DOI: 10.14260/jemds/2015/522

ORIGINAL ARTICLE

STUDY OF ECG CHANGES IN ACUTE SEVERE ASTHMA

D. Sudheer[1], P. Sasidhar[2], G. Prataprao[3], K. Ramya Priyadarsini[4]

HOW TO CITE THIS ARTICLE:
D. Sudheer, P. Sasidhar, G. Prataprao, K. Ramya Priyadarsini. "Study of ECG Changes in Acute Severe Asthma". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 21, March 12; Page: 3622-3632, DOI: 10.14260/jemds/2015/522

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. We can quantitate the severity of airway obstruction also by electrocardiographic techniques. Patients with acute severe asthma will manifest electrocardiographic changes ranging from sinus tachycardia, P and ST & T wave changes etc. These changes were reverted to normal after therapy.

KEYWORDS: Acute severe asthma, ECG, Peak flow meter, Salbutamol, Nebulization, Chest x ray, Sinus tachycardia, P wave changes.

INTRODUCTION: Definition of asthma**:** Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyper responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread, but variable airflow obstruction within lung that is often reversible either spontaneously or with treatment.[1]

PREVALENCE: Prevalence of asthma worldwide is around 300 million with a mortality of around 0.2; 10-12% of population at some stage during life suffers from asthma. The estimated burden of asthma in India is more than 15 million nearly, the disease can occur at any age, but in majority it can start before the age of 10 years. It is more common among boys up to the age of adolescence.

ACUTE SEVERE ASTHMA: DEFINITION: Originally, the term "Status asthmaticus" was used to describe a severe attack of asthma, which has continued for more than 24 hrs. Since many patients die within minutes/hours of onset of symptoms duration of attack is not relevant. The most important aspect is severity. Therefore status asthmaticus is not a useful description. Instead the term" acute severe asthma" is used to define severe air flow obstruction that has become unresponsive to therapy.

Criteria for assessment of acute severe Asthma: A 60L/min (Or 20% or more pre bronchodilator PEF) improvement after inhalation of bronchodilator or diurnal variation in PEF of more than 20% (With twice daily readings, more than 10%) suggests diagnosis of asthma.

Speech assessment: Too breathless to speak

Speech restricted to monosyllable or groups of words.

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Parameter	Acute Severe Asthma
Breathlessness	At rest
Talks	In words
Alertness	Usually agitated
Respiratory Rate	Often >30/min
Accessory muscles and suprasternal contractions	Usually
Wheeze	Usually loud/ silent chest
Pulse /min	>120
Pulses paradoxus	Often present >25mmHg
PEF after initial bronchodilator %predicted or % personal best	<60% predicted or personal best (<100L/min adults) or response lasts <2hrs
PaO_2 (on air) and or $PaCo_2$	<60mmHg, Possible cyanosis >45mmHg, Possible respiratory failure
SaO_2 % on (air)	<90%

Table 1: Criteria for assessment of Acute Severe Asthma.[1]

INVESTIGATIONS: PEFR <40% of predicted or best-known value of patient's absolute value <100 l/min. Arterial blood gas in an attack of asthma:- the PO_2 falls progressively but $PaCO_2$ falls initially due to hyperventilation and later rises due to fatigue.

AIMS & OBJECTIVES:

1. To study the various Electrocardiographic abnormalities in acute severe asthma.
2. To correlate the Electrocardiographic abnormalities with severity of airway obstruction.
3. Demonstration of reversibility of the Electrocardiographic abnormalities that are recorded during the acute attack of asthma, after therapy.

MATERIALS & METHODS: 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 disease is unpredictable in onset of attacks and progression. We can quantitate the severity of airway obstruction by clinical methods, Spirometry, peak expiratory flow meter readings, blood eosionophilia (To some extent) arterial blood gas analysis and also by electrocardiographic techniques.

In our study we have taken 50 cases of acute severe asthma based on clinical assessment like general examination, pulse rate and also examination of chest.

