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Intensive Care Management of Acute Respiratory Failure in a Severe Asthmatic Patient in a Developing Hospital- Case Report

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Abstract: Miss LTA, a 28 year old, known asthmatic presented to the accident and emergency unit of Federal Medical Centre, Gombe with 30 minutes history of breathlessness, wheezing, chest tightness and altered consciousness. Examination revealed a young a lady who was restless, dysphoeic and drowsy, with central cyanosis. Respiratory rate was 42/min and rhonchi on auscultation. Heart rate was 130 per minute; oxygen saturation was 50% with pulsus paradoxus of 20. An assessment of respiratory failure in acute severe asthma was made. She had bronchodilators, endotracheal intubation and mechanical ventilation in the intensive care unit for 48 hours. She made a full recovery and was discharged on the 8th day of admission on salbutamol inhaler and tablets of prednisolone.
Keywords: Intensive care management, respiratory failure, severe asthma, developing hospital.

INTRODUCTION

Asthma is a chronic inflammatory disease of the airways which results in increase responsiveness of the lower airways to multiple stimuli and subsequent reversible obstruction and this manifests clinically with cough, chesty tightness, dyspnoea and wheezing [1].

The prevalence is increasing and over 150 million people are believed to have asthma worldwide.[1] Asthma is multi-factorial in origin, arising from the interaction of both genetic and environmental factors. Airways inflammation characterizing asthma occurs genetically hiccup here susceptible individuals are exposed to environmental factors e.g. aero-allergens which stimulate the production of IgE [1].

Acute severe asthma is a term describing life-threatening attacks of asthma. Patients are usually extremely distressed; tachypoeic and the lungs are hyper-inflated. They also have tachycardia, pulsus paradoxus and central cyanosis[2]. The aim of management of acute severe asthma is to prevent death, to restore the patient's pulmonary function as quickly as possible, and to prevent early relapse. Treatment involves oxygen therapy, use of B-adrenoceptor agonist and corticosteroids.

All severely ill asthmatic patients should be admitted to an intensive care unit and many of such patients require intubation and mechanically ventilation. In Nigeria Intensive Care Units (ICU) are parts of anaesthesia departments and critical care constitutes a substantial part of the workload and responsibilities of anesthetists.

The prognosis of individual asthma attacks is generally good. There is occasionally a fatal outcome, especially if treatment is inadequate or delayed. Therefore, the aim of this report is to bring out the positive role played by the intensive care unit in the management of acute severe asthma and to encourage the establishment of more ICU in the developing hospitals.

CASE REPORT

Miss LTA, a 28 year old lady, was rushed into the accident and emergency unit of the Federal Medical Centre, Gombe with a 30 minute history of breathlessness, restlessness and altered level of consciousness. She was a known asthmatic that was discharged from the emergency unit 8 hours earlier, during which she presented with difficulty in breathing, wheezing and coughs with associated chest tightness that was precipitated by exercise.

The patient was a member of the National Youth Service Corp (NYSC) and the informant was the NYSC camp medical doctor. She had four attacks within 3 days in the NYSC camp. These were treated in the camp clinic. Most of the attacks were in the mornings and precipitated by exercise or inhalation of dust. She neither smoked cigarettes nor took alcohol.

Her mother was also an asthmatic. She was not a known hypertensive or diabetic. She had been on salbutamol inhaler, 5mg of prednisolone tablets and 960 mg of cotrimoxazole tablets twice a day for three days prior to presentation.

On examination, she was a young lady, restless, dyspnoeic, sweating, drowsy, not pale, afebrile but with cyanosis of the lips and tongue. Her weight was 76kg. The respiratory rate was 42 cycles per minute with intercostal and subcostal recession, hyper-inflated lungs with hyper-resonant percussion note. There was decreased air entry, with rhonchi on auscultation. The heart rate was 130 beats per minute and oxygen saturation was 50% in room air. The blood pressure was 140/70 mmHg during expiration and 120/70 mmHg during inspiration. There were first and second heart sounds with no murmurs. The abdomen was full, moved with respiration, and no organ was palpable.

A diagnosis of acute severe asthma was made. While history and physical examination was going on, the patient was started on humidified 100% oxygen by polymask at a flow rate of 6 litres per minute. Intravenous access was secured with a size 18G cannula after which intravenous hydrocortisone 200mg was given followed by Intravenous Aminophylline 250mg bolus given slowly over 15 minutes. The results of investigations were: RBS, FBC and CXR. RBC was 5.1mmo1/L. A consult was sent to the anesthetists to review the patient for possible admission into ICU.

