

Extensive Pneumomediastinum With Subcutaneous Emphysema In A Case Of Olivopontocerebellar Degeneration With Bronchial Asthma

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Citation

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Abstract

A 35-year old female known to have Olivopontocerebellar degeneration and bronchial asthma was admitted with a severe episode of breathlessness, wheezing and cough along with retrosternal pain. Clinical examination revealed features consistent with severe asthma and subcutaneous emphysema of cervical and upper thoracic areas. Radiographic imaging revealed extensive subcutaneous emphysema, pneumomediastinum and pneumopericardium. Treatment of the asthma resulted in rapid recovery of the bronchospasm and resolution of the pneumomediastinum over a period of 2 weeks. Development of chest pain or sudden worsening of asthma in a patient should prompt a search for the appearance of pneumomediastinum.

INTRODUCTION

Pneumomediastinum, presence of air or other gas in the mediastinum, is an infrequently encountered clinical condition that can result from a variety of causes. Pneumomediastinum most commonly results from microscopic alveolar rupture but can also result from air escaping from the upper respiratory tract, intrathoracic airways, or gastrointestinal tract. Gas can be generated by bacteria causing infection of the visceral space, and outside air can reach the mediastinum after trauma or surgery (1). Pneumomediastinum occurs in approximately 1 per 10,000 hospital admissions but may in fact be an under-diagnosed condition (2). Spontaneous pneumomediastinum generally occurs in young, healthy patients without serious underlying pulmonary disease. Pneumomediastinum is a rare complication of asthma especially in young men. Although pneumomediastinum, subcutaneous emphysema, and pneumothorax are uncommon complications of acute bronchial asthma, they are quite alarming and physicians unfamiliar with their management can panic and seek surgical management which in reality is very rarely required. We report the case of a middle aged female with atopic asthma who presented with pneumomediastinum which resolved on conservative management.

CASE REPORT

A 35-year-old female, known asthmatic presented with a 2-day history of breathlessness, cough, wheezing and scanty mucoid expectoration.. Few hours after the onset of her symptoms she developed retrosternal chest pain that was aggravated by movement, deep inspiration and coughing which was accompanied by worsening of her breathlessness. There was no preceding history of trauma or recreational drug use. Her family denied a history of asthma. She had a history of multiple exacerbations of her asthma in the past and five years earlier was diagnosed to have olivopontocerebellar degeneration for symptoms of gait ataxia. Physical examination revealed a thin built young dyspnoeic, cyanosed female with RR of 32/min, pulse of 100/min and a blood pressure of 110/70 mm Hg. Accessory muscles of respiration were active and upon auscultation of the chest polyphonic rhonchi with expiratory wheeze were audible. There was clinical evidence of subcutaneous emphysema in the neck and the upper thoracic area with swelling, crunchy feel, palpable crepitus and noisy crackling sounds on auscultation. Central nervous system evaluation revealed bilateral cerebellar signs with ataxia, pyramidal tract signs and abnormal eye movements. Rest of the general physical and systemic examination was normal. Investigations revealed hemoglobin of 13.4 g/dL, TLC of 7,000/mm³ with a differential count of 65 %, polymorphs and 28% lymphocytes, 5% eosinophils, normal platelet count with a normal peripheral smear. The serum levels of

urea, creatinine, glucose, cholesterol, bilirubin, alanine and aspartate aminotransferases, alkaline phosphatase, proteins, albumin, cholesterol, triglycerides, lipid subfractions, sodium, potassium and chloride were normal. Arterial blood gas analysis revealed a pH of 7.38, PaO₂ of 61 mmHg, PaCO₂ of 29 mmHg, bicarbonate of 23 mmol/l and SaO₂ of 94 %. She was not able to blow into a peak expiratory flow meter. Blood culture, Gram's smear of the sputum and cultures were negative. A radiograph of the chest revealed hyperinflated lung fields with evidence of subcutaneous emphysema with air tracking

along fascial planes in the neck and pneumomediastinum (Fig 1).

{image:1}

CT of the neck and the chest (Figs 2-3) revealed subcutaneous emphysema, pneumomediastinum and pneumopericardium.

{image:2}

{image:3}

An ECG showed sinus tachycardia. She was treated conservatively with oxygen therapy, nebulised bronchodilators, steroids and analgesics. Her bronchospasm responded and the pneumomediastinum and subcutaneous emphysema resolved over a period of 2 weeks. Subsequent spirometry was consistent with a bronchodilator responsive obstructive type of airflow limitation. On a discharge prescription of budesonide/formoterol, her symptoms of asthma have been controlled over a followup of 2 months.

DISCUSSION

Our patient had pneumomediastinum, subcutaneous emphysema and pneumopericardium associated with an attack of bronchial asthma. The patient's basic disease of OPCA was seemingly unrelated to his current problem. Although upper airway dysfunction has been reported (3), there is no reported association with bronchial asthma OPCA.

