Management of Perioperative Bronchospasm

An Essay
Submitted For the Partial Fulfillment of Master Degree
In Anesthesiology

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2014

Acknowledgement

First and Foremost Thanks to **Allah**, the most gracious and merciful.

I would like to express my deepest thanks to ***Prof. Amr Essam El Din Abdelhameed***, Professor of Anesthesia,Intensive Care and Pain Management, Faculty of Medicine, Ain Shams University, for his perpetual help, close supervision and keen encouragement.

Also I would like to thank ***Dr. Sahar Mohammed Talaat,*** Lecturer of Anesthesia, Intensive Care and Pain Management, Faculty of Medicine, Ain Shams University, for her great help and advice, and for granting me a great deal of her time and effort.

I wish also to express my sincerest thankfulness to ***Dr. George Mikhail Khalil***, Lecturer of Anesthesia, Critical Care and pain management, Ain Shams University, for his continuous support and advice through the performance of every part of this work.

Finally, I'd like to express my gratefulness, heartfelt respect and renewed thanks to all my Professors, Family who helped me during the accomplishment of this work especially my Parents and my husband.

Dina El Sayed Ibrahim

كيفية التعامل مع إنقباض الشعب الهوائية المصاحب للعمليات الجراحية

رسالة توطئة للحصول على درجة الماجستير في التخدير

من

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بسم الله الرحمن الرحيم

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم

سورة البقرة
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ABG	:	Arterial Blood Gases
AIA	:	Aspirin-Induced Asthma
ARDS	:	Acute Respiratory Distress Syndrome
cAMP	:	cyclic Adenosine Monophosphate
CD	:	Cluster Of Differentiation
COPD	:	Chronic obstructive airway disease
CO_2	:	Carbon Dioxide
DVT	:	Deep Vein Thrombosis
ECG	:	Electrocardiogram
ECMO	:	Extracorporeal Membrane Oxygenation
ET_{CO2}	:	End Tidal Carbon Dioxide
ETT	:	Endotracheal Tube
FEV_1	:	Forced Expiratory Volume in One Second
FVC	:	Forced Vital Capacity
GA	:	General Anesthesia
H_1	:	Histamine$_1$ receptor
H_2	:	histamine$_2$ receptor
HMEF	:	Heat and Moisture Exchange Filter
ICU	:	Intensive Care Unit
Ig E	:	Immunoglobulin E
iPEEP	:	Intrinsic Positive End Expiratory Pressure
IPPV	:	Intermittent Positive Pressure Ventilation
IL	:	Interleukine
I:E	:	Inspiration:Expiration

IV	:	Intravenous
kPa	:	Kilopascal
LMA	:	Laryngeal Mask Airway
LT	:	Leukotrien
mg	:	Milligram
ml	:	Milliliter
MMP	:	Metalloproteinase
Na	:	Sodium
NFκβ	:	Nuclear Factor-κβ
NSAIDs	:	Non Steroidal Anti-Inflammatory Drugs
nTS	:	Nucleus Tractus Solitarii
Pa CO2	:	Partial Pressure Of Arterial Carbon Dioxide
Pa O2	:	Partial Pressure Of Arterial Oxygen
PE	:	Pulmonary Embolism
PEEP	:	Positive End Expiratory Pressure
PFT	:	Pulmonary Function Test
PG	:	Prostaglandin
SaO2	:	Oxygen Saturation
TEE	:	Trans Esophageal Echocardiography
TNFα	:	Tumor Necrosis Factor Alpha
TTE	:	Trans Thoracic Echocardiography
URTI	:	Upper Respiratory Tract infection
UAO	:	Upper Airway Obstruction
V/Q	:	Ventilation/Perfusion
UK	:	United Kingdom

INTRODUCTION

Bronchospasm is an anesthetic emergency that can lead to disastrous outcomes if treatment is irresolvable. An anesthesia provider must immediately initiate treatment if bronchospasm is suspected in order to avoid negative sequelae ***(Linck , 2007).***

