Electrocardiographic changes in Chronic Obstructive Pulmonary Disease patients with elevated Pulmonary Artery Systolic Pressure

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Abstract

Background

Chronic Obstructive Pulmonary Disease (COPD) is a common global problem and most common medical problem in Nepal having significant morbidity and mortality. One of the pathogenesis of COPD in long run is the elevation of Pulmonary Artery Systolic Pressure (PASP) leading to right heart failure. A simple investigation - an Electrocardiograph (ECG) is assessed to co-relate with elevated PASP measured by Echocardiography in COPD patients of Dhulikhel Hospital.

Methods and Materials

A retrospective case control study of 342 COPD patients was done with assessment of ECG to co-relate with elevated PASP and with normal PASP. Data were analyzed using SPSS 17.

Result

There was significant difference in mean age, P amplitude in Lead II, III and aVF, QRS axis and R wave in V_1 and S in V_6 between two groups.

Conclusion

ECG changes are fairly sensitive and specific for elevation of PASP.

Key Words

COPD, PASP, Electrocardiograph, Echocardiography

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common global problem. It has been estimated that COPD will rise from sixth rank as the cause of death in 1990[1] to third most common cause of death world wide by 2020[1,2,3]. According to a hospital based study, COPD is the most common medical problem in Nepal and has significant morbidity and mortality[4]. One of the pathogenesis of COPD in long run is the elevation of Pulmonary Artery Systolic Pressure (PASP) leading to right heart failure, reason for high morbidity and mortality[5]. PASP can be estimated correctly using echocardiography but unfortunately this is available only in few of tertiary hospitals[6]. It is expensive and requires highly specialized manpower. So in this study we try to look at a very simple investigation an Electrocardiograph (ECG) and co-relate this with the elevated PASP. ECG is simple, cheap, and available in most of the places and can be read even by the general physician.

Methods and Materials

Patients who were admitted in medical ward of Dhulikhel hospital, Kathmandu university hospital with the diagnosis of COPD from January 1st 2010 till December 31st 2010 and who were indicated for echocardiographic study by the clinicians were selected retrospectively. Patients with PASP more than 30 mm Hg were taken as cases and those with less than 30 mm Hg were taken as control.

160 patients with PASP > 30 mmHg were identified and ECG was collected from each patient file. Similarly there were 182 patients with PASP < 30 mm Hg.

PASP was calculated according to TR Gradient[6]

$$4TR^2 + 10 \text{ mm Hg}$$

Echocardiogram is performed by either one of the two cardiologists present at Dhulikhel Hospital using TOSHIBA power vision 6000 echocardiography machine.

ECG was taken using the standard method and calibration of 25mm/s and 10mm/mv by the ECG technician. ECG in both the groups were analyzed by the cardiologist.

Statistical Analysis was done using SPSS 17.0. Student's t-test was done to compare between the two groups.

Results

In patients with PASP > 30 mmHg, mean age is 58.5 ± 6.2 and in patients with PASP< 30mmHg, mean age is 54.5 ± 5.5 (p=0.01). Number of female patients were more than male patients in both the group.

There was significant increase in P wave amplitude of Lead II, III and aVF in elevated PASP group. Mean QRS axis was also increased significantly in PASP > 30 mmHg group (P=0.001). Similarly R and S amplitude are also increased in elevated PASP group.

Table 1 Difference in characteristic feature

	PASP > 30 mmHg	PASP < 30 mmHg	P value
Age			
Mean (years)	58.5 ± 6.2	54.5 ± 5.5	0.01
Range (years)	42 - 86	40 - 76	0.03
Sex			
Male (%)	71 (44.4)	86 (47.3)	0.5
Female (%)	89 (55.6)	96 (52.7)	0.5
Mean P wave amplitude			
Lead II (mm)	3.1 ± 0.8	2.3 ± 0.6	0.01
Lead III (mm)	2.4 ± 0.7	1.9 ± 0.4	0.03
Lead aVF (mm)	2.6 ± 0.6	2.1 ± 0.5	0.03
Frontal plane QRS axis (degree)	129 ± 12.5	73 ± 8.5	0.001
R amplitude in V_1 (mm)	7.6 ± 1.3	1.2 ± 0.3	0.001
S amplitude in V_6 (mm)	18.0 ± 5.6	3.9 ± 0.9	0.02

Similarly P wave amplitude of more than 2.5 mm in lead II was 78.6% sensitive and 78% specific for elevated PASP. Complete RBBB was more specific (93.4%). Poor progression of R wave was neither sensitive (33.6%) nor specific (27.4%) for elevation of PASP group.

