

# A Study of Electrocardiographic Changes in Asthmatic Patients

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### **Abstract:**

**Background:** Asthma is a common chronic respiratory disease affecting (7-10%) of the population in different countries. It is characterized by variable symptoms of wheeze, shortness of breath, chest tightness and cough. Asthma is usually associated with airway hyper-responsiveness to direct or indirect stimuli. Patient with acute asthma will manifest electrocardiographic changes ranging from sinus tachycardia, P, ST and T wave changes etc, these abnormalities persisted for up to 10 days, may reflect positional changes of the heart, were reverted to normal after therapy.

**Aims:** To study the various ECG abnormalities in acute asthma and to demonstrate the reversibility of these abnormalities after treatment.

**Material and Methods:** This study was conducted on (110) patients all were adults, their age ranged from 20 years to more than 50 years who attended to the emergency department in Kirkuk General Hospital over a period of one year from April 2017 to April 2018 with a confirmed diagnosis of acute severe asthma were studied. Ten patients were excluded from study because their ECG changes were related to cardiac and thyroid disorder. Patients were included in the study only if an ECG had been performed on admission and within 24 hour of their stay in hospital. A standard 12 Lead ECG was performed on admission for each patient and it repeated until it had returned to normal. In each patient, on admission, plasma urea, full blood count, thyroid functions test (in selected cases) cardiac enzymes (in those with chest pain) were performed. The diagnosis of acute asthma is made by taking a thorough history and physical exam, and patients without a previous diagnosis of asthma, a spirometry performed to confirm airway obstruction and reversibility.

**Result:** The commonest ECG changes were sinus tachycardia which was found in (50%) of patients with a heart rate ranging between 120-130 beats/ min, while the lowest rate of ECG changes was right bundle branch block were observed in (2%) of patients. The male patients were affected more than female and the highest incidence observed was among age group 41-50 years. The ST segment depression, T wave inversion & sinus tachycardia are the main ECG changes in (30%) of life threatening type of acute asthma. While P pulmonale & ST segment depression were observed in only (4%) of patients with near fatal asthma.

**Conclusion:** Patients with acute asthma can manifest various ECG changes and these changes returned to normal after medical therapy, usually within 10 days.

**Key words:** Acute severe asthma, Peak expiratory flow meter, Electrocardiography, Sinus tachycardia.

### **Introduction:**

Asthma is one of the most common diseases in developed countries and has a worldwide prevalence of (7-10%) <sup>(1)</sup>. It is also a common reason for urgent care

and emergency department visits <sup>(2)</sup>. In the United States, asthma accounted for an average annual of approximately 504,000 hospitalizations and 1.8 million

emergency department visits, women made twice the number of emergency department visits as men<sup>(3)</sup>. Asthma is heterogeneous disease, with varied triggers, manifestations, and responsiveness to treatment<sup>(4)</sup>. Some patients with acute a severe asthma presenting to the emergency department have a disease that responds rapidly to aggressive therapy, and they can be discharged quickly; others require admission to the hospital for more prolonged treatment<sup>(5)</sup>. Although the development, course of the disease and response to treatment, are influenced by genetic determinants, the rapid rise in prevalence implies that environmental factors are critically important in the development and expression of the disease<sup>(6)</sup>.

Asthma is a chronic inflammatory disorder of air ways 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<sup>(7)</sup>. The term status asthmaticus was used to describe a severe attack of asthma, which has continued for more than 24 hrs, instead the term "Acute Severe Asthma" is used to define severe airflow obstruction that becomes unresponsive to therapy<sup>(8)</sup>. Several "ECG" abnormalities have been reported in patients with an acute severe asthma, these include Tachycardia, P pulmonale, Right bundle branch block, Right axis deviation, ST segment and Twave abnormalities<sup>(9)</sup>. The severity of ECG abnormalities may correlate with degree of air way obstruction, and each type of acute severe asthma may have its own specific ECG signs<sup>(10)</sup>. The mechanism of these 'ECG' changes

