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Correlation of ECG Changes with Pulse Oxymetry in Bronchial Asthma.

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ABSTRACT

Bronchial asthma is such a common disease that nearly 10-12 % of total population is suffering at some stage of life with this disease. The study attempts to assess the severity of asthma with help of electrocardiogram ranging from sinus tachycardia, P,ST and T wave changes etc with O₂ saturation showing hypoxia with the help of pulse oxymetry. The study conducted by us shows the female predominance with 36 (72%) patients without any age prevalence. In pulse oxymetric studies, 2 (44%) had Grade I oxygen saturation, while 7 (14%) with Grade III oxygen saturation. Out of all these patients, only 3 (6%) patients with Grade III oxygen saturation indicating severe hypoxia required ventilator support and had fatal outcome. the commonest arrhythmias found was sinus tachycardia in 42 (84%). other ECG findings were 'P' pulmonale in 11 (22%), ST-T abnormalities were seen in 5 (10%), Right bundle branch block which was seen in 8 (16%) patients.

Keywords: Bronchial asthma, ECG changes, pulse Pulse Oxymetry

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INTRODUCTION

Asthma is a heterogenous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness, and cough that vary over time and in intensity, together with variable expiratory airflow limitation (global initiative for asthma [GINA] 2014).[1] Asthma is currently one of the world's most common long-term noncommunicable disease; affecting about 300 million people worldwide[2,3] and number could increase further by another 100 million by year 2025.[1] Prevalence of asthma among developed countries is more (2.7–20%)[4-9] than reported from India.[10]

Definition: Bronchial Asthma is a disease characterized clinically by episodic / paroxysmal dyspnoea, cough and wheezing which is characterized by increased responsiveness of trachea and bronchi to a variety of stimuli and manifested by widespread narrowing of airways that changes in severity either spontaneously or a result of therapy. The mortality due to asthma results mainly from acute exacerbations. It varies considerably with age, race and geography. It has been showing an increasing trend in recent years. The most likely causes of this increasing trend in mortality are improper assessment of severity of episodes by the patient, physician or both, inadequate treatment in hospital and limited availability of health care because of socio-economic constraints. For efficient management and to prevent recurrent relapses, the severity of asthma has to be assessed.

This study attempts to assess the severity of asthma with the help of Electrocardiographic changes, including cardiac arrhythmias with oxygen concentrations showing hypoxia with the help of pulse oxymeter.

Aim & Objective:

To study the changes in ECGs due to acute bronchial asthma and correlate with oxygen concentration by pulse oxymetry.

MATERIALS AND METHODS

Fifty patients who presented with acute exacerbations of bronchial asthma and admitted in the Krishna Hospital and Medical Research Centre, Karad in Department of Medicine over a period of one year.

Inclusion Criteria :-

- Age above 14 years of either sex.
- Newly diagnosed asthmatic patients.
- Patients already suffering from asthma currently on treatment.

Exclusion Criteria :-

- Patients with history of smoking.
- Patients with ischemic heart disease.
- Patients with hypertensive heart disease.

Study Protocol :- Fifty patients who satisfied the inclusion criteria were included in the study. Detailed history regarding age of onset, duration of illness, frequency of attacks, variations with weather changes, history of allergy was asked. In all 50 patients, Electrocardiograms were done on admission, second day and third day. With all above we have assessed the oxygen concentrations to assess hypoxia with the help of pulse oxymeter on Day 1, Day 2 and Day 3. Before doing above procedures, patients were explained about all the procedures in detail. The other investigations like haemoglobin, urine, blood sugar, serum creatinine and chest X-ray were done using standard methods, as mentioned in the proforma.

RESULT

The study of 50 patients with acute exacerbation of Bronchial asthma was carried out in the Krishna Institute of Medical Sciences.

