International Journal of Biomedical and Health Sciences
Vol. 5, No. 3, September 30, 2009
Printed in Nigeria

0794-4748/2009 $12.00 + 0.00

http://www.asopah.org

IJBHS 2009061/5303

Seizure disorder complicating acute severe Asthma: An unusual presentation

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(Received June 29, 2009)

ABSTRACT: Asthmatic attack is one of the commonest disorders presenting in the medical outpatient clinic in Nigeria. There are attendant complications in untreated acute severe exacerbations of asthma. In this article, we report seizure disorder complicating an acute severe exacerbation of asthma affecting a young Nigerian student. There is widespread bronchoconstriction in acute severe asthma leading to hypoventilation in the phase of normal perfusion. The Hypoxemia that ensues causes a sequence of respiratory alkalosis, respiratory acidosis and metabolic acidosis which makes the patient more susceptible to anoxic brain damage. The seizure in this circumstance was easily reversed by treatment with adequate Oxygen therapy in addition to the nebulised salbutamol and steroid. The seizure could be mistaken to be a primarily neurological condition coexisting with asthma and hence referred to a neurologist.

Key words: Asthmatic attack, seizure disorder, bronchoconstriction, hypoventilation, Hypoxemia and metabolic acidosis.

Introduction

Asthma is a common disease and its frequency sometimes detracts from its potential seriousness. Severe asthma in children is the third most common cause of hospital admission and the most common cause of paediatric ICU admission[1, 2]. In adult asthmatics, only 5-10% have severe disease but these individuals carry a substantial proportion of the cost (both in terms of morbidity and economic) and run the highest risk of acute severe exacerbations and death[3].

Acute severe asthma is a common respiratory emergency, occurring on the background of brittle asthma and or as a result of exacerbation of asthma in the presence of triggering factors like viral infection, allergen exposure etc[4]. Status asthmaticus is severe asthma that does not respond well to immediate care and is a life-threatening medical emergency.

Ensuing respiratory failure results in hypoxia, carbon dioxide retention and acidosis. The exact mechanism underlying the development of an acute severe asthma attacks remains elusive but there appear to be two phenotypes[5, 6].

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Gradual onset - in about 80%, severe attacks develop over more than 48 hours. These are associated with eosinophilic infiltration and slow response to therapy.

Sudden onset - often in association with significant allergen exposure. Patients tend to be older and to present between midnight and 8 am. These types of attacks are associated with neutrophilic inflammation and a swifter response to therapy.

Common complications of acute severe asthma include pneumothorax, subcutaneous emphysema, pneumomediastinum and respiratory arrest. Other complications are hypokalemia, hypomagnesemia, respiratory alkalosis, respiratory acidosis, metabolic acidosis, hypophosphatemia, myocardia ischaemia and infarction in the elderly with coronary heart disease[7, 8]. However, seizure disorder is a rare complication and a risk factor of acute severe asthma[9,10,11] The criteria for diagnoses of severe acute asthma includes a past history of asthma, presenting with recurrent attacks of asthma which does not respond to its usual asthma therapy. Examination of the patient with asthmatic attacks would reveal severe central cyanosis, inability to complete a sentence in one breath and widespread rhonchi. The pulse rate is usually over 120/min, respiratory rate is ≥25 breaths/minute, Peak expiratory flow rate of ≤50% of predicted normal or personal best and there may be pulsus paradoxus. The life threatening features of acute severe asthma include silent chest, cyanosis, feeble respiratory effort, bradycardia, hypotension, exhaustion, confusion, coma, severe hypoxaemia (Po_2 <8kpa (60mmHg) and normal (5-6kpa (38-60mmHg) or high Pco_2. The wall of the airway in asthma is thickened by oedema, cellular infiltration, increased smooth muscle mass and glands. Mucus plugging of the airway in asthma is a prominent feature of acute severe asthma[9]. Increased bronchoconstriction and hypersecretion of the goblet cells in the airway leads to reduced ventilation in the phase of normal perfusion. As a result, there is Hypoxaemia which causes cerebral hypoxia.