appears to depend on effect of acute asthma on the heart by, **first**; lung hyper-expansion causes external compression of the heart and lowering of the diaphragms, with consequent elongation and vertical orientation of the heart. **Second**, due to its fixed ventricle anteriorly attachments to the great vessels, the heart undergoes clockwise rotation in the transverse plane, with movement of the right and displacement of the left ventricle posteriorly. **Third**, hypoxic pulmonary vasoconstriction and destruction of lung tissue with loss of pulmonary capillaries results in chronic elevation of pulmonary arterial pressures with consequent compensatory right atrial and right ventricular hypertrophy<sup>(11)</sup>. So various 'ECG' changes can occur in acute severe asthma which is nonspecific and these may mimic an acute cardiac event and can cause diagnostic dilemma, most of these changes are reversible usually within 10 days of treatment<sup>(12)</sup>.

The diagnosis of acute asthma in the emergency department is made by taking a thorough history and physical exam, the clinical presentation of asthma in a patient with a history of asthma is typically enough to diagnose and determine further management<sup>(13)</sup>. For patient without a previous diagnosis of asthma and atypical symptoms, a more thorough evaluation may be needed to exclude other etiologies<sup>(14)</sup>. Spirometry should be performed in patients with suspected asthma to confirm airway obstruction and reversibility, and patients with suspected asthma who have normal spirometry should undergo a bronchial challenge test to assess for airway hyper-responsiveness; a negative test generally excludes asthma<sup>(15)</sup>.

## **Materials and Methods:**

This study was conducted in the **KGH** at Emergency Unite and general medical word, over a period of one year starting from April 2017- to April 2018. The study involved " 110 " patients attending "**K G H**" emergency unite with sever acute asthma within few hours of onset of attack, 10 patients were excluded from the study because their " ECG " changes were cardiac (in 7 patients) and hypothyroid (in 3patients), and one hundred patients included in the study, (90%) of them known to be asthmatic, the remaining (10%) are new cases presented for the first time <sup>(16, 17)</sup>. In these new cases we measured the " PEF " meter reading, then we gave nebulization of salbutamol and we waited for15 minutes and again we took " PEF" reading and we selected patients only who showed airway reversibility of more than (20%) based on the 'PEF' reading <sup>(18)</sup>. Patients known to have coronary artery disease, valvular heart disease, systemic hypertension, thyroid disorder were excluded from the study <sup>(19)</sup>. A standard 12 lead ' ECG' was performed on admission and repeated 24 hours after giving treatment for acute asthma attack; the 'ECG' in most cases was repeated for 7 to 10 days until it had returned to normal. Sinus tachycardia is considered present when heart rates exceeding 100 b/min, with acute asthma pulse rate typically is 120b/min or more <sup>(20)</sup>. P wave changes is best seen in standard lead ll, it is duration is usually in the range of 0.08 sec to 0.1 sec, P wave axis is directed to the region of  $\pm 45^\circ$  clockwise to  $\pm 65^\circ$  and axis greater than  $\pm 70^\circ$  usually reflects right axis deviation of the p wave, p wave axis less than  $\pm 45^\circ$  usually reflects left axis deviation, right axis deviation of the p wave correlates with lung function than

does p wave amplitude <sup>(21)</sup>. P pulmonale is reflected by tall and peak waves in standard lead ll, lll and AVF in association with right axis deviation of P wave <sup>(22)</sup>. T wave is considered inverted when it is deeper than 1 mm; its considered flat when it is peak amplitude is between 1.0mm and -1.0mm <sup>(23)</sup>. ST segment depression was defined at least 0.1mv horizontal or down slopping ST segment depression compared with baseline ST level <sup>(24)</sup>. Poor R wave progression refers to the absence of normal increase in size of the R wave in the precordial leads when advancing from lead V1 to V6 <sup>(25)</sup>.

We quantitate the severity of airway obstruction into three categories depending to clinical finding, PEF meter reading and arterial blood gas analysis ,each category has it is own criteria these includes;

■ Acute severe asthma: \*PEF (33-50%) predicted (< 200 L/ min).

