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Review Article

Review Article on Asthma

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ABSTRACT

Asthma is a long-term inflammatory condition that affects the airways, leading to breathing difficulties, wheezing, coughing, and chest tightness. It is marked by airway inflammation, hyperresponsiveness, and variable airflow obstruction. The immune system plays a significant role in asthma, with cells such as mast cells, eosinophils, and T cells contributing to airway inflammation. Elevated nitric oxide levels serve as an indicator of inflammation in asthmatic patients. Asthma is classified based on its underlying inflammatory patterns, including T2-high and T2-low types. Proper management involves following treatment guidelines, using inhaled corticosteroids, and identifying potential triggers. Poor asthma control is commonly linked to inadequate treatment adherence, misdiagnosis, or severe disease forms. This study examines the mechanisms, classification, and management of asthma while emphasizing the need for timely diagnosis and appropriate therapy to enhance patient well-being.

INTRODUCTION

Asthma is a chronic inflammatory disease of the airway characterized by airflow obstruction and bronchial hyper-responsiveness. Varying clinical features in heterogeneous demographic population leadsto diverse clinical phenotypes (1). Higher prevalence is seen in women, African Americans, and in those from a lower socioeconomic status (2). In 2016, the Centers for Disease Control and Prevention (CDC) reported that 8.3 percent of the adult population in the United States (> 20 million) suffer from asthma (2). Within the last decade, over 400,000 annual discharges 1.7 million annual emergency department Visits were reported among Americans with Asthma as the primary diagnosis (2,3). The Average cost of a hospital admission for Treatment in 2010 was $9,000 per case, with A total cost of $2.9 billion (4). The estimated Health care (both inpatient and outpatient) Cost of asthma in 2011 was $56 billion (3).Despite specific guidelines for in-hospital Management, medical audits have shown Inconsistent compliance by healthcare Providers (5). We review the evaluation and Management of asthma exacerbations in adult Patients requiring

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hospitalizations. Specifically, objective assessment of severe Exacerbations, factors that help to determine the appropriate level of care based on Severity, management of the asthmatic Airway, and discharge planning will be discussed.

Asthma Classification

Asthma has been broadly classified as:

1. Type 2 (T2) high inflammation
2. T2-low inflammation

T2-high inflammation is currently the best-understood inflammatory endotype. Exposure to a detector antigen-Presenting cell induces a T-helper cell type 2 (Th2) response. Th2 cells secrete cytokines such as interleukins 4, 5, and 13, which drive the downstream recruitment of effector cells and facilitate the isotype switching of B Cells to secrete immunoglobulin E (IgE). IL-4 and IL-13 promote subepithelial fibrosis, airway remodeling, Mucus hypersecretion, and airway hyperreactivity. T2-low inflammation accounts for about 50% of cases, where eosinophilic airway inflammation is absent. A Different class of T lymphocytes, known as Th17 cells, secretes IL-17, which is associated with neutrophilic