In our study we used peak expiratory flow meter (Vitalograph) and salbutomol inhalation with spacer and ECG machine:

I. Peak Flow Meter: A popular instrument for assessing airflow obstruction is the peak flow meter, which is suitable for use at home by individual patients or for keeping in the doctor's bag. These machines measure the maximal rate of flow, which is achieved during a forced

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expiration. Most healthy people will achieve values of greater than 400l/min. Patients with lung fibrosis and restrictive changes on the Spirogram may also have normal expiratory flow rates so that the meter is not suitable for assessment of their disability. Patients with airflow obstruction will have reduced flow rates with values <200l/min, being very significant and those below 100l/min, extremely severe. Peak expiratory flow meter reading will depend on the patient's height, age and sex of patient.

II. Inhaler: Each metered dose inhalation provides 100 μg of salbutamol IP. Suspended in inert aerosol propellant. Salbutomol is a β2 adrenergic stimulant which has selective action on β2 receptors in the bronchial muscles. Salbutomol binds to β2-adrenergic receptors in the cell membrane causing conversion of ATP to cAMP, which causes relaxation of smooth muscles.
III. Spacers: Provides smooth inhalation of salbutomol even if patients are unable to cooperate. It causes less wastage of the drug. It is useful even at low tidal volumes.
IV. Electrocardiogram: We have taken 12 leads electrocardiogram with three complexes in each lead.

First we measured the PEF meter reading, then we gave 2 puffs of Salbutomol inhalation i.e., 200μg through spacer and we waited for 15 minutes and again we took PEF reading and we selected patients only who showed airway reversibility of >20% based on the PEF reading.

We took ECG immediately after admission and again 24-48 hours after giving therapy for acute asthma, i.e., nebulization of β2agonists, parental hydrocortisone, oxygen inhalation and I.V. fluids.

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 and peak expiratory flow meter reading.

In our study, we have taken 50 cases of acute severe asthma, among them 29 cases are female and remaining 21 are male.

Total No. of Patients	Male	Female
50	21	29

Table 2: Sex Distribution

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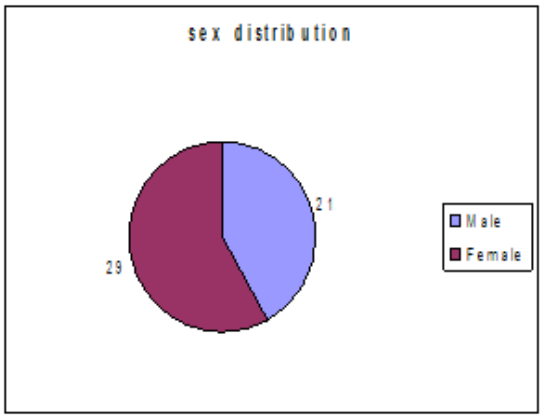

Fig. 1: Sex Distribution

Age Group	Male	Female
<20	0	1
21-30	0	6
31-40	6	8
41-50	11	9
>50	4	5

Table 3: Age Distribution

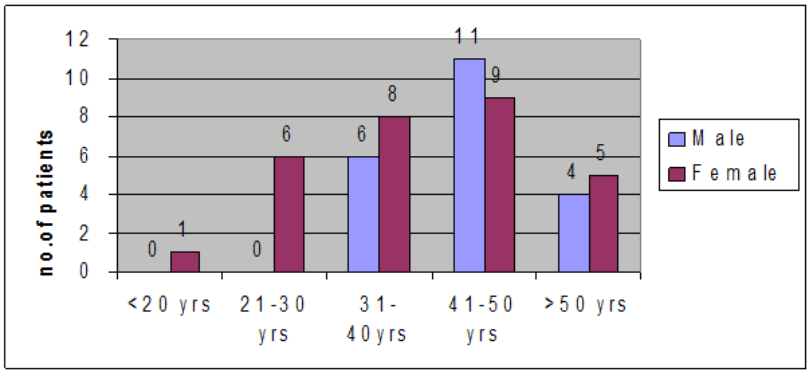

Fig. 2: Age Distribution

In our study of 50 cases of acute severe asthma, clinical assessment is made by:

1. Patient's Symptoms: Breathlessness at rest, usually agitated Speech restriction to monosyllables.
2. Physical signs like Pallor, Sweating, exhaustion, cyanosis and unconsciousness, tachycardia >120bpm and tachypnoea >30/min, pulsus paradoxus, silent chest.
3. We depended on peak expiratory flow meter reading for assessing severity by laboratory method (Bedside) in addition to clinical assessment, We have eliminated those patients who did not show reversibility of peak flow meter readings by more than 20% of the previous readings i.e., before bronchodilator inhalation like 400µg of salbutamol inhalation.

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If the PEFR <60% of the predicted or best known value of the patient or absolute value <100 l/min, we have taken it as severe asthma. We have taken the ECG immediately after admission into hospital and we have given nebulization with β2 agonists, parenteral hydrocortisone, oxygen inhalation and IV flui//ds. In 3 of our patients we have given aminophilline, IV drip because they did not respond to any other drugs. After 24-48 hours of treatment, we have taken the ECG again and compared with the first one. We did not estimate arterial blood gases because we donot have the facilities. In our study we got the PEFR value <200 l/min in all of the cases. In almost all the cases we got the value <60% of the PEFR for that age and height and sex.

We have taken a X-ray chest to rule out pneumonia and pneumothorax. The following changes were recorded.

Sl. No.	Chest X-ray abnormality	No. of cases	Percentage
1	No abnormality	25	50%
2	Hyper inflation	12	24%
3	↑Bronchovascular markings	9	18%
4	Consolidation	3	6%
5	ABPA	1	2%
6	Pneumothorax	1	2%

Table 4: Chest X-ray Abnormalities

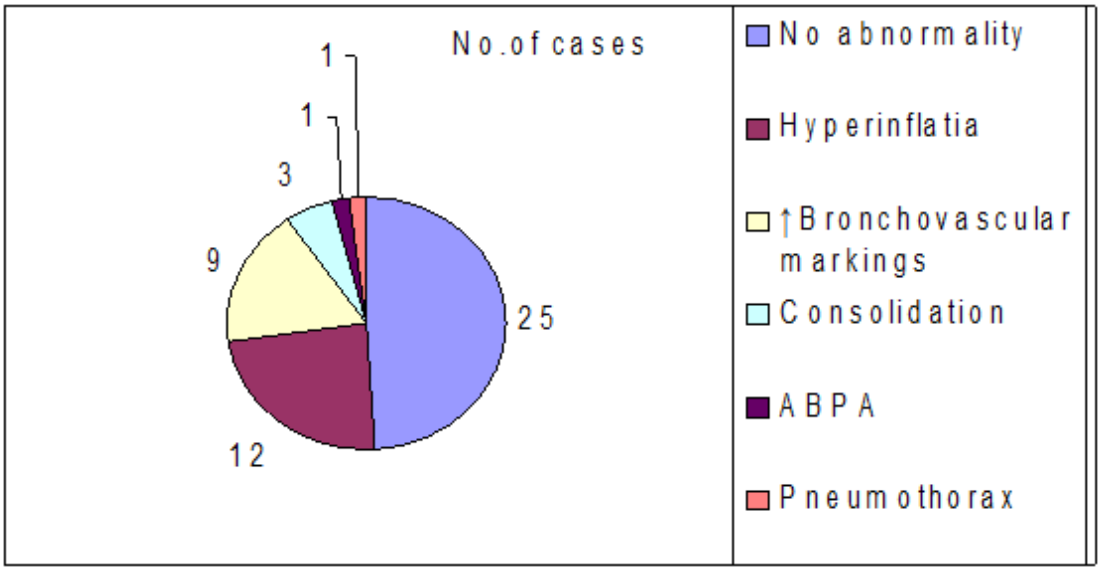

Fig. 3: Chest X-ray Abnormalities

The following electrocardiographic changes were noted by Crofton and Douglas.[2] and Barry E Brenner.[3]

1. Sinus tachycardia.
2. P –Pulmonale.
3. Right bundle branch block.
4. Poor progression of R wave in precordial leads.

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5. T wave inversion in inferior and lateral leads.
6. ST segment changes.
7. Atrial and ventricularr ectopics.