\* Respiratory rate  $\geq$  25 beats /min.

\*Heart rate  $\geq$  100 beats / min.

\*Inability to complete sentences in 1 breath.

■Life threatening asthma:

\*BEF < (33%) predicted \*SPO2 < (92%).

\*Normal or raised Paco2 \*Silent chest

\*Cyanosis \*Bradycardia

\*Confusion or hypotension \*Coma or exhaustion

■ Near - fatal asthma:

\* Raised Paco2 and /or requiring mechanical ventilation <sup>(26)</sup>.

In our study we used 'PEF', salbutamol nebulization, 'ECG ' machine and blood tests like cardiac enzymes, thyroid functions, renal functions and electrolytes to exclude other causes for ECG changes <sup>(16, 17)</sup>. We have taken a chest X-Ray to rule out pneumonia and

pneumothorax <sup>(27)</sup>. 'PEF' meter is a popular instrument for assessing air flow obstruction , most healthy people will achieve values of greater than 400L/min. Patients with airflow obstruction will have reduced flow rates with values < 200L/min, being very significant and those below 100L/ min extremely sever <sup>(28)</sup>. The data collected from questionnaire form were represented as tables, data were analyzed statistically using percentage, and statistical significance was performed by one-way ANOVA analysis of variance. P value less than 0.05 were considered to be statistically significant <sup>(29)</sup>.

## **Results:**

We found that nearly (50%) of patients have got Sinus tachycardia, with average heart rate ranging between 120-130 beat/ min, while the lowest rate of 'ECG' changes was right bundle branch block were observed only in (2%) of 100 patients. The ST segment depression and T wave inversion in inferior and lateral leads were near to each other (10%) and (12%) respectively, while the poor progression of R wave was observed in only (4%), table (1).

Age categorization done for patients presenting with acute severe asthma, were divided into five groups including age less than < 20 years to more than 50 years. It is clear from this table that male patients were affected more than female patients (63%), (37%)

respectively. The acute exacerbation was least among age group < 20 years and attacks escalating with increasing age and the highest incidence observed among age group 41-50 years. The high incidence in male patients was among age group 41-50 years at rate (22%), while in female patients was among age group 31-40 years at rate (12%), table (2).

This table shows 'ECG' changes among three types of acute asthma, (66%) of patients presenting with acute severe asthma have 'ECG' changes and common 'ECG' finding was Sinus tachycardia, peaked p wave. While (30%) of life threatening form of acute asthma has 'ECG' changes and ST segment depression, T -wave inversion and sinus tachycardia are the main 'ECG' changes, the remaining (4%) of acute asthma was Near fatal asthma in whom the P - pulmonale and ST-segment depression was a common encountered ECG changes, table (3).

This table shows chest X -Ray abnormalities among one hundred patients presenting with acute severe asthma. A normal CXR was a common finding were encountered in (70%) of patients, only (2%) of patients have consolidation and no one has pneumothorax. Hyperinflation of lungs from air trapping in sever airway obstruction was (15%) of the patients, table (4).

**Table (1):** ECG changes among 100 asthmatic patients during acute exacerbation

NO	ECG changes in acute asthma	No of cases	Percent %
1	Sinus tachycardia	50	0.5
2	P wave Tall and peaked P wave	16	0.16
3	ST segment depression in inf and lat leads	10	0.1
4	T wave inversion in inf and lat leads	12	0.12
5	Poor progression of R waves	4	0.04
6	RBBB	2	0.02
7	Arrhythmias atrial , ventricular ectopics	6	0.06
Total		100	1
P-Value = 0.0311 < 0.05 :- Significant			

**Table (2):** Age distribution in 100 patients with acute exacerbation of asthma.

Age group	Male	Female
< 20	0	3
21 - 30	15	8
31 - 40	14	12
41 - 50	22	10
> 50	12	4
Total	63	37