- The study conducted by us shows the female predominance with 36 (72%) patients without any age prevalence.
- The symptomatology, Dyspnoea in 47 (94%), wheezing in 40 (80%) and cough in 33 (66%) were most prevalent symptoms. Chest pain was in small number in our study.
- In our study, Tachycardia and Tachypnoea were present in 42 (84%) and 38 (76%) respectively which are invariably present in acute asthmatic exacerbation.
- In pulse oxymetric studies, 2 (44%) had Grade I oxygen saturation, while 7 (14%) with Grade III oxygen saturation. Out of all these patients, only 3 (6%) patients with Grade III oxygen saturation indicating severe hypoxia required ventilator support and had fatal outcome.
- In our study, the commonest arrhythmias found was sinus tachycardia in 42 (84%) patients and almost all patients reverted to normal rate with appropriate treatment. The supraventricular tachycardia and atrial flutter with fibrillation was found in 3 (6%) and 2 (4%) patients respectively. Out of these patients, three patients having supraventricular tachycardia had fatal outcome. All the above 5 patients with atrial tachyarrhythmias had > 25 years of total duration of illness.
- In our study, other ECG findings were 'P' pulmonale in 11 (22%) patients. Out of them, 9 (18%) reverted to normal while 2 (4%) had persistent 'P' pulmonale.
- While the ST-T abnormalities were seen in 5 (10%) patients and all of them reverted to normal.
- The other finding was Right bundle branch block which was seen in 8 (16%) patients. Out of them, 2 (4%) reverted to normal and 6 (12%) remained as RBB

DISCUSSION

Bronchial asthma is such a common disease that nearly 10-12% of total population is suffering at some stage of life with the disease. The disease is unpredictable in onset of attacks and prognosis. We can quantitate the severity of the airway obstruction by clinical methods, spirometry, peak expiratory flow meter reading, blood eosinophilia (To some extent), arterial blood gas analysis and also by electrocardiographic technique.

Here we assessed the severity of Asthma with electrographic tracing and correlation with clinical parameters of severity.

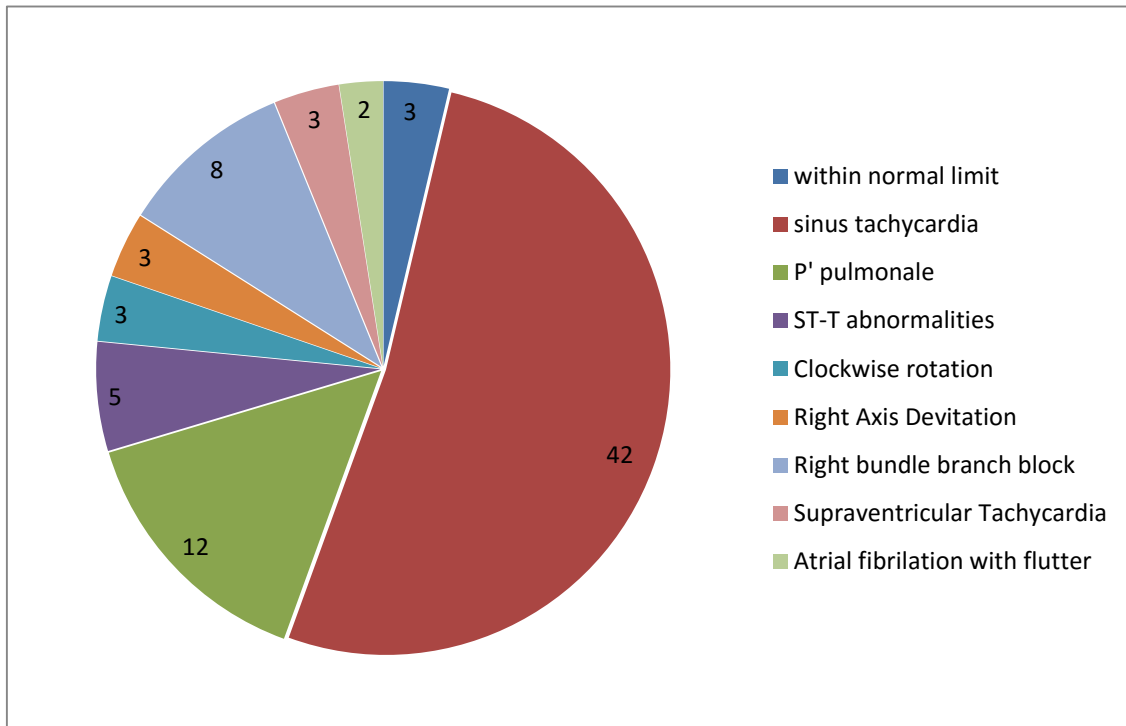
The following electrocardiographic changes were noted by Crofton and Douglas.[11] and Barry E Brenner.[12]

- Sinus tachycardia.
- P –Pulmonale.
- Right bundle branch block.
- Poor progression of R wave in precordial leads.
- T wave inversion in inferior and lateral leads.
- ST segment changes.
- Atrial and ventricular ectopics.