TREATMENT

Patients with acute asthma Exacerbation require prompt treatment and Evaluation. The main goals of therapy are to Relieve airflow obstruction, improve work of Breathing, and maintain oxygenation and Tissue perfusion. Key pharmacological Components of acute asthma therapy include Serial or continuous short-acting Bronchodilators and systemic steroids. Bronchodilators: Beta-adrenergic agonism Has long been the mainstay of asthma Treatment dating back when ephedra, a Chinese herbal remedy was used for Wheezing (19). Epinephrine was the first Synthetic bronchodilator in early 1900s; Followed by isoprenaline (19,20). Unfortunately, many of the earlier agents Exhibited both alpha- and beta-adrenergic Agonism, resulting in tachycardia and Hypertension (20). The first beta-2 specific Therapy, salbutamol, became available in 1969 and remains the most globally used Bronchodilator to this day (20). Inhaled short-Acting beta-2 agonist (SABA) therapy is the Cornerstone of acute asthma management. Inhaled SABA can be administered as Frequent as every 15 minutes or as a Continuous nebulization for refractory cases. Adding ipratropium bromide (a muscarinic Agonist) to beta-2 agonist therapy provides Improved bronchodilation when compared to Beta-2 agonist alone (1,5). Lactic acidosis is A known adverse effect of with high doses of Inhaled beta-2 agonists (21,22). During Asthma exacerbations, both type A lactic Acidosis (from anaerobic metabolism due to (Hypoxia) and type B lactic acidosis (decreased lactic acid metabolism from beta Agonists) have been postulated to occur (22). Beta-adrenoreceptor activation causing an Increase in plasma glucose and free fatty Acids increase pyruvate levels. Free fatty Acids also block the conversion of pyruvate to Acetyl-coenzyme A. This excess pyruvate is Converted to lactic acid by pyruvate Dehydrogenase (22). Hence, exercising Vigilance for worsening of metabolic acidosis in those receiving frequent or continuous Beta-agonists, may be necessary.

Systemic steroids: Corticosteroids are Essential in the control of ongoing Inflammation and the stabilization of acute Asthma exacerbation. The addition of Corticosteroids reduces mortality, relapses, And subsequent hospital admission in acute Asthma exacerbation (5). A meta-analysis of 12 emergency department studies suggests That early therapy with systemic Corticosteroids (within 1 hour) yielded Significantly lower rates of

hospital Admission. Benefit from corticosteroids is Manifested >6 hours after administration. Therefore, we recommend continued Monitoring in the emergency department for At least 6 hours after corticosteroid Administration to determine its assumed Favorable impact (5,23). Of note, adults Included in this meta-analysis only received corticosteroids, so no recommendation Can be made on the effect of oral Corticosteroids in adults with asthma Exacerbation. The most commonly used. Dosages of steroids were hydrocortisone 500mgIV (N=1study) and Methylprednisolone 125mg IV (N=5 studies)(23) For patients admitted to the hospital Ward, the recommended doses of Corticosteroids are oral prednisolone 1mg/kg (with a maximum dose of 50mg) daily or Intravenous hydrocortisone 400mg daily (often dosed as 100mg injections every 6 Hours). Currently, there is insufficient Evidence to distinguish between intravenous Versus oral route of administration. However, The British Thoracic Society recommends Oral route of administration as there is no Significant difference in efficacy between Oral and intravenous corticosteroids (5). Corticosteroid therapy should be continued for at least five days or until recovery (5,24). Both the BTS and the EPR3 guidelines Recommend concurrent use of ICS with Systemic steroids (1,5). Clinicians should Ensure that ICS is at least started prior to Discharge and continued thereafter (5,24).

Magnesium Sulfate: Intracellular Magnesium, which is important for airway Smooth muscle relaxation and possibly mast Cell release of histamine, was found to be low in patients with acute asthma exacerbation(25). Serum levels of magnesium have not Found to correlate to intracellular magnesium Stores, therefore are not markers of risk or Intracellular magnesium depletion and should not be monitored (25). A single dose of 1.2-2g of magnesium sulfate IV has been Shown to be safe and reduce hospital Admission and intubation rates in asthma Exacerbation (5). Magnesium sulfate should Be considered in patients with PEFR<50% Who have not had a significant response to Inhaled bronchodilators (5). Repeated doses Have not been studied and may result in Hypermagnesemia precipitating respiratory Muscle weakness. Recent interest in Nebulized magnesium in a sub-group of Severe asthmatics unresponsive to standard Treatment had prompted meta-analyses Suggesting modest benefit (26,27). However, more robust investigations are warranted Before a we can advise this modality for acute Treatment of severe asthma.

Aminophylline/Theophylline: During an Acute exacerbation, aminophylline or Theophylline does not result in significantly More bronchodilation when compared to Inhaled bronchodilators and steroids. The use of these medications is not recommended for Acute treatment (1,5). If patients are being Treated with these agents on an outpatient Basis, it is reasonable to check a serum level to ensure that the patient is within the Therapeutic window (1).