**Table (3):** Common ECG changes among three types of acute asthma .

No	Type of acute asthma	No of cases	Common associated ECG changes	Percent %
1	Acute severe asthma	66	Sinus tachycardia Peaked P wave	0.66
2	Life threatening asthma	30	ST depression,T inversion & sinus tachycardia	0.3
3	Near fatal asthma	4	P pulmonale, ST segment depression	0.04
	Total	100		1
P-Value = 0.042 < 0.05 :- Significant				

**Table (4):** Chest X-Ray abnormality in 100 patients of acute asthma.

No	Chest X-Ray abnormality	No.	Percent
1	No abnormality	70	0.70
2	Hyperinflation	15	0.15
3	Increased Broncho - vascular marking	13	0.13
4	Consolidation	2	0.02
5	Pneumothorax	0	0
	Total	100	1
P-Value = 0.046 < 0.05 :- Significant			

## **Discussion:**

We found that the sinus tachycardia is a common 'ECG' finding and encountered in (50%) of patients, with heart rate ranging between 120-130 b/m<sup>(30)</sup>. Some investigators found that sinus tachycardia correlates with the severity of airflow obstruction and is a reliable

indicator of severity of asthma<sup>(31)</sup>. But other investigators stated that sinus tachycardia is not a reliable indicator and they explained that sinus tachycardia could be a reflection of anxiety, stress, sympathetic stimulation, or could be from other mechanisms like

drug induced B2 agonist and theophylline drugs<sup>(32)</sup>. We found that (96%) of patients with acute asthma presenting to the emergency unite have sinus tachycardia especially acute severe asthma and life threatening type of acute asthma. The western figure widely differ from our result which counted sinus tachycardia in (84%) of patients by Gordan D et al<sup>(33)</sup>, while in our study sinus tachycardia alone counted in (50%), this may be explained by the fact that European people may react more with stressful condition than Iraqi people who exposed to great stressful events on his life.

S T segment depression and T wave inversion were noted in (22%) of patients which is near to the result obtained by Barry E Brenner<sup>(34)</sup>. These changes may be due to decreased oxygen supply to the heart because of hypoxia, which were reversed to normal after therapy<sup>(35)</sup>. P pulmonale in leads ll, lll, AVF was found in (26%) of patients and persisted for few days after correction of hypoxia and is presumed to result from increased transmural right atrial pressure which in turn is a reflection of severity of obstruction in asthmatics<sup>(36)</sup>.

In conformity with result obtained by Sorial and Ethimiouj, RBBB was found in only (2%), it correlates with lung function more than does P wave amplitude and it is reversible after therapy<sup>(37, 38)</sup>. Premature ventricular and atrial ectopics are also noted in small number of patients (6%), arrhythmias may be a result of hypoxia, dehydration, acidosis and hypokalemia which render the patients with acute asthma vulnerable to cardiac dysrrhythmia<sup>(39)</sup>.

The arrhythmias are mostly supraventricular in origin and include atrial fibrillation or flutter, and multifocal

atrial tachycardia. Ventricular extra systoles and ventricular tachycardia may also occur<sup>(40)</sup>. In three categories of acute asthma, ST segment changes and T wave inversion found in (30%) of patients presented to Emergency unite with life threatening asthma, it may correlates with severity of airway obstruction in this type of acute asthma, this result may open the door to a new study in the future to explain this correlation. This result is near to that obtained by ZabMohsenifar et al, ST segment in (16%) and T wave inversion (9%)<sup>(41)</sup>. In our study 'ECG' changes was more among male patients (age group" 41 - 50 " 22%) than female patients (age group " 31 - 40 " 12%), this figure is differ from the that obtained by Stfano R et al...<sup>(42)</sup>, were female patients shows 'ECG' changes more than male, this may be explained by nature of job and exposure to the environmental risk factors, nearly which is same between male and female in European people.

We found normal CXR is a common finding in (70%) of the patients during acute attack, however, other finding can include hyperinflation of the lungs with flattened diaphragm if there is significant air trapping. A CXR may be obtained if the cause of the patient's wheezing or difficulty breathing is uncertain, this is particularly helpful in patients with other co-morbid conditions, so there is no urgency for CXR during acute asthma, unless when you have clinical evidence of complication (ie-pneumothorax) or associated co - morbid condition<sup>(43)</sup>.