Soria R, Lobnosse J ET al.[13] studied 42 cases of status asthmatics patients with ECG during attacks. The following ECG changes were observed. The pulmonary p wave is common. Sometimes with exaggerated form in peripheral leads. Most cases have clockwise rotation of the heart and mild right axis deviation. S1 Q2 Q3 and transitional zone shifted to the left. Ten cases also had a S1 S2 S3 and three cases had Q1 Q2 Q3 simulating myocardial infarction. There is poor progression of R wave in precordial leads. In some cases a QS-complex dominated the right precordial leads. A variation in the amplitude of the QRS complex with respiratory rhythm is often seen in V1, and V2. Ventricular repolarisation shows a lowered J point with an upward oblique ST segment in the peripheral leads. However in the precordial leads, the repolarisation is normal except for the three cases who presented with frank hypokalemia. The mechanism of the ECG changes findings appear to depend on vertical position of heart caused by over expansion of lungs and pulmonary arterial hypertension.

In our study of 50 cases we noted the following ECG findings in acute severe asthma.

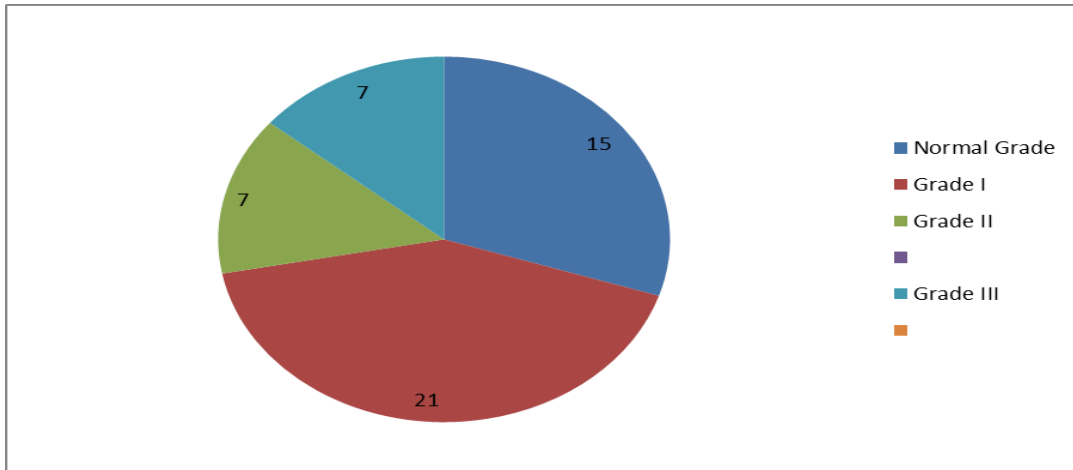
Group	ECG changes	No. of patients	Reversible (%)	Irreversible (%)
1	Within normal limits	3	-	-
2	Sinus tachycardia	42	42 (84%)	-
3	'P' pulmonale	12	10 (20%)	2 (4%)
4	ST – T abnormalities	5	5 (10%)	-
5	Clockwise Rotation	3	2 (4%)	1 (2%)
6	Right axis deviation	3	3 (6%)	-
7	Right Bundle branch block	8	2 (4%)	6 (12%)
8	Supraventricular Tachycardia	3	-	3 (6%)
9	Atrial Fibrillation with flutter	2	-	2 (4%)



Oxygen Saturation

Group	Oxygen Saturation	No. of patients	Reversible (%)	Irreversible (%)
1	Normal Grade	15	15 (30%)	-
2	Grade I	21	21 (42%)	-
3	Grade II	7	7	-
			6 – Normal 1 – Grade I	-
4	Grade III	7	7	3 (6%)
			2 – Normal 2 – Grade I	-

O₂ saturation : Normal - >95 %
 Grade I - 90 to 95 %
 Grade II - 85 to 89 %
 Grade III - <85 %



Correlation of ECG changes with Pulse Oxymetry

ECG	O ₂ Saturation			
	Normal (15)	Grade I (21)	Grade II (7)	Grade III (7)
Within normal limits	3	-	-	-
Sinus Tachycardia	13	20	7	2
'P' pulmonale	3	6	3	0
ST-T abnormalities	2	0	1	0
Clockwise rotation	0	2	1	0
Right axis deviation	1	4	0	0
Right Bundle Branch Block	1	3	1	3
Supraventricular Tachycardia	0	0	0	3
Atrial fibrillation with flutter	0	0	0	2

Sinus tachycardia: It is a common finding in patients with acute asthma. sinus tachycardia correlates with the severity of airflow obstruction and is a reliable indicator of severity of Asthma. Other mechanisms like drug induced β_2 agonist, adrenaline SC injection and theophylline. In our study we noted sinus tachycardia in 43 cases. Heart rate 101-110 in 9 cases, 111-120 in 15 cases, 121-130 in 12 cases, 131-140 2 cases, 141-150 in 3 cases and >150 in 2 case.