Soria R, Lobnosse J ET al.[4] 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. S_1 Q_2 Q_3 and transitional zone shifted to the left. Ten cases also had a S_1 S_2 S_3 and three cases had Q_1 Q_2 Q_3 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 V_1, and V_2. Ventricular re polarisation 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.

Sl. NO.	ECG Changes	No. of Cases	Percentage
1	Sinus tachycardia	45	90%
2	P Wave Abnormality- Tall & Peaked P waves- P wave axis RAD	24 16	48% 32%
3	Q waves in inferior leads	3	6%
4	ST depression in inferior and lateral leads	16	32%
5	T wave inversion in Inferior and Lateral leads	9	18%
6	Poor Progression of R waves	16	32%
7	U waves	4	8%
8	Right Bundle branch block	1	2%
9	Arrhythmias -atrial ectopics / ventricular ectopics	5	10%
10	No other changes except sinus tachycardia	5	10%

Table 5: ECG findings in Acute Severe Asthma

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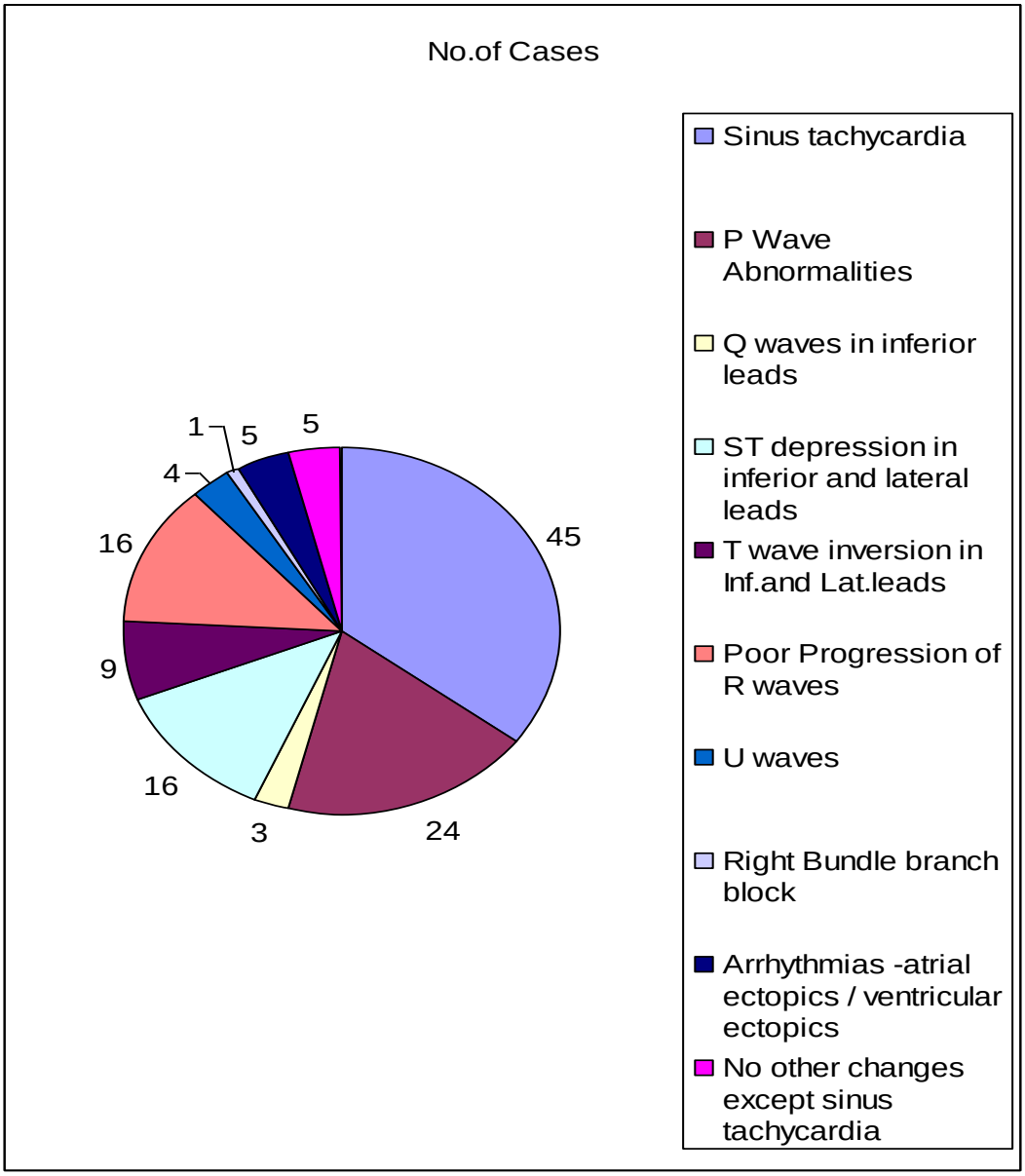

