischemic heart disease	52	47%
Chronic obstructive lung disease(C.O.P.D.)	28	25%
Collages diseases	10	9%
Total	90	

This table shows that Pulmonary Hypertension could be detected in the different forms of medical disorders .

Table II: The 90 cases distribution according to the sex

Patients	Male		Female	
	No..	%	No.	%
90	60	54%	30	27%

This table shows that no sex discrimination in the development of pulmonary hypertention.

Table III: Physical signs in % in those 90 cases in which pulmonary pressure was above 30mm H

Physical signs	No.	%
Pulse 80-100 minute	90	100%
P2 accentuated sound	90	100%
Ejection systolic murmur	77	86%
Ejection click	72	80%
Jugulor venous pressure	7	8%
Tricuspid parsystolic murmur	5	6%
Left parsternal heave	2	2%
Palpable thrill	2	2%
Pulsus parodoxicus	1	1%
Hepatomegaly	1	1%

The main physical findings were: the heart rate of 80-100 minute (100%), high pitched pulmonary sound100%, ejection systolic murmur 86%, and ejection click 80%.

Table IV: Distribution of patients according to CXR data

Xray findings	%
Prominent pulmonary marking	33 %
Cardiomegaly	19.6 %
Percardial effusion	11.4 %
Kerly B lines	2 %
Emphysematous lung	1.9 %
Pleural effusion	1.9 %

The most common radiological changes were : prominent pulmonary marking 33% , cardiomegaly 19% and pericardial effusion 11.4%.

Table V: Distribution of patients according to ECG data

Electrocardiographic changes	%	No.
Right ventricular hypertrophy	28%	31
Left bundle block	12%	13
Left ventricular hypertrophy	12%	13
P-mitrale	8%	9
Right bundle branch block	6%	7
P-pulmonale	4%	4
Left axis deviation	2%	2
Right axis deviation	2%	2
Total		81

The main electrocardiographic changes shown in Table V were; Right Ventricular Hypertrophy 28%, Left Bundle Block 12%, Left Ventricular Hypertrophy 12%, in 9 of the 90 cases the electrocardiographic findings were not conclusive.

Table VI: Distribution of pulmonary hypertension findings according to Echo data

Echocardiographic findings	%	No.
Tricuspid regurgitation	12.5 %	14
Mitral valve abnormalities	11.3 %	13
Septal hypertrophy	5.3 %	6
Left ventricular dysfunction	5 %	6
Left ventricular diltation	3.4 %	4
Right ventricular diltation	2.8 %	3
Thickened mitral valve	2.8 %	3
Mild pericardial effusion	1.4 %	2
Total		51

From the 90 cases, 51 cases Echo findings were positive and 39 cases Echo findings were not conclusive.

DISCUSSION:

These clinical, electrocardiographic,radiological, and echocardiographic changes will be more frequently seen in those patients if pulmonary artery pressure around 24 mm hg, in this study there was no association between the echocardiographic and radiological changes in the estimation of pulmonary hypertention in this study the level of pulmonary hypertention is 30 mm Hg because a study done on normal persons showed that pulmonary arterial pressure may reach the level of 24 mm Hg in normal persons with exercise, also the results in this study may differ from other studies because the selection of the patients is different in these studies[8], for example the range of mitral valve abnormalities is from(6%-30%) in this study it was 14.5%[(5)]. These variations could be seen with other changes like the pericardial effusion and tricuspid regurtation in many studies may range from(11.55-77%) in this study it was(11.4%), the pulmonary pressure hypertention reached >25mm Hg in 8 cases only, again this depend on the type of the cases(e.g. smokers or non smokers, racial, environmental, familiale,e.t.c) also the pulmonary artery pressure may reach higher level up to 35-40 mm hg as in emphysema[(6)].

CONCLUSION:

1-Types and severity of the disease may affect the level of pulmonary hypertension.

2-Pulmonary hypertension is not necessarily correlated with clinical electrocardiographic, radiological and echocardiographic findings.

3-The changes mentioned in(2) depend on the cause of pulmonary hypertention.

RECOMMENDATION:

Further work is possible in studying the pulmonary hypertension using the non invasive methods which can give avery helpful results.

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