P-Wave changes: Normal P wave is best seen and studied in standard lead II because the frontal plane P wave axis is usually directed to the positive pole of this lead. The P wave in standard limb lead II is pyramidal in shape with somewhat rounded apex. Its limbs are smooth with no irregularities.

The duration of P wave is usually in the range of 0.08 sec to 0.1 sec. The maximum normal amplitude is 2.5mm, but the normal p wave is usually not greater than 2mm. The frontal plane P wave axis is directed to the region of +450 clockwise to +650. Most of the normal p wave axis are, however usually directed to the region of +450 to 550. P wave axis greater than +700 thus usually reflects right axis deviation of the p wave, P wave axis less than +450 usually reflects left axis deviation.

Even a P wave of relatively normal amplitude in the frontal plane leads, for example a P wave of 2 mm in amplitude in standard lead II, should arouse suspicion of right atrial enlargement if it is pointed. A tendency of a tall or relatively tall peak P wave in the frontal plane leads may occur in healthy individuals with sinus tachycardia or asthenic built. Right axis deviation (RAD) of the P wave correlates with lung function than does P wave amplitude, RAD is reversible after therapy. [12,13,14,15,16,17,18]

P Pulmonale: It is reflected by P wave which is tall and peaked in standard leads II and III and avF and in the expression of right atrial enlargement. This P wave form is best seen in these particular leads because the P wave axis is usually directed to +900 and is thus most aligned with these leads. The P wave will, as a result also is directed away from the positive pole of lead avL and thus be negative in these leads.

The manifestation of tall and peak waves in standard lead II, III and avF in association with right axis deviation of P wave, constitute a P-pulmonale.

Gelb A.F ET al⁷ studied 129 patients with acute severe asthma. P pulmonale in leads II, III and avF was found in 49% of patients with PaCO₂ of >45mmHg or more and arterial pH of 7.37 or less during a given asthma attack and in only 2.5% of asthmatic without hypercapnia and acidosis. P pulmonale persisted for 12-60 hours after correction of hypoxemia and is presumed to result from increased transmural right atrial pressure which in turn is a reflexion of severity of obstruction in asthmatics.

In our study we found P pulmonae , 11(22%) out of 50 patients in acute attack. These changes were reserved to normal 10(20%) after therapy and 2(4%) irreversible. **P wave axis:** In the present study, 37(74%) patients had p axis more than 90°. Normal p axis lies between 0° to 90°. Ambivagor M et al noticed it in 66% patients. Seigler D noticed it in 48(78%) and Gupta S.C et al in 17(68%).**Right bundle branch block(RBBB):** 8(16%) patients had right bundle branch block. Out of them 2(4%) were having incomplete right bundle branch block while 6(12%) were having complete right bundle branch block. Out of these patients, two incomplete right bundle branch block remained with complete right bundle branch block changes and one reverted to normal. **Clock wise rotation:**In the present study, clockwise rotation was present in 3(6%) patients.Clockwise rotation results from shift of cardiac position due to hyperinflation of lungs.**ST-T abnormalities:** ST-T changes in acute severe asthma are reversible after therapy,[13,19,20] these changes may be due to decreased oxygen supply to the heart because of hypoxia. ST-T abnormalities are found in 5(10%), patients in our study, which was reversible after therapy.**Sinus**

Tachycardia: Sinus tachycardia is the commonest in our study. The 42(84%) of patients in our study had sinus tachycardia. It correlate with the severity of airflow obstruction and is a reliable indicator of severe asthmatic attack. Almost all patients reverted to normal rate after treatment of acute attack.Tachycardia may be due to i)Increased plasma Norepinephrine levels found during acute severe asthma.ii)Other mechanisms like histamine induced bronchoconstriction.iii)Whenever there is a high PaCo₂ >6 KPa and severe hypoxemia PaO₂ < 8 KPa and low falling arterial pH, patients with severe asthmatic exacerbation and has heart rate ≥ 130beats/min. i.e sinus tachycardia.But it is observed that sinus tachycardia resolves with appropriate treatment of asthmatic attack.

In our study,special investigation done was pulse oxymetry showing correlation of the ECG changes with hypoxia.

