

ECG Changes in Acute Severe Asthma

Thesis

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Tist of Abbreviations

Abb.	Full term
ADAM22	Adapintangnin and matallannataingas 22
	Adesintergrin and metalloproteinase 33 Airways hyperresponsiveness
AH	
Bpm	-
	Cyclic adenosine monophosphate
	Dipeptidyl peptidase 10
	Electrocardiography
ET	•
ET-1	
	Environmental tobacco smoke.
	Forced expiratory volume in 1 sec
	Forced vital capacity
	Global Initiative for Asthma,
	G-protein related receptor for asthma
HR	
	Inhaled corticosteroids
<i>IFN-α</i>	•
O	\ldots Immunoglobulin E
<i>IL</i>	
=	Interquartile range
<i>IV</i>	
	Isovolumetric contraction time
	Isovolumetric relaxation time
	Long-acting beta2-agonists
	Long QT Syndrome
	Leukotriene receptor antagonist
LV	
	Myocardial contraction band necrosis
<i>Mg</i>	_
	Myocardial performance index
<i>PAC</i>	Premature atrial contraction

Tist of Abbreviations

Abb.	Full term
Da CO o	Canhan Diavida Drassavna
	Carbon Dioxide Pressure
	Plant homeodomain zinc finger protein 11
_	Pressurized metered dose inhalers
	Pulmonary systolic arterial pressure
<i>P-value</i>	. Probability
<i>PVC</i>	Premature ventricular contraction
<i>RAD</i>	Right axis deviation
<i>RAP</i>	. Right atrial pressure
	. Right bundle branch block
<i>RR</i>	Respiratory rate
<i>RV</i>	. Right-ventricle
<i>RVH</i>	Right ventricular hypertrophy
<i>SA</i>	. Sinoatrial
SABA	Short-acting B2 agonist
<i>SD</i>	Standard deviation
<i>SPINK5</i>	Serine protease inhibitor Kazal type 5
SVT	Supraventricular tachycardia
<i>TDE</i>	Tissue Doppler Echocardiography
<i>TFPV</i>	Transmitral flow propagation velocity
<i>Th</i>	. T helper
<i>TNF-α</i>	Tumor necrosis factor-alpha
<i>URTI</i>	Upper respiratory tract infection
<i>VBG</i>	Venous blood gases

Introduction

sthma is considered a heterogenous disease, characterized by chronic inflammation of the airways resulting in airflow obstruction, which may completely or partially reverse with or without specific therapy. Airway inflammation is the result of interactions between various cells, cellular elements, and cytokines (*Holgate and Polosa*, 2008).

Bronchial asthma is one of the most common chronic inflammatory disorders of childhood. The prevalence of bronchial asthma has further increased over the last decades, especially so in children, and still there is no sound explanation for this increase (*Akinbami et al.*, 2009).

In susceptible individuals, airway inflammation may cause recurrent or persistent bronchospasm, which causes symptoms that include wheezing, breathlessness, chest tightness, shortness of breath and cough, particularly at night (early morning hours) or after exercise (*Hargreave*, 2009).

It imposes a serious burden on patients, their family and the community. It causes respiratory symptoms, limitation of activities and flare ups, that sometimes require immediate medical attention and may be fatal (*Global initiative for asthma*, 2006).

Although bronchial asthma has been accused in causing arrythmias and affecting the cardiac function and anatomy

especially during the acute exacerbation attacks, there isn't much literature available discussing its effect on the heart, its function and electrophysiology (Shedeed, 2010).

It is essential to study the role of bronchial asthma in cardiac dysfunction, this will not only help clinicians to assess the risk of death and institute the appropriate level of care, but it will also help to explain the mechanism of death and to guide for other treatment (Salpeter et al., 2004).

AIM OF THE WORK

This work was done to study the various ECG abnormalities in acute asthma as well as the relation between the ECG abnormalities with severity of airway obstruction. Furthermore, the work included the effect of bronchodilators used on the ECG and their potential reversibility after the acute episode.