Bronchospasm encountered during the perioperative period and especially after induction/intubation may involve an immediate hypersensitivity reaction including IgE-mediated anaphylaxis or a nonallergic mechanism triggered by factors such as mechanical (*i.e.*, intubation-induced bronchospasm) or pharmacologic-induced bronchoconstriction in patients with uncontrolled underlying airway hyperreactivity (for example, due to histamine-releasing drugs such as atracurium or mivacurium). The differential diagnosis includes inadequate anesthesia, mucous plugging of the airway, esophageal intubation, kinked or obstructed tube/circuit and pulmonary aspiration. Unilateral wheezing suggests endobronchial intubation or an obstructed tube by a foreign body (such as a tooth). If the clinical symptoms fail to resolve despite appropriate therapy, other etiologies such as pulmonary edema or pneumothorax should also be considered ***(Dewachter et al., 2011).***

Active and passive heavy smokers, patients suffering from chronic obstructive pulmonary disease and children with mild to moderate upper respiratory tract infections have a high incidence of bronchospasm at extubation ***(Karmarkar and Varshney, 2008).***

Bronchospasm during general anesthesia can present in isolation or as a component of a more serious underlying pathology such as anaphylaxis. It is characterized by prolonged expiration, wheezes and increased peak airway pressures during Intermittent Positive Pressure Ventilation (IPPV). If untreated, it can cause hypoxia, hypotension and increased morbidity and mortality. Suspected bronchospasm during anaesthesia should be assessed and treated promptly. Ongoing management should address the underlying cause ***(Westhorpe et al., 2005).***

AIM OF THE WORK

The purpose of this Essay is to identify the causes of bronchospasm in the perioperative period and provide methods for their early detection and management.

PATHOPHYSIOLOGY OF PERIOPERATIVE BRONCHOSPASM

Bronchospasm and wheezes are common features of reactive airway disease. Patients with bronchial asthma and some with chronic obstructive pulmonary disease (COPD) show hyperreactive airway responses to mechanical and chemical irritants. In these groups there is a combination of constriction of bronchial smooth muscle, mucosal edema and mucus hypersecretion with plugging. Perioperative bronchospasm in patients with reactive airway disease is however relatively uncommon. In patients with well-controlled asthma and COPD the incidence is approximately 2%. The overall incidence of bronchospasm during general anaesthesia is approximately 0.2% ***(Olsson , 1987).***

Exposure to tobacco smoke, history of atopy and viral upper respiratory tract infection (URTI) all increase the risk of bronchospasm during anaesthesia. In many patients with bronchospasm during anaesthesia there is no history of reactive airway disease ***(Pepe and Marini, 1982).***

Gas exchange abnormalities, particularly hypoxemia, are well known accompaniments of exacerbations of asthma. The degree of hypoxemia shows a poor correlation with indices of

severity of airflow obstruction. Several studies have indirectly recognized ventilation perfusion (V/Q) mismatching as the fundamental mechanism underlying abnormal gas exchange in asthma ***(Ballester et al., 1989).***

In asthmatic patients, the distributions of V/Q ratios were typically characterized by a bimodal pattern in blood flow distribution, with a significant proportion of the cardiac output (approximately 25%) perfusing lung units with low V/Q ratios (< 0.1). There was no shunt and there were no areas with high V/Q ratios, and dead space was normal. The bimodal pattern of blood flow distribution was attributed to the effectiveness of collateral ventilation, which prevented complete airway occlusion by mucus plugging, edema, and/or bronchoconstriction. This explains the absence of the development of shunt and the failure of the low V/Q units to collapse beyond the occluded airways ***(Lagerstrand et al., 1992).***

Likewise, it is consistent with the hypothesis that the changes involved in abnormalities of pulmonary gas exchange take place peripherally rather than centrally, where collateral ventilation is less likely to play a major role. The presence of this bimodal pattern with modest or no airflow limitation is most consistent with persistent peripheral airway involvement ***(Schmekel et al., 1994).***