Airway management, oxygen and mechanical Ventilation: Hypoxemia should be treated with supplemental oxygen in all cases of Asthma exacerbation with a goal SpO2 of 93-95% (1,5,23). If the patient is awake and meets the criteria for a severe asthma Exacerbation as defined in Table 3, frequent Reassessments are necessary. Ominous signs of “life threatening” asthma in whom Intubation should be considered include Mental status changes, normalization or high PaCO2, a silent chest (very severe Bronchospasm), escalating oxygen Requirements, or failure of response to Treatment (12,28). A wide diameter Endotracheal tube (size 8 Fr), rapid sequence Intubation with ketamine or propofol as the Preferred sedating agent, increased Expiratory

time (by reducing respiratory rate in controlled modes), and increased Respiratory flow rate are some considerations While using mechanical ventilation (28-30). Hypercapnia often ensues due to reduction in Minute ventilation from reduced respiratory Rate and this is allowed (permissive Hypercapnia) to avoid worsening of auto-PEEP. In most cases patients tolerate it, sometimes to pH as low as 7.2 and PCO2 as high as 90 mmHg. When minute ventilation cannot be decreased on spontaneous modes, increasing sedation and neuromuscular paralysis should be considered to decrease the risk of barotrauma and avoid dys-synchrony (30,31).

Antibiotics: A recent systematic review of antibiotic use in asthma exacerbations evaluated 6 studies and 681 patients and found very limited evidence for benefit from antibiotics during asthma exacerbations in those without signs and symptoms of infection. Limitations were an overall low quality of evidence due to a low number of patients overall, poor outcome measures (specifically hospital admission, ICU admission, and repeated exacerbations), and limited information on the side effects of antibiotic use. Guidelines recommend against routine use of antibiotics in acute asthma exacerbations (1,5).

Rescue therapies: Medications and interventions such as Heliox (21% O2 and 79% Helium) may be helpful in the setting of acute exacerbation because helium has a lower density and higher viscosity than nitrogen, both of which allow for more laminar air flow in constricted airways. This offloads the patient's work of breathing, but there is no data supporting routine use of Heliox (5,33). A systematic review concluded that Heliox did not significantly impact the rate of hospital admission (RR 0.83; 95% CI 0.66-1.08). Sevoflurane is an anesthetic agent which causes bronchodilation via a voltage-dependent calcium channel and modulation of intracellular cyclic adenosine monophosphate levels. Multiple case reports have shown promising results of sevoflurane in refractory, mechanically-ventilated asthmatic patients . Sevoflurane appears to be well tolerated in children with life-threatening asthma, with clinical improvement within an hour of administration and improvement in peak pressures, PCO2, and pH within two hours of administration

Hospital and Indirect Costs

- **Hospital Costs**: Hospitalization generally occurs when asthma management has failed to prevent an acute severe attack, leading to high expenses.
- **Indirect Costs**: Indirect costs include loss of work productivity, premature withdrawal, and time spent by Caregivers. These costs vary based on patient age and disease severity.

Minimum Treatment

Minimum Treatment refers to the lowest dose of inhaled steroids required to maintain asthma control in Phase 1 for one month. This is synonymous with maintenance treatment.

Study Treatment

The study treatment was directed by two doctors in each of the four centers in an effectiveness (rather than Efficacy) manner to make it more applicable to routine clinical practice. Treatment followed the Canadian Asthma Consensus Group Guidelines.

Strategy management included:

1. Avoidance strategies for allergens and intolerance to non-steroidal anti-inflammatory medicines.

2. Patient education regarding treatment adherence, reinforced at each visit in Phases 1 and 2.
3. Review of factors affecting asthma control, such as rhinosinusitis, nasal cysts, and gastroesophageal Reflux, along with their treatment.

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