Acute severe asthma: A case presentation

Sona Chaturvedi[1*], S B Mohan[1]
[1]Department of Anaesthesia and Intensive Care, Gulf Medical College Hospital & Research Centre, Ajman, UAE

*Presenting Author

ABSTRACT
Acute asthma is an episode of progressive increase in shortness of breath, cough, wheezing, or chest tightness, or some combination of these symptoms. If not treated immediately there will occur increase in flow resistance causing increased work of breathing, gas exchange inefficiency, respiratory muscle exhaustion and finally hypercapnic and hypoxemic respiratory failure. Acute severe asthma carries a high morbidity and mortality. Globally over 180,000 people die from asthma each year. A quarter of the adults is reported missing work because of asthma. This presentation discusses a patient, a smoker, with known history of bronchial asthma who suddenly developed an acute attack of severe asthma following an injection of Augmentin. She was initially managed as a case of anaphylaxis and subsequently shifted to the ICU and managed for severe asthma. Due to the severity of the attack she rapidly desaturated and was intubated and provided mechanical ventilatory support. Over three days on the ventilator her condition gradually improved and she was extubated on the 4th day. She was also prescribed bronchodilators, steroids and other supportive medications. Laboratory investigations included complete blood count, C reactive protein, and serial arterial blood gases. Post-extubation, she was stable and was advised to continue Tab. Augmentin, Prednisolone, and nebulisation with $\beta_2$agonist (Albuterol), anticholinergic (Ipratropium bromide) and steroid (Budesonide). She was also advised to quit smoking. She has since been doing well.

Key words: acute, severe, bronchial asthma, management

INTRODUCTION

Acute severe asthma is one of the most common causes of visits to hospital emergency departments worldwideand accounts for the greater part of the economic and healthcare burden from the disease[1]. Failure to appreciate the severity, inadequate emergency treatment, and delay in referral may lead to increased mortality and morbidity[2]. Asthma is best described as a chronic inflammatory disorder of the airways, characterised by episodic dyspnoea, cough, wheezing, and chest tightness. It is associated with hyper-responsiveness of the airways to a variety of environmental stimuli that may resolve either spontaneously or with treatment[3]. Acute severe asthma is an asthmatic exacerbation characterized by persistent dyspnea that is not relieved by the usual standard therapy with bronchodilators within 30 mins to one hour.

CASE REPORT

A 24 year-old female presented to the GMC Hospital emergency room with breathing difficulty. The patient was a known case of bronchial asthma since three years of age. She was on β2 agonist with or without anticholinergic puffs as on need basis. She used to smoke sheesha as well as half packet cigarette each day. There were no known drug allergies or any other positive medical or surgical history. Neither had she been hospitalized for similar complaints in the past.

On presentation to the ER, she was hemodynamically stable, and auscultation revealed B/L wheeze. Chest X ray showed B/L infiltrates with increased bronchovascular markings. She was immediately nebulised with Salbutamol and Ipratropium and admitted to the ward, with advice to give Inj. Augmentin, Inj. Hydrocortisone and oxygen supplementation by mask.

Five minutes following administration of Inj. Augmentin (after an equivocal test dose), she became restless and had severe breathing difficulty, and within the next few minutes her saturation dropped to 65%. An empirical diagnosis of anaphylactic drug reaction was made and the patient was given Inj. Adrenaline, subcutaneously and hydrocortisone and chlorpheniramine intravenously. She was shifted to the ICU, by which time she was severely breathless, unable to speak and had developed carpo-pedal spasms. Her heart rate and BP increased and saturation dropped further. She was immediately given IV Midazolam and Atracurium, intubated and provided ventilatory support (IPPV) with low tidal volume, low frequency and initial fractional concentration of oxygen of 100% which was gradually reduced to 60%. Maintenance infusions of Midazolam and Atracurium were started. Other medications prescribed included IV Augmentin, Hydrocortisone, Pantaprazole, subcutaneous LMWH, insulin infusion and nebulisation with Salbutamol and Ipratropium. Relevant specimens were sent to the laboratoryfor investigation. On the second day, the ventilatory mode was changed to SIMV. She was febrile with elevated total counts. She continued to have tachycardia and extensive ronchi on both sides. The following day, her chest was clear and Atracurium infusion was discontinued, midazolam infusion was tapered and mandatory frequency was reduced to 5/min. Early the next morning, midazolam infusion was discontinued and she was weaned to CPAP and then extubated. She showed good recovery with hemodynamic stability and was transferred to the ward the next day. On discharge from the ward she was advised to continue Tab. Augmentin, Prednisolone, and nebulisation with Albuterol, Ipratropium and Budesonide. She was also advised to quit smoking. The patient came for follow up three weeks after discharge and was asymptomatic, though auscultation revealed inspiratory ronchi. She was advised to continue Ciclisonide puff daily and Albuterol puff as required. She again attended the OPD another three weeks later and was reportedly doing fine.

DISCUSSION

Clinical Presentation

Acute Asthma is an episode of progressive increase in shortness of breath, cough, wheezing, or chest tightness, or some combination of these symptoms.