ICU Management

The anesthetists review was done in the accident and emergency unit. The history and examination are as documented above.

A diagnosis of respiratory failure (stage V asthma) due to acute severe asthma was made. The immediate plan was for endotracheal intubation and transfer into the ICU for mechanical ventilation. The vital signs were: pulse rate of 128 per minute, oxygen saturation of 56% and blood pressure of 130/70 mmHg.

The patient was allowed to breathe 100% oxygen using a Mapleson A circuit (Magill's circuit) and facemask for 5 minutes. She was given intravenous ketamine 100mg and cricoids pressure applied by an assistant. When patient was asleep intravenous suxamethonium 100mg was administered. After laryngoscopy, intubation was carried out with a size 7.5mm ID cuffed endotracheal tube. The chest was auscultated for bilateral air entry and the endotracheal tube cuff was inflated. The cricoid pressure was released and oropharyngeal air way inserted. The endotracheal tube was secured with adhesive plaster. The patient was ventilated manually for 10 minutes using FiO_2 of 1. The oxygen saturation improved from 56 to 80%. Additional intravenous ketamine 25mg was given before the patient was transferred to the ICU. During transport to the ICU, the patient was ventilated using an Ambu bag attached to the endotracheal tube while the pulse rate and oxygen saturation were monitored.

In the Intensive Care Unit, she was placed on the Bear 1000 ventilator (continuous flow, time cycled, pressure limited mechanical ventilator) for mechanical ventilation. The initial mode was controlled mechanical ventilation with FiO_2 of 1 which was later reduced to 0.5. Tidal volume was 0.6L, with a respiratory rate of 12 per minute. The inspiration: expiration ratio was 1:1. Initial sedation was with 500mg of ketamine added to 500ml of 5% dextrose water to run at 100mls/hour and intravenous diazepam 20mg in 500ml of 0.9% saline which was infused at 100mls per hour. Neuromuscular paralysis was with intravenous pancuronium 4mg, 4 hourly for 24hours. Other medications included intravenous aminophylline 250mg diluted to 20ml given by continuous infusion with a syringe pump at the rate of 2ml every hour, iv hydrocortisone 200mg stat, iv ciprofloxacin 200mg 12 hourly for 48 hours.

Patient monitoring include the pulse rate, oxygen saturation, air entry/breath sound, temperature and urine output. The vital signs after starting mechanical ventilation were a pulse rate of 120 beats per minutes, oxygen saturation of 95% and blood pressure was 130/70mmHg. Airway pressures were also monitored. After 5 hours of starting ketamine sedation, the sedative agent was changed to propofol. Propofol 500mg was added to 500ml of 5% dextrose water to run at 100ml per hour. Diazepam sedation was also stopped.

After 24 hours (2^{nd} day) on the ventilator, the patient's vital signs improved as evidenced by oxygen saturation of 98% on FiO_2 of 021, pulse rate of 90/min and blood pressure of 130/70 mmHg. The pancuronium was discontinued and intravenous atropine 1mg and neostigmine 2.5 mg were given to antagonize the residual effect of pancuronium. The ventilator mode was changed to synchronized intermittent mandatory ventilation with a FiO_2 of 0.21.
On the morning of the 3^{rd} day in ICU, she made good respiratory effort and so she was disconnected from the ventilator. Propofol sedation was also discontinued. The endotracheal tube was left in situ and supplemental oxygen using a catheter was continued through the tube. Vital signs remained stable with oxygen saturation of 97-99%.

Four hours later, the patient was fully conscious (obeyed command) and attempted to extubate herself, as such she was extubated. Post extubation, the

patient was stable with no signs of respiratory distress and maintained good oxygen saturation.

On the 4th day in ICU, intravenous fluids, intravenous antibiotics and intravenous aminophylline were discontinued. She was placed on tables of ciprofloxacin 500mg 12 hourly for five days, tablets of prednisolone 30mg daily and tablets of salbutamol 4mg 8 hourly. Lung function tests were carried out and the result together with other investigations were as follows:

		Test Values	Normal Values
• PCV		36%	
• White Blood Count		8.8x10^9/L	(4-11.0x10^9)
• Neutrophils	-	60%	(40-75%)
• Lymphocytes	-	25%	(20-45%)
• Eosinophils	-	13%	(1-6%)
• Basophils	-	2%	(0-2%)
• ESR	-	27mm/hr	(10-15mm/L)
• Lung Function Test			
• Peak expiratory flow rate (PEER)-		300/min	450L/min
• Forced expiratory volume in one second (FEV_1)		2.8L/min	
• Forced vital capacity		4.0L	5.L/min
• Urea and electrolyte			
• Na+	-	132 mmo1/L	(134-145 mmo1/L)
• K+	-	3.4 mmo1/L	(3.5-5.5 mmo1/L)
• C1-	-	102 mmo1/L	(96– 106 mmo1/L)
• H CO_3	-	22 mmo1/L	(21-31 mmo1/L)
• Urea	-	5.8 mmo1/L	(2.5–6.6 mmo1/L)

On the same day, the patient was transferred to the ward. In the ward, Prednisolone was tailed down to 20mg daily and then later to 10mg daily. Salbutamol tablets were changed to salbutamol inhaler.