Table 2 Sensitivity and specificity for different ECG characteristics

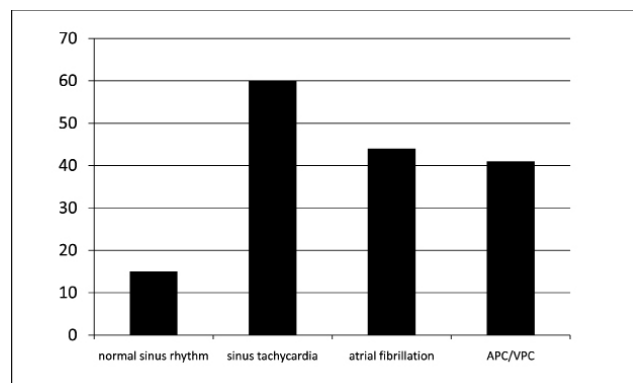| ECG characteristics	PASP >30 mm Hg	PASP <30 mm Hg	Sensitivity %	Specificity %
P in lead II >2.5 mm	126	42	78.6	78.0
R In V_1 > 7 mm	116	32	72.5	82.4
R/S ratio > 1 in V_6	112	35	70.0	80.8
Incomplete RBBB	106	82	66.3	54.9
Complete RBBB	52	12	67.5	93.4
Poor progression of R wave	54	132	33.6	27.4
T inversion in lead II,III, aVF	120	26	75.0	85.7
QRS axis >110 degree	127	38	79.3	78.0

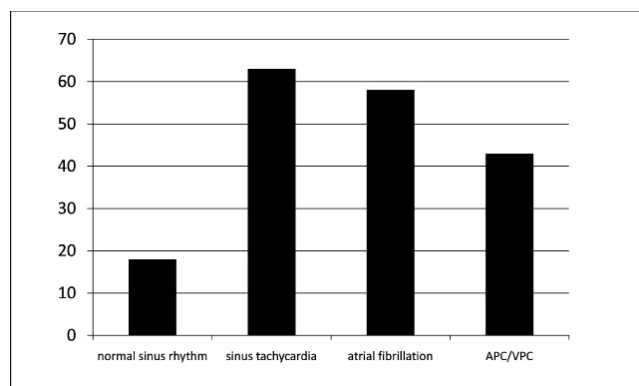*Fig 1 Rhythm in patients with PASP > 30 mm Hg*

Fig 2 Rhythm in patients with PASP< 30 mm Hg

Discussion

Pulmonary Artery Hypertension is a common complication of long standing COPD and is also a bad prognostic indicator[5]. ECG abnormalities corresponding with raised PASP is present in patients long before they have the symptoms of right heart failure[7,8]. Recent studies in rats and humans have illustrated that even a mildly increased right ventricular pressure load is associated with substantial changes in myocardial electrical properties, detectable in standard 12 lead ECG recording[9,10].

In our study, we found that there was significant difference in P wave amplitude in lead II, III and aVF between the two groups. P wave amplitude in lead II > 2.5 mm was 78.6% sensitive and 78% specific for elevated PASP. However Ivor R. Henkens et.al. found it to be 30% sensitive and 91% specific[10]. P wave amplitude in lead II increases as result of progressive hypertrophy – associated diastolic dysfunction and RV dilatation leading to associated tricuspid regurgitation[11]. Karliner et al. documented increase in P amplitude in lead II in healthy men who ascended from sea level to height of 6300 meters above sea level on Mount Everest and suffered from hypoxia induced PAH[11]. Our study being at hilly area is probably the reason for being more sensitive and less specific compared to that of Ivor R Henkens.

Height of R wave in V_1 was 72.5% sensitive and 82.4% specific where as Henkens et al. found 53% sensitive and 94% specific[12]. Similarly R/S ratio > 1 was 70% sensitive and 80.8% specific. In Henkens et al. it is 51% sensitive and 98% specific. We found complete RBBB to be 66.3% sensitive and 93.4 % specific whereas Henkens et al. found it to be 18 % sensitive and 96% specific. Increased height of R wave , R/S ratio >1 in lead V_1 and RBBB was found in yet another study by Henkens et al[12]. Poor progression of R wave was less sensitive and specific for elevated PASP, however T inversion in lead II, III and aVf was 75% sensitive and 85.7% specific.

QRS axis > 110 degree was 79.3 % sensitive and 78.0 % specific. Henkens et al found QRS axis >90 degree to be 84% sensitive and 96 % specific. Similar ECG changes are reported in other studies also[13,14,15].

When abnormalities are present in an ECG it is helpful to establish diagnosis but diagnosis cannot be ruled out in absence of ECG abnormalities. Numanik et al. also has the similar view[16].

Conclusion

From this study we can conclude that recording 12 lead ECG is fairly sensitive and specific way to recognize elevated PASP in COPD patients. It can be used routinely in COPD patients as a screening purpose in cost effective manner. However whenever clinically required and echocardiogram is available, echocardiographic measurement of PASP is advised.

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