

## LIFE THREATENING PNEUMOMEDIASTINUM COMPLICATING CHILDHOOD ASTHMA

Dr. M. S. M. Nawaaz

### SUMMARY

A nine year old asthmatic, presented with marked dyspnoea and subcutaneous emphysema (SCE). A diagnosis of acute exacerbation of asthma complicated by pneumomediastinum (PM) and pneumopericardium (PPC) was made. Management was progressively escalated from conventional asthma treatment to insertion of bilateral intercostal tubes, mechanical ventilation (in the head up position), invasive monitoring and finally an incision at the suprasternal notch. However, the pressure effects of PM and PPC resulted in severe hypotension, hypoxia and metabolic acidosis. A sternotomy was then planned. The position of the patient was changed from head up to supine, for transfer to the operation theatre and resulted in a dramatic improvement of blood pressure and oxygenation.

However, the severe protracted hypotension and hypoxia that prevailed earlier resulted in multiorgan failure, and she needed multi disciplinary care in the ICU for 32 days before she recovered.

**Keywords :** *Pneumomediastinum, Childhood asthma.*

### Introduction

PM is a rare occurrence in paediatric practice and a literature search shows only retrospective individual case reports and small series of cases. The aetiology is multifactorial<sup>1</sup> and usually secondary to alveolar rupture in to the pulmonary interstitium, followed by dissection of gas towards the hilum and mediastinum.

Many pathological and physiological events can lead to alveolar rupture,<sup>2</sup> but the commonest cause in children is asthma.

### Case report

A 9 year old girl with a history of bronchial asthma was admitted to the casualty medical ward at Lady Ridgeway Hospital for Children with severe dyspnoea, tachypnoea (52 breaths /min) and SCE around the neck and chest. She had a cough and difficulty in breathing and had been on treatment with salbutamol and amoxicillin for 2 weeks. The night prior to admission, she had an episode of violent coughing and became acutely short of breath, which got worse with the development of SCE.

A diagnosis of acute exacerbation of bronchial asthma was made and treatment started with salbutamol and ipratropium nebulisation, aminophylline infusion and prednisolone. In spite of therapy, the child became increasingly dyspnoeic with further spread of SCE involving the hands and abdominal area. She became cyanosed and

could not speak. Arterial blood gases (ABG) showed PaO<sub>2</sub> mmHg of 86 mmHg, SaO<sub>2</sub> of 82% with a respiratory alkalosis (pH=7.51). She was admitted to the Cardiothoracic Intensive Care Unit (ICU), about six hours after admission to hospital.

In the ICU, a chest X-ray revealed a bilateral pneumothorax and a narrow rim of PPC. Bilateral IC tubes were inserted and PPC was left alone as it did not appear to be clinically significant. Following the insertion of IC tubes, the SCE reduced substantially. However, the patient went on to status asthmaticus with a silent chest and SpO<sub>2</sub> reduced to 80%.

She was intubated and ventilated, in the head up position. Magnesium sulphate, ketamine and adrenaline infusions were added. Invasive blood pressure monitoring was commenced and central venous access obtained through the femoral vein due to SCE around the neck.

Three hours later, SCE increased spreading all over the body and the blood pressure reduced to 60/28 mmHg with a tachycardia of 180-190/min. Fluid boluses and inotropes (infusions of adrenaline 1 mg/kg/min and dopamine 8 mg/kg/min) gave no improvement.

An urgent echocardiogram was done but no proper ECHO windows of the heart could be visualised in the precordial area due to air in the mediastinum. An ECHO window was just detected through a subcostal view, which showed a compressed heart with global wall hypokinesia with an ejection fraction <30%. An ECHO guided pericardiocentesis was done and about 20 ml of air was aspirated, but with no immediate improvement.

M.D., FCARCS (Ireland).  
Consultant Cardiac Anaesthetist.  
Lady Ridgeway Hospital for Children.  
Colombo, Sri Lanka  
E-mail : nawaz\_m@hotmail.com

To relieve the air in the thoracic cavity, a 4 cm transverse surgical incision was made at the suprasternal notch, under local anaesthesia, but still no improvement occurred. The BP was 52/22 mmHg, heart rate 210/min and ABG showed pH 7.19, PaO<sub>2</sub> 78 mmHg, PaCO<sub>2</sub> 29 mmHg, SaO<sub>2</sub> 82%, and BE-8.

A midline sternotomy was planned and informed consent was obtained. The patient was transferred to a trolley and positioned supine with a pillow under the head, to be transferred to the adjoining cardiac theatre. An improvement of blood pressure and heart rate was immediately noticed. On reaching the operating table, the vital signs were greatly improved. (BP 100/60 mmHg, heart rate 140/min, PaO<sub>2</sub> 133 mmHg, SpO<sub>2</sub> 100%, pH 7.22 and BE -5.7). A 2D-ECHO repeated on the operation table revealed effective cardiac contractions with ejection fraction >60% through a precordial view which was not obtained earlier. Sternotomy was therefore abandoned. The landmarks of the neck became distinct enough to insert a CVP line to the internal jugular vein, which read a CVP of 12 mmHg, denoting an adequate filling pressure

The patient was transferred back to the ICU. The mechanical ventilation with sedation was continued with adrenaline and dopamine infusions, antibiotics (Co-Amoxycylav), and treatment for bronchial asthma.

On the 2<sup>nd</sup> day, there was acute renal failure with a blood urea of 21mmol/l and serum creatinine of 334mmol/l, anuria for 18hours with generalized swelling of the body. Although pH remained at 7.41 and K<sup>+</sup> was 4.7 mmol/l peritoneal dialysis (PD) was commenced. The Nephrologist was consulted for his advice. Nutrition was given through a nasogastric tube at 750-1250 kcal/day.