Fig. 4: ECG findings in Acute Severe Asthma

Sinus tachycardia: It is a common finding in patients with acute asthma. Pulse rate typically is 120 beats /min or more in 42 patients (84%) and 6 patients (12%) have rates in between 100 and 120. Some but not all investigators find that sinus tachycardia correlates with the severity of airflow obstruction and is a reliable indicator of severity of Asthma. Other mechanisms like drug induced β2 agonist, adrenaline SC injection and theophylline. In our study we noted sinus tachycardia in 45 cases. Heart rate more than >130 bpm in 23 cases and more than 100 bpm in 48 cases. In two patients we found heart rate less than 100 bpm 48 cases. In two patients we found heart rate less than 100 bpm, in spite of severe airway obstruction.

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Gordon D et al studied heart rate variability in bronchial asthma. They found that heart rates are higher in acute asthmatics when compared to healthy normal subjects and also in asymptomatic, untreated patient. Sympathetically mediated heart rate variability was significantly lower in asymptomatic and acute asthma subjects when compared to controls.

SL. No.	Pulse Rate(/min)	No. of patients	Percentage
1	≥ 100	48	96%
2	≥110	45	90%
3	≥120	42	84%
4	≥130	23	46%

Table 6: Pulse Rate Distribution

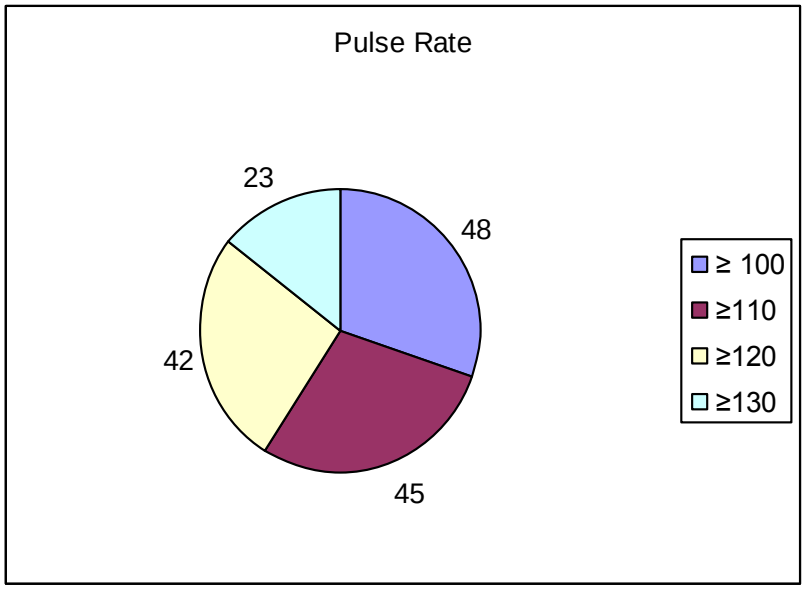

Fig. 5: Pulse Rate Distribution

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 $+45^0$ clockwise to $+65^0$. Most of the normal p wave axis are, however usually directed to the region of $+45^0$ to 55^0.P wave axis greater than $+70^0$ thus usually reflects right axis deviation of the p wave, P wave axis less than $+45^0$ usually reflects left axis deviation.

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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. [3,4,5,6,7,8,9]

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 $+90^0$ 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 al[7] studied 129 patients with acute severe asthma. P pulmonale in leads II, III and avF was found in 49% of patients with $PaCO_2$ 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 pulmonlae in 48% of cases, these changes were reversed to normal after therapy.

T wave abnormalities:

In a previous study, reversible T wave abnormalities studied in 70 consecutives patients with severe acute asthma on admission to Hospital and during recovery. Twenty two (22) i.e., 34% had inferior lead T wave inversion on ECG, when performed with in an hour of admission, whereas the rest did not.[(20)]