### **Conclusion:**

We concluded from this study that, patients with acute severe asthma manifest several electrocardiographic

changes like sinus tachycardia, P and ST wave changes etc.. These changes were reverted to normal after therapy, usually within 10 days.

## **Reference:**

- [1]. Stephen C, Lazarus, M .D. Emergency treatment of asthma N Engl J Med 2010; 363: 755 - 64.
- [2]. Rowe BH, Voaklabder DC, wang D, et al .asthma presentations by adults to emergency departments in alberta .canadian Respiratory Journal. 2009 Aug; 135: 57 - 65.
- [3]. Moorman JE, Rudd RA, Johanson CA, etal. National surveillance for asthma-United States. MMWR SurveillSumm 2007 Jan; 56: 1- 54.
- [4]. ClementinBostantzoglou, Vicky Delimpoura, KanstantinosSamitas. Clinical asthma phenotypes in the real world opportunities and challenges. Breathe 2015 December; 11: 186 - 193 .
- [5]. Kelly AM, Kerr D, Powell C. severity assessment after one hour of treatment better for predicting the need for admission in acute asthma; Respir Med 2004 Nov; 98: 777 - 781.
- [6]. Paul Cullinan, Anthony Newman Taylor. Asthma: environmental and occupational factors. British Medical Bulletin. 2016 December; 68: page 227 - 242.
- [7]. Anthony Seaton and Graham Eropont Global Strategy for asthma management and prevention 2014 (up dated GINA report), 2 - 7, 16 -19.
- 8- Kelly HW, Menendez, R and Voyle W. Asthma: clinical features: Respiratory diseases 2014 Mar; 34: 957 - 965.
- [9]. Harigan RA, Jones K. ABC of clinical electrocardiography. conditions affecting the right side of the heart. BMJ .2002 May; 324: 1201 - 1204.
- [10]. IromIbungo, K Susie, SaradaNingthoujam. Electrocardiographic changes in obstructive airway disease. Journal of medical Society. 2013 Jan volume 27; page: 19 -24.
- [11]. Soris R, Tenacallon A, Labrouses J, Massart JD, Lissac J. Changes in the electrocardiography in status asthmaticus . Int. J cardiol, 2002 Feb 15; 45 (4): 525 - 530.
- [12]. Batemen JR etal. Compersision of serial ECG and vector cariographic changes during recovery from status asthmaticus Thorax 2013 May; 32(3) 356 - 358.
- [13]. Rodrigo GJ, Rodrigo C, Hall JB, Acute asthma in Adults. Chest 2014 Jun; 125:181- 125.
- [14]. Stephen C, Lazarus, MD. Emergency Treatment of asthma. N Engl J Med 2015 Nov; 363: 755 - 764.
- [15]. Ryu JH, Daniels CE, Hartmen TE; Diagnosis and management of asthma, Myoclinproc. 2007 Dec, 82(8): 976 – 986.
- [16]. K Ramesh, Balaji. prasadNayk. A study of cardiovascular involvement in hypothyroidism. IAIM, 2016 Jan; 3:76 -80.
- [17]. Mirvis D M, Goldberger AL, Electrocardiography; in Braunwalds Heart disease 10th edition philadelphia 2015: page 345 - 365.
- [18]. Sterk, P. J. Fabbri, P. H. Quanjer, D W. Cockeroft, P. M. Airway responsiveness. standarizedchallange testing with pharmacological and physical stemuli in adults. Eur. Respi. J. 2018 Feb; 6: 53 - 83.
- [19]. Chazan R, Droszcz W. Electrocardiographic changes in patients with airway obstruction. Pol Arch Med, 2014Mar; 87: 237-410.
- [20]. Alan G Jap, Colin Robertson, Iain H. cause of sinus tachycardia, macleods clinical examination. 2012: pag 224-228.
- [21]. Chapman T, Barker P, Lung function tests and a vertical Pwave axis in the ECG. Thorax 2009 Jun; 39: 106-109.
- [22]. Gunstone, R. F Lary, D. Right heart pressures in bronchial asthma. Thorax 2012 Jan; 209: 39-45.
- [23]. Togha M, Sharifpour A, Ashraf H, Moghadam, Sahrian MA. Electrocardiographic abnormalities in patients with/ without cardiovascular diseases Annal of Indian academy of cardiology. 2013 Jan; 16(1): 66-71.