The same day, she also developed a tonic clonic seizure. A CT scan of brain was not done as she was too ill to be transported to the radiology department. Meticulous control of fluid balance, electrolytes, blood glucose, temperature and normocarbia were maintained. Phenobarbitone was added to control the seizures.

Hypoxic liver injury was also present with evidence of increased Alanine Transaminase (ALT) and Aspartate Transaminase (AST) values up to 12300u/l and 3400 u/l respectively. A bolus of N-Acetyl Cystine (NAC) 150 mg/kg and an infusion of 100 mg/kg/ 24 hours and metronidazole were started, and micro-enemas given. The gastroenterologist was consulted for his advice. After 48 hours of NAC treatment both AST and ALT reduced to

<1000 u/l and liver functions improved slowly over the 2 weeks.

Ventilator associated pneumonia (VAP) with large quantities of tracheobronchial secretions was another major complication.

At the end of first week, although the cardio respiratory parameters were maintained without any inotropic support and a FiO<sub>2</sub> of 0.4, ventilatory support was needed to minimize the respiratory embarrassment secondary to abdominal distension as a result of the PD. On the 8<sup>th</sup> day, a tracheotomy was performed.

On the 10<sup>th</sup> day, cultures confirmed the presence of an infection in the PD and haemodialysis was commenced. She was weaned off the ventilator on day 18 but tracheostomy was left insitu since the cough reflex was weak. After 10 haemodialysis sessions, over a period of 21days, renal function returned to normal.

On the 32<sup>nd</sup> day she was transferred to the ward with a fenestrated tracheostomy tube insitu. On the 45<sup>th</sup> day the tracheostomy was removed. She was fully conscious and rational and had no memory of her ICU stay

## Discussion

PM rarely leads to clinically significant complications. Although rare, tension PM has been reported in which elevated mediastinal pressure leads to diminished cardiac output because of direct cardiac compression or reduced venous return. When extensive subcutaneous and mediastinal gas is present, airway compression also occurs. These problems were encountered in this case report.

A PM should be ruled out in all patients with subcutaneous emphysema around the neck and thoracic area. Subcutaneous air, the presence of subcutaneous crepitations, although not pathognomic of PM, suggests free air is present within the thoracic cavity. Stack<sup>3</sup> reported finding subcutaneous emphysema in 73% of patients presenting with asthma subsequently found to have PM. The positive predictive value of this sign for PM in their series was 100%. Stack et al<sup>3</sup> also reported, a 0.3% incidence of PM in association with asthma presenting to their institution over a 10-year period. The mean age of affected patients was 11 years. No sex differences were observed in their cohort.

The dissection of free air may not be confined solely to the mediastinum. Zylak<sup>4</sup> et al note that the mediastinum communicates with the submandibular space, the retropharyngeal space, and vascular sheaths within the

neck. As a result, air may dissect through these tissue planes, causing PPC, pneumothorax, SCE, pneumoperitoneum, or pneumoretroperitoneum.

Patients on mechanical ventilation are often positioned 45° head up to reduce the risk of VAP<sup>5,6</sup>. This is the guideline and practice in our ICU. The head up position also facilitates respiration by reducing diaphragmatic splinting secondary to abdominal distension.

Patients with severe asthma also prefer to maintain a seated, tripod posture to facilitate their expiration. The recommendation<sup>7</sup> for mechanical ventilation of sepsis-induced acute lung injury / ARDS, patients is also 45° head up. In head injury, head up position of 30° to facilitate venous drainage of the brain.

The dramatic improvement seen in our patient on changing from head up to supine shows that when there is PM the position of the patient is critical. The likely explanation is that air tracks upwards in the thoracic cavity in the head up position and compresses the major vessels and heart obstructing venous return cardiac output. We suggest keeping the patient supine to prevent air tracking and compressing the major vessels at thoracic inlet impeding venous return to the heart and pressure on the heart reducing cardiac output. A major catastrophe could have been averted had we placed our patient in the supine position throughout. We found no guidelines or recommendations in the literature regarding posture in the management of PM.

Conclusion : When a patient with a history of asthma develops SCE, there should be a high index of suspicion of

PM and PPC and treatment initiated without delay. When a patient with PM and PPC needs to be ventilated, the supine position is recommended to prevent air from compressing the large vessels and heart.

## References

1. *Damore DT, Dayan PS* : Medical causes of pneumomediastinum in children. *Clin Pediatr (Phila)* 2001 Feb; 40(2): 87-91.
2. *Weg JG, Anzueto A, Balk RA et al*: The relation of pneumothorax and other air leaks to mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998 Feb 5; 338(6): 341-46.
3. *Stack AM, Caputo GL*: Pneumomediastinum in childhood asthma. *Pediatr Emerg Care* 1996 Apr; 12(2): 98-101
4. *Zylak CM, Standen JR, Barnes GR, Zylak CJ*: Pneumomediastinum revisited. case report *Radiographics* 2000 Jul-Aug; 20(4): 1043-45.
5. *Drakulovic MB, Torres A, Bauer TT, Nicolas JM, Nogue S, Ferrer M*. Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients; a randomized trial. *Lancet* 1999; 354: 1851-58.
6. *Al-Haddad M, McLeod M, McLeod S*. Can we implement the 45 degree head up position for ventilated patients by educating nursing and medical staff? *Br J Anaesth* 2004; 92: 612P.
7. Surviving sepsis campaign guidelines for management of severe sepsis and septic shock. Copyright 2004, by the society of Critical care medicine. [www.sepsisforum.gov](http://www.sepsisforum.gov)