Reversible T wave abnormalities in inferior leads correlates with severity of asthma.[5, 9,10,11,12,13,14] In our study T wave abnormalities in inferior and lateral leads were recorded in 9 cases i.e., 18% and inversion in V_1-V_3 in one case.

ST Changes: ST segment depression in inferior and lateral leads in acute severe asthma are reversible after therapy.[4,10,11] These changes may be due to decreased oxygen supply to the heart because of hypoxia.

In our study, we have noted ST depression in inferior and lateral leads in 32% of cases which were reversed to normal after therapy.

U-Wave abnormalities: U-wave changes observed in 4(8%) cases may be due to β-agonist administration and intracellular shift of potassium.

Arrhythmias: Hypoxia, dehydration, acidosis and hypokalaemia render the severe acute asthma patient vulnerable to cardiac dysrrhythmias and cardio respiratory arrest.[9] Premature ventricular contractions and ectopics are also noted in small no. of patients.[3,15,16]

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In our study, we have noted atrial ectopics in 5 cases (10%).

SL. No.	Electrocar diogram	% of cases	Comparison with previous studies
1	Sinus Tachycardia	84%	90%
2	P wave changes	48%	49% (Gelb A.F et al) 50% (Barry E Brenner)
3	S-T segment changes	32%	
4	T wave abnormalities	18%	18.9% (Karwat K et al) 34% (Effthmiouz et al)
5	U waves	8%	
6	Atrial ectopics	10%	20%(Josephson G.W et al)
7	RBBB	2%	

Table 7: ECG findings, Comparison with Previous Studies

CONCLUSION: From this study it is concluded that, patients with acute severe asthma will manifest electrocardiographic changes ranging from sinus tachycardia, P and ST & T wave changes etc. These changes were reverted to normal after therapy.

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AUTHORS:

1. D. Sudheer
2. P. Sasidhar
3. G. Prataprao
4. K. Ramya Priyadarsini

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Pulmonary Medicine, Government Medical College, Jagdalpur.
2. Pulmonologist, Chetana Chest Clinic, Nellore.
3. Associate Professor, Department of Anaesthesia, Viswabharati Medical College, Kurnool.
4. Senior Resident, Department of Anaesthesia, Government Medical College, Jagdalpur.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. D. Sudheer,
Associate Professor,
Department of Pulmonary Medicine,
Government Medical College &
Maharani Hospital, Jagdalpur,
Chattisgarh State.
E-mail: dsudheer.dr@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None

Date of Submission: 18/02/2015.
Date of Peer Review: 19/02/2015.
Date of Acceptance: 28/02/2015.
Date of Publishing: 10/03/2015.