The pathogenesis of seizure disorder occurring on the background of acute severe asthma has been ascribed to severe cortical hypoxia, cerebra anoxia and electrolyte imbalance. The few asthma patients who presented with seizure disorders may not be known epileptics and have no concomitant medical condition that may cause seizure disorder. Treatment of acute severe asthma with adequate Oxygen therapy in addition to nebulised bronchodilators and systemic steroids abolishes both acute severe asthma and consequently, the seizure disorder.

Case Report

A 22year old female Nigerian student who was diagnosed to have asthma in 1997, presented with the cardinal symptoms of Cough, Wheeze, Chest tightness and breathlessness at the Accident and emergency unit of University of Benin Teaching Hospital, (U.B.T.H) Benin. She was noticed to have a generalized tonic –clonic seizures that lasted for 30seconds in the emergency room. She was given 100% Oxygen by face mask, nebulised salbutamol and intravenous hydrocortisone. She improved clinically few minutes later and was placed on tablets prednisolone and salbutamol. Past medical history revealed a known asthmatic that was not compliant with her therapy. There was a strong family history of asthma. Examination showed a young lady who was in obvious respiratory distress and cyanosed. There were features of chest hyperinflation. However, patient was conscious and oriented in time, place and person. Laboratory investigations revealed a Forced Expiratory Volume in one second (fev_1) of 45% and Peak Expiratory Flow Rate (PEFR) of 40%. Oxygen saturation prior to treatment was 80%. Full Blood Count showed eosinophilia. Electrolyte, Urea and creatinine, Random blood sugar, skull X-ray were essentially normal. She was discharged home on seretide accuhaler and salbutamol inhaler. In addition, the cautious avoidance of known triggering factors has also contributed to the maintenance of her asthma control.

Discussion

Seizure disorder occurring following severe asthmatic attack is a rare complication of asthma which normally presents in the emergency room. It has been ascribed to severe cortical hypoxia, cerebral anoxia and electrolyte inbalance[12]. Our patient's Po_2 of 80% showed that there was a degree of hypoxia causing cerebral anoxia. However, there was no electrolyte derangement. Cerebral anoxia occurred as a result of the widespread peripheral airway occlusion that led to reduction of ventilation in the phase of normal perfusion[13]. The FEV_1 of 45% and PEFR of 40% revealed that there was peripheral airway obstruction which led to reduced ventilation that caused the hypoxia. Hypoxemia that ensues caused a sequence of respiratory alkalosis, respiratory acidosis and metabolic acidosis which made the patient more susceptible to anoxic brain damage[14].

Hypoxia may also occur from cardiac arrest complicating a Pre-existing myocardiac infaction[15]. However, there was no concomitant debilitating disorder in this patient. Other possible causes of seizure disorders may have been uncompensated respiratory alkalosis, metabolic acidosis, hypokalemia, hypomagnesaemia which occurs as a result of excessive use of Beta-2 agonist[12].The patient in question has not been very compliant with therapy and there was no history of overdose of her drugs. Hypoglycaemia which may occur in acute severe

asthma may present with seizure disorder. However, the patient's random blood sugar was normal. Hence, electrolyte, urea and creatinine analysis and blood sugar levels of patients with severe asthmatic attack should routinely be done. Spirometry, Peak Expiratory Flow Rate and Pulse oximetry are necessary in assessing the level of airway obstruction and Oxygen saturation. Furthermore, prompt treatment of the asthmatic attack with adequate Oxygen, nebulised bronchodilators and systemic steroids reversed the airway obstruction and the seizures subsided. Correction of any electrolytes and blood sugar levels will also help tremendously.

Conclusion and Recommendation

In conclusion, Seizure disorder as in this case can be mistaken to be primarily a neurological condition coexisting with asthma. Such patients could be referred to a neurologist. In addition, a detailed knowledge of the pathophysiology of asthma and its consequences could avert such diagnostic pitfalls. Physicians should also have high index of suspicion of seizure disorder complicating severe acute asthma. Further surveillance is therefore advanced in order to discover those patients whose seizures are due to electrolyte imbalance and blood sugar abnormality.

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