[24]. Diederik Boon, Jeroen van Goudoever, Jan J. Peick. Gert A. Hypertention. 2003 Dec; 41: 476-481.

[25]. Surawicz B, etal. AHA /ACCF /HRS, Recommendation for the standardization and interpretation of the electrocardiogram. Circulation 2009 Aug; 116: 234-241.

[26]. James, D. R and Lytle, M. British guideline on the management of asthma: SIGN Clinical guideline, 2016 Novenber; 141: 319-322.

[27]. Gupta D, Hansell A, Nichols T etal. Epidemiology of pneumothorax in Englannd. Thorax 2000 Feb; 55:666-671.

[28]. Castro - Rodriguez JA, Montaner AE, Garde - JG, Bernable JJ. The use of spirometry and peak flow meters in the diagnosis and management of asthma. Spanish National Guidelines for asthma 2004 Aug; 15 (4): 66-71.

[29]. Arifin WN, Sarimah A, Norsaadah B, NajibMajdi Y, Siti - Azrin A H, Aniza AA, Naing L. Reporting statistical results in medical journals. Malays J Med Sci. 2016 Oct; 23 (5): 1-7.

[30]. Reid P. T, Innes J A, Respiratory disease, Diagnosis of asthma. Davidson's principles and practice of medicine 22nd edition, 2014, page 667-678.

[31]. Dn costa J L, Chia B C. The ECG changes in bronchial asthma and their relationship to the severity of airways obstruction. Singapore Med J. 2014 April; 32: 101-115.

[32]. Biberman L, Sama R N, Surawicz B - T wave abnormalities during hyperventilation. Am Heart J. 2010 Feb, 81(2): 166-176.

[33]. Gordan D etal. Study of Electrocardiographic changes in acute asthma. Thorax 2016 Jan; 58(1): 81-88.

[34]. Barry E Bremer, Global Strategy for Asthma Management and Presentation, update GINA report. 2014: 16-19, and 65-66.

[35]. Crofton and Douglas's Asthma: Clinical Feature Respiratory Diseases. 2004 Mar; 34: 957-972.

[36]. Galb AF, etal. Ppulmonale in status asthmatics. J Allergy clin - Immunol. 2010 Dec; 38 (2): 64-81.

[37]. Sorialetal. Changes in electrocardiography in status asthmatics - Ann cardiolAngeiol. 2008 Feb; 33(3): 153-158.

[38]. EthimioujHasson A BetallRversibleTwave abnormalities in sever acute asthma. Respi. Med. 2013 Jun; 85 (3): 195-202.

[39]. Josephson G. w etal - Cardiac dysrrhythmias during the treatment of acute asthma. Chest, 2014 Feb; 78: 429-438.

[40]. Harrigion RA, Jones K, ABC of clinical electrocardiography -conditions affecting the right side of the heart. BMJ. 2002 May; 324: 1201-1204.

[41]. ZabMohseniferGujsooHoo. Practical Pulmonary and Critical care medicine, 2006 Jan V 213; 448-468.

[42]. Stefano F, Gabriella C San, Raffaels. Emergency treatment of asthma. N Engl. J Med. 2010 Aug 19; 363 (8): 755-764.

[43]. Samuel Y. Ash, M D. Alejandro A, Draz M D, M. P. H. The Role of Imaging in the Assessment of Severe Asthma. Curropinpulm Med. 2017 Jan; 23 (1): 97-102.