On the 8th day after admission into the hospital, she was discharged home on 5mg of prednisolone tablets twice a day for one week and salbutamol inhaler. She was advised to avoid precipitating factors and the need for compliance with her medication. The assistant director of NYSC was informed about her medical condition.

DISCUSSION

Pathologic studies of persons with fatal asthma clearly demonstrated the inflammatory nature of the disease. Typically, the lungs of such patients are over distended, with mucus exudates that obstruct the air way lumen. The thick and tenacious mucus plugs contain fibrin and inflammatory cells such as eosinophils, macrophages and plasma cells[3].

Factors that provoke an asthmatic attack include irritants like smoke, perfumes, cold weather; pollens in the air, dust, etc[2]. Cold weather, exercise and dust were found to be the precipitating factors in this patient. Exposure to cooking gas has also been found to provoke attacks. This is because cooking gas contains butane and nitrogen dioxide [4].

Patients with status asthmaticus can present with increasing respiratory distress during acute asthmatic attack unresponsive to usual treatment[1]. Severe respiratory distress in status asthmaticus is usually marked by hyperinflation, wheezing with absent breath sounds, fatigue, and cyanosis and pulsus paradoxus [5]. Most of these symptoms and signs were present in this patient. In status asthmaticus, as in this patient there is intractable bronchospasm and respiratory rate is greater than 25/min, heart rate greater than 110/min and peak respiratory flow rate less than 50/% of predicted normal [5] Severe life threatening attack of asthma is when the patient having sign of wheeze is unable to complete a sentence due to breathlessness and if heart rate is more than 100 beats/min [6] as seen this patient.

Laboratory studies should not delay the immediate treatment of patients with acute asthma. Arterial blood gas measurements are important for patients with severe respiratory distress or impending respiratory failure. There were no facilities for measuring end tidal carbondioxide and Arterial Blood Gases (ABG) in our centre therefore; blood was taken for urea/electrolyte and FBC.

Hypoxia caused by ventilation/perfusion inequality may be present in a severe asthma exacerbation [7]. Supplemental oxygen and hospital admission should be strongly considered for this acidosis with hypercarbia. Arterial Blood Gases (ABG) was not done in this patient due to lack of facilities in our centre. ABGs may also be used to stage asthma.

It was documented [3] that all severe ill asthmatic patients with respiratory failure require intubation and mechanical ventilation as in this case. This patient was severely ill with respiratory failure because respiratory rate of the patient decreased from 43/min (respiratory fatigue), so she was admitted into the Intensive Care Unit (ICU) for ventilator support. Respiratory failures was found to be the second major medical reason for admission into the Intensive Care Unit of Jos University Teaching Hospital between 1994 -2002 and formed about 1.4% of the total ICU admissions within that period [7].

Indications for early tracheal intubation include apnoea or near apnoea, central cyanosis, mental status changes, or depressed level of consciousness. Inability to adequately oxygenate or ventilate an asthmatic patient mandates tracheal intubation [8] This patient presented with central cyanosis and depressed level of consciousness hence she was intubated. In intubating asthmatics the choices of induction agents include benzodiazepines, narcotics, ketamine and propofol. Ketamine offers an advantage due to its bronchodilating effect as well as its minimal respiratory depression potential [9] neuromuscular blockade may also be required in some patients to facilitate intubation.

Once an asthmatic patient is intubated successfully and mechanical ventilation has been instituted, a combination of benzodiazepine available and opioids offer optimal sedation [8]. Diazepam which was the only benzodiazepine available was part of the sedation used in this patient. Ketamine was also used for 5 hours because ketamine is an effective bronchodilator and can be used for the patient who is not responding to conventional bronchodilators such as salbutamol and aminophylline [10]. The doses of ketamine required are very low and problems with hallucinations are rare.

CONCLUSION

The intensive care management of a severe asthmatic patient though challenging but with proactive intervention and dedication the outcome can be good even in the developing hospitals with minimum investigative and interventional facilities. Careful planning and medical follow up results in effective control.

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