Most patients suffering from acute asthma present with a constellation of complaints consisting of dyspnea, cough, and wheezing[4,5]. No sign or symptom is uniformly present. Dyspnea is absent in 17 to 18% of cases[5] and wheezing is absent in 5%[4,5]. The physical signs that are encountered aretachypnea, tachycardia, wheeze, hyperinflation, accessory muscle use, pulsus paradoxus, diaphoresis, cyanosis, and obtundation. The use of accessory muscles is observed in about 30% of cases at presentation, a paradoxical pulse in 15–20%, sweating in 12%, and cyanosis in less than 1%[4,5]. Sweating, the use of accessory muscles, a paradoxical pulse,and the inability to communicate in full sentences are all associated with the presence of substantial airway narrowing[6].

Pathophysiology

Acute severe asthma involves a nonuniform, reversible increase in airway resistance that results in diminished flow rates, premature airway closure, hyperinflation of the lungs and thorax, increased work of breathing, changes in elastic recoil, and frequency-dependent behavior[7-10]. In addition, there is abnormal distribution of ventilation and perfusion and altered arterial blood gases[8].

Differential Diagnosis

Chronic obstructive lung disease, bronchiectasis, endobronchial lesions, foreign bodies, extra- or intrathoracic narrowing of the trachea, cardiogenic and noncardiogenic pulmonary edema, pneumonia, and pulmonary emboli[12,13].

Immediate Management of Acute Severe Asthma[11]

- Supplemental oxygen (sitting position) – humidified, high

Table 1: Comparison between Acute severe and life threatening asthma[11]

SYMPTOMS	ACUTE SEVERE	LIFE THREATENING
Dyspnea	At rest	
Speech	Single words, not sentences of phrases	
Alertness	Agitated	Drowsy or confused
SIGNS		
Respiratory rate	≥25 breaths/min	Poor respiratory effort
Heart rate	≥110 beats/min	Arrhythmias
Pulsusparadoxus	>25 mmHg	Absence (muscle fatigue)
Use of accessory muscles	Evident	Abdominal paradox
Wheeze	Present – loud	'Silent chest'
FUNCTIONAL ASSESSMENT		
PEF	33 - 50% of predicted/best	<33% of predicted/best
PaO_2	<60 mmHg	Severe hypoxia
$PaCO_2$	>42 mmHg	Normo/hypercarbia
SaO_2		< 92%

concentration using a reservoir bag with an aim to maintain SpO2 between 94 – 98%.

- Inhaled B2 agonists are the first line of treatment.
- Inhaled anticholinergics (adjunct to B2 agonists).
- IV steroids, to be continued as oral after the acute phase of the illness.
- Antibiotics if indicated.
- IV fluids

Other Therapeutic Options

Magnesium sulphate, Ketamine, volatile anaesthetics[14,15], heliox[16], adrenaline[16], mucolytics, bronchoscopy, leukotriene antagonists/modifiers[17].

Mechanical Ventilation

Aim is to improve gas exchange, to reduce the work of breathing and to avoid complications while maintaining optimal conditions for recovery. The indications could comprise at least two of the following conditions: (1) moderate–severe dyspnea, with use of the accessory muscles and abdominal breathing; (2) hypercapnic acidosis (pH<7.35); and (3) tachypnea (>25bpm). The most widely employed ventilatory modes are supportive pressure with PEEP and the BIPAP modes if the respiratory stimulus proves insufficient[18].

Ventilatory Strategies[19]

- Volume controlled modes.
- Low tidal volumes (5-7ml/kg) to reduce barotrauma.

- Long I:E ratios; increasing expiratory times reduces auto PEEP.
- Low respiratory rates
- Extrinsic PEEP

Criteria for starting weaning[20]

Adequate oxygenation: PaO2 ≥60 mm Hg on FiO2 ≤ 0.4
PaO2/FiO2 = 150–300 with PEEP≤5 cm H2O

Hemodynamic stability: No myocardial ischemia or significant hypotension
Temperature: < 38°C

Hemoglobin: ≥8–10 g/dl
Adequate mental status: Patient awake or easily aroused

Discharge and After care

- Criteria for Discharge Home: Patients should be on reducing amounts of β2 agonist and on medical therapy they can continue safely at home. Evidence suggests that patients discharged with PEF <75% best or predicted and with diurnal variability>25% are at greater risk of early relapse and readmission[21,22].
- Home Treatment: Inhaled B2-agonist, either alone or in combination with an anticholinergic, alongwith oral steroid.
- Close medical follow-ups are to be emphasised.
- "Be in Control" asthma action plan from Asthma UK
    1. Personal asthma action plan (what to do if symptoms change)
    2. Peak flow diary
    3. Asthma medicine card
    4. Making the most of your asthma review(discuss with doctor/nurse)

Practical tips for improving compliance[11]

*Ask open-ended questions like "If we could make one thing better for your asthmawhat would it be?" This may help to elicit a more patient-centered agenda.
*Make it clear you are listening and responding to the patient's concerns and goals
*Reinforce practical information and negotiated treatment plans with written instruction
*Consider reminder strategies
*Recall patients who miss appointments

CONCLUSION

Acute severe asthma carries a high morbidity and mortality. It may be classified as mild/moderate/severe or life threatening. Peak Expiratory Flow rate is a good indicator of severity of the attack. B2 agonists by nebulisation are the first line of therapy for acute severe asthma. Nebulisation with anticholinergics is an adjunct and has no role as the sole medication in an acute asthmatic attack. Steroids are to be given in all patients, but antibiotics to be prescribed only where indicated. Other modalities of treatment include Magnesium, ketamine, volatile anaesthetics, heliox. Mechanical ventilatory strategies are aimed at maintaining oxygenation while avoiding hyperinflation & barotrauma. Permissive hypercapnia is acceptable.

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