- In this, there were 12(28%) patient with normal grade oxygen saturation,which remained normal till the third day.(95%)
- 22(44%) patient had Grade I (90-94%) oxygen saturation and all of them revereted to normal Grade.
- 7(14%) patients were in Grade II (85-89%). Out of these patients, 6(12%) reverted to normal while1(2%) reverted to Grade I.
- 7(14%) patients were found in Grade III(<85). Out of these patients, 2(4%) remained in Grade III and required ventilator support and expired.

Hypoxia was reported, as one of the commonest blood gas abnormalities in acute bronchial asthma by McFadden et al, Tai et al and Rees et al and the reason given was disturbance in ventilation perfusion ratio.[21,22,23]

Hypocarbica(PaCO₂<35 mm Hg) can be present in acute asthma. The mechanism could be due to increased ventilation drive resulting in decreased carbon dioxide tension and respiratory alkalosis.

McFadden et al, Rees et al and Rebeck et al have reported hypocarbica in their respective studies of bronchial asthma patients. Sometimes hypercarbica (PaCO₂ <45 mm Hg) has been reported in few studies. The incidence of hypercarbica deferred in the studies by McFadden et al, Tai et al and Rees et al.[2]

CONCLUSIONS

- According to pulse oxymetric, the sinus tachycardia and ST-T abnormalities were non-specific findings and did not have predictive value of hypoxia.
- The atrial tachyarrhythmias (SVT and Atrial Fibrillation) were indicators of greater severity of hypoxia in our study and were indicators of grave prognosis.
- 'P' pulmonale and Right bundle branch block are also the indicators of greater severity of hypoxia.
- Irreversible 'P' pulmonale, atrial tachyarrhythmias and irreversible Right bundle branch block were also correlated with the longer duration of illness and indicate the development of changes of chronic Cor pulmonale.

REFERENCES

- [1] GINA. Global Strategy for Asthma Management and Prevention. [Last revised on 2014 Aug 12] web link www.ginasthma.org.
- [2] Chapman KR. *Respir Med* 2005;99:1350-62.
- [3] Ehrs PO, Nokela M, Ställberg B, Hjemdahl P, Wikström Jonsson E. *Chest* 2006;129:925-32.
- [4] Burney P, Malmberg E, Chinn S, Jarvis D, Luczynska C, Lai E. *J Allergy Clin Immunol* 1997;99:314-22.
- [5] Chinn S, Burney P, Jarvis D, Luczynska C. *Eur Respir J* 1997;10:2495-501.
- [6] *Eur Respir J* 1996;9:687-95.
- [7] Devereux G, *et al.* *Thorax* 1996;51:169-74.
- [8] Peat JK, Haby M, Spijker J, Berry G, Woolcock AJ. *BMJ* 1992;305:1326-9.
- [9] Peat JK, Gray EJ, Mellis CM, Leeder SR, Woolcock AJ. *Eur Respir J* 1994;7:1805-13.
- [10] Aggarwal AN, Chaudhry K, Chhabra SK, D'Souza GA, Gupta D, Jindal SK, *et al.* *Indian J Chest Dis Allied Sci* 2006;48:13-22.
- [11] Anthony Seaton and Graham crompton, -Asthma: clinical features: Crofton and Douglas's Respiratory diseases, 34:957, 2004.
- [12] Barry E Brenner - *Emergency asthma*, 257:1999.
- [13] Sorial *et al.* *Ann cardiol Angeiol* 33(3) 153-8, Apr1984
- [14] Ahohen A. *Respiration* 37(2):85-90, 1979.
- [15] Batemen Jr *et al.* *Thorax* 35(5): 355-8 May 1980.
- [16] Gelb A.F., *et al.* *J Allergy Clin Immunol.* 64:18 July 1979.
- [17] Kelly HW, Menendez R and Voyles W. *Ann Allergy* 54:405, 1985.
- [18] Phipp P Gerrardc S. *Thorax*.12: Jan 2003.
- [19] Karwat K *et al.* *Wrad lek* 55 (9-10):525-34, 2002.
- [20] Ethimiouj Hasson A.B *et al.* *Respir. Med* May: 85 (3):195-202.
- [21] McFadden ER, Lyons Harold A. *The New England Journal of Medicine.* 278: 1027-1031, 1968
- [22] Rees HA, Miller JS, Donald KW. *Quarterly Journal of Medicine.* 37 : 541-561, 1968
- [23] Tai E, Read John. *Lancet* 1 : 644- 646, 1967.