Oesophagography and oesophagoscopy are not necessary in patients with spontaneous pneumomediastinum

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ABSTRACT

Background: Because the condition is rare, the proper assessment of spontaneous pneumomediastinum (SPM) remains controversial. The purpose of this study was to determine whether additional oesophageal investigations beyond chest *x* ray and chest computed tomography (CT) scan are necessary for the diagnosis of SPM.

Methods: The medical records of 25 patients diagnosed and treated for SPM from March 1986 to December 2007 were retrospectively reviewed.

Results: There were 22 men and 3 women, with a median age of 19 years (range 15–57 years). All patients received chest *x* rays, which revealed air shadows within the mediastinum or subcutaneous emphysema in 24 patients. Twenty-two patients underwent chest CT scans, which showed pneumomediastinum in all cases. Oesophagography was performed in 14 patients and oesophagoscopy in three. All oesophagographies and oesophagoscopies were clear. Despite conservative treatment, no patients developed mediastinitis or complications associated with oesophageal injury.

Conclusions: Chest *x* ray and CT scan are sufficient to diagnose SPM. Additional diagnostic assessments such as oesophagography and oesophagoscopy are not necessary in patients without evidence of mediastinitis or a history of oesophageal injury.

Pneumomediastinum, a condition in which free air is present in the mediastinum, is most commonly caused by trauma, mechanical ventilation or iatrogenic barotraumas. Pneumomediastinum without an obvious cause is referred to as a *spontaneous pneumomediastinum* (SPM). Because the condition is rare, the proper assessment of SPM is still controversial.[1–4] SPM can be confirmed by plain chest *x* ray showing a radiolucent outline around the heart and mediastinum. Chest computed tomography (CT) scan shows more precise information about the extent of the pneumomediastinum and the presence of mediastinitis. Mediastinitis is the most severe complication of pneumomediastinum, and the clinical course can be fatal, even with invasive treatment. The possible origins of mediastinitis combined with pneumomediastinum include an injured oesophagus, injured large airway, cervical infection or buccal infection. Accordingly, some doctors perform additional oesophagography, oesophagoscopy or bronchoscopy to rule out oesophageal or large airway injury. This study was conducted to determine whether such additional investigations beyond chest *x* ray and CT scan are necessary for the management of SPM.

METHODS

We retrospectively reviewed all medical records of patients diagnosed with pneumomediastinum and treated at the Severance Hospital, a tertiary referral centre of a large university hospital in Seoul, South Korea, between March 1986 and December 2007.

Four thoracic surgeons participated in diagnosis and treatment. Regardless of causes, all patients were admitted to the hospital for diagnosis and treatment. Patients with pneumomediastinum caused by trauma, pneumothorax, mechanical ventilation, barotrauma or lung parenchymal disease were excluded. Patients with an evident history of foreign body aspiration and those <15 years were also excluded.

SPM was confirmed using chest *x* ray or chest CT scan. Additional diagnostic tests such as oesophagography with barium or iodinated water-soluble contrast media, oesophagoscopy or bronchoscopy were performed in some patients.

Medical records were reviewed for history, presenting symptoms and signs, precipitating events, diagnostic tests, treatment modality, length of hospital stay, complications and prognosis by one thoracic surgeon. Standardised collection forms were used to guide data collection. The study's corresponding author supervised the data collection. Approval of this study was obtained by the institutional review board of our institution.

RESULTS

Of 167 patients identified with pneumomediastinum, 25 were eligible for this study (table 1). There were 22 men and 3 women, with a median age of 19 years (range 15–57 years). The mean body mass index was 21.4 (SD 3.7) kg/m^2), which was not different from the normal range. Two patients were smokers. Seven patients reported a history of bronchial asthma, and two patients had a history of pulmonary tuberculosis. One patient experienced an episode of spontaneous pneumothorax.

At the time of initial presentation, all patients complained of symptoms related to pneumomediastinum. The most frequent symptoms were chest pain and dyspnoea, which were each found in 15 patients. Eight patients complained of persistent cough. Other symptoms included neck pain (six patients), increased sputum (four patients), sore throat (three patients), foreign body sensation in the neck (five patients) and chilling sensations (two patients). Subcutaneous emphysema was observed on the upper body, particularly the neck, in 21 patients. Fever (body temperature >37.5°C) was present in three patients (table 2).

Table 1 Causes of pneumomediastinum

Cause	No. of patients (%)
Barotrauma of mechanical ventilator	34 (20.4)
Trauma	26 (15.7)
Underlying lung disease	26 (15.7)
Iatrogenic	16 (9.6)
Aspiration of foreign body	5 (3.0)
Oesophageal perforation	5 (3.0)
Colon perforation	3 (1.8)
Thoracic surgery	2 (1.2)
Tooth extraction	2 (1.2)
Tonsillar abscess drainage	1 (1.0)
Spontaneous	25 (15.0)

Six patients with incomplete medical records and 16 patients <15 years were excluded.

The initial mean white blood cell count was 12.9 (SD 5.2)$\times10^9$ per litre, and leucocytosis (white blood cell $>11.0\times10^9$ per litre, normal range in our hospital 4.0–11.0$\times10^9$ per litre) was noted in 15 patients.

The most common precipitating event of SPM was vigorous coughing induced by upper