SHORT REPORT

Spontaneous pneumopericardium, pneumomediastinum and subcutaneous emphysema: unusual complications of asthma in a 2-year-old boy

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A 2-year-old boy presented to the emergency department with a history of sudden onset of cough, dyspnoea and a slight expiratory wheeze on the right lung base. He also had subcutaneous emphysema on the left side of the chest anteriorly. Chest *x* ray confirmed subcutaneous emphysema and also revealed pneumomediastinum and pneumopericardium. He had had no previous episode and was not known to have asthma. He was apyrexial but had a raised white cell count. The eosinophil count was within normal limits. He was successfully treated with nebulised salbutamol, steroids, antibiotics and high flow oxygen. He made a good recovery and was discharged after 7 days. This case highlights the need for a high index of suspicion of asthma in very young children presenting for the first time with such complications.

A 2-year-old boy was admitted to our emergency department with a history of sudden onset of dyspnoea and cough, which started a few hours before presentation. He had had no previous episode and was not known to have asthma. He was fit and well.

Physical examination revealed the child had severe dyspnoea and cyanosed lips. He had marked flaring of the alae nasae and subcostal and intercostal recession. He was also found to have subcutaneous emphysema on the left side of the chest and reduced air entry on the left side with mild expiratory wheeze. His respiratory rate was 48 per min and he had an oxygen saturation of 94% on room air.

His heart rate was 153 beats per min. Heart sounds were muffled but no murmur was heard. Chest *x* ray confirmed subcutaneous emphysema and also revealed pneumomediastinum and pneumopericardium. Laboratory investigations showed a raised white cell count of 26 000 mm^3. The eosinophil count was normal. The electrolytes and urea were also normal. The capillary blood gas showed a pH of 7.36 PCO_2 of 5.99 kPa and a PO_2 of 11.8 kPa.

The patient had an electrocardiogram (ECG) and an echocardiogram which revealed no abnormality. Further investigations undertaken included complement fixation tests for mycoplasma pneumonia, influenza A virus and B virus, respiratory syncythial virus , chlamydia, Q fever and adenoviruses. The titres were non-diagnostic. Pulmonary function tests (peak flow and spirometry) could not be done because of the age of the patient.

The patient was successfully treated with regular nebulised salbutamol, steroids, antibiotics and high flow oxygen. The radiological signs of pneumopericardium and pneumomediastinum completely resolved and subcutaneous emphysema significantly improved following treatment. He was discharged after 7 days.

DISCUSSION

Spontaneous pneumopericardium, pneumomediastinum and subcutaneous emphysema are rare in children. The most common cause is asthma and the incidence has been reported to be 0.3% following an acute attack.[1]

The clinical diagnosis is based on the symptom triad of dyspnoea, chest pain and subcutaneous emphysema. It is also based on the Hamman's sign, which consists of a crunching, rasping sound synchronous with the heart beat and best heard over the precordium. Other entities such as bronchiolitis caused by viral infections and airway irritants must be considered.[2 3] Oesophageal rupture is also another important differential diagnosis which can be confirmed by contrast oesophagogram.

The mechanism for this is alveolar rupture with air leak into the interstitial tissues. This then tracks through the mediastinum, neck and into the subcutaneous space. This rupture is due to the existence of a pressure gradient between the alveolus and the surrounding tissues. The pressure within adjacent alveoli is generally assumed to be equal so interalveolar walls should remain intact. Overinflation and increased alveolar pressure commonly occurs with obstructed expiratory flow leading to the creation of a pressure gradient. A pressure gradient favouring alveolar rupture and dissection of air may occur whenever alveolar pressure rises or interstitial pressure is reduced. Other causes of a raised intra-alveolar pressure apart from the aforementioned include violent cough, Valsalva manoeuvre, emesis and barotrauma in patients receiving mechanical ventilation.

Alveolar rupture caused by a reduction in lung interstitial pressure may result either from extreme respiratory effort as in pulmonary function testing, vigorous exercise, diabetic acidosis and marijuana smoking or from a rapid reduction in atmospheric pressure as in air travel, mountain climbing and diver's decompression sickness.[4]

The diagnosis of pneumopericardium and pneumomediastinum is confirmed by chest radiography. Pulmonary function tests (peak expiratory flow rate and spirometry) are indicated after the acute episode to determine whether the patient has asthma. However, this is usually only possible from about the age of 4–6 years.

Other investigations such as skin tests (for allergy) can also be done. In a similar case described by Kucukosmanoglu *et al* in a 4-year-old girl, the only positive finding was a skin reaction to *Dermatophagoides pteronyssinus* and *D farinae,* mould, tree and grass mix. This patient also responded well to steroid and salbutamol inhalers.[5]

Further investigations such as total IgE, radioallergosorbent tests for specific IgE antibodies and the immunoglobulin subclasses can also be undertaken. The functional residual

Abbreviation: ECG, electrocardiogram

capacity and thoracic gas volume can be done where facilities are available.[6]

In most cases of mediastinal and soft tissue emphysema conservative management is indicated as spontaneous resolution occurs.[4 7] Management consists of treating the underlying cause (if identified), rest, analgesia and simple clinical monitoring. Where no cause is identified it is reasonable to treat the patient empirically with bronchodilators, steroids and oxygen, as was done for our patient.[8]

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