Subsequent to the initial description of mediastinal emphysema by Laennec in 1819 (4), myriad etiologies have been reported for its occurrence. In the current times of invasive diagnostic and therapeutic measures, mediastinal emphysema is encountered in a greater variety of clinical circumstances including mechanical ventilation and other

aspects of critical care, decompression in diving and other settings, chest trauma, asthma, and numerous other situations in addition to childbirth (5). Pneumomediastinum most commonly results from microscopic alveolar rupture but can also result from air escaping from the upper respiratory tract, intrathoracic airways, or gastrointestinal tract. Gas can also be generated by bacteria causing infection of the visceral space, and outside air can reach the mediastinum after trauma or surgery (1). Mediastinal emphysema has however, only rarely been reported in bronchial asthma. In a study of 479 radiographs of children with bronchial asthma, pneumomediastinum was demonstrable in 5.4% cases (6).

The disruption of alveolar walls and entry of air into the bronchovascular sheath occur when a sudden increase in the alveolar pressure or a fall in the perivascular interstitial pressure results in a pressure gradient enough to disrupt the alveolar walls at the bases and introduce air into the pulmonary interstitium (7-10). Although it has been speculated that a sufficient increase in intrapulmonic pressure was capable of producing alveolar rupture (10), animal experiments have demonstrated that an increase in alveolar volume, due to increased transpulmonary pressure, is a more important determinant of disruption of the alveolar walls (11). This finding may explain the rarity of alveolar rupture following coughing and sneezing, acts that buttress the chest and abdominal walls to counter the transient high intrapulmonic pressures (12). Entry of air into the bronchovascular sheath results in pulmonary interstitial emphysema. Since the mean pressure in the mediastinum is always somewhat negative to the pulmonary parenchyma, the pressure difference causes the air to move centripetally, the movement facilitated by the pumping action by the normal act of breathing (13, 14). Once in the mediastinum, air may follow the path of least resistance and may rupture through the mediastinal fascia and overlying pleura into the pleural space resulting in pneumothorax. Extraalveolar air might also find its way through the loosely packed paraesophageal connective tissue into the peritoneal cavity resulting in pneumoperitoneum or pneumoretroperitoneum (15, 16). Air has also been reported to enter the pulmonary vasculature resulting in arterial embolism (17,18). Compression of the great veins within the mediastinum and subsequent decrease in cardiac output may occur if large quantities of air accumulate in the mediastinum under tension.

The diagnosis of pneumomediastinum should be suspected when an asthma patient experiences substernal chest pain during an acute asthmatic attack. Pain arises from the stretching of the mediastinal tissues (6,19), and is often aggravated by movement and position change (19,20). Discomfort may extend to the neck and dysphonia or dysphagia might result from retropharyngeal or perilaryngeal air dissection (19, 21). Subcutaneous emphysema in the neck and in supraclavicular areas is evident and a “crunching” precordial sound synchronous with the heartbeat (Hamman’s crunch) is the most characteristic finding (6,19). In pneumopericardium a metallic splashing sound called as the “mill-wheel murmur” is heard that is generated due to the presence of air along with fluid in the pericardium (22,23). None of the two characteristic findings were observed in our patient.

Pneumomediastinum can be confirmed with the help of the chest radiograph, however, the sensitivity is low and a mild pneumomediastinum may be missed. A postero-anterior film may show a thin line of radiolucency along the heart borders, best

seen the left side. The other radiographic signs include highlighting of the aortic knob

by surrounding increased lucency; the “continuous diaphragm sign”- an unbroken radiolucent line extending from one hemidiaphragm to other beneath the heart; and longitudinal gas shadow adjacent to the thoracic aorta and around the pulmonary artery termed the “ring around the artery sign”. Lateral view may show retrosternal air or vertical lucent streaks outlining the aorta and other mediastinal structures.(6,24,25). Radiographs performed at end-expiration may accentuate the extra-alveolar air (25).

Subcutaneous emphysema may be evident in the initial chest radiographs in most patients. CT thorax is very useful in diagnosing pneumomediastinum since it has very high sensitivity and specificity and diagnostic in mild pneumomediastinum especially when the clinical presentation is atypical. In most cases no therapy other than careful treatment of the acute asthmatic attack is required. (26, 27). Extension of air into the subcutaneous tissues tends to decompress the mediastinum and relieve any potential mediastinal circulatory disturbances. However, should marked circulatory and respiratory distress occur, the patient should be promptly placed in 100 percent oxygen atmosphere (28). This increases the diffusion pressure of

nitrogen in the subcutaneous tissues, permitting an increased rate of reabsorption and relieves any other conditions, pressure symptoms caused by ambient air. If this fails to relieve the tension pneumomediastinum, cervical mediastinotomy should be performed by making incisions in the supraclavicular fossae posterior to the sternocleidomastoid muscles and in the suprasternal notch (29-32). Tracheostomy under these circumstances can be helpful since it not only decompresses the mediastinum but also decreases the intra-alveolar pressure and prevents further escape of air into the mediastinum.

Our report, in concert with similar recent reports (33,34), emphasizes the importance of considering a diagnosis of mediastinal emphysema in a patient of bronchial asthma who has a sudden intensification of his symptoms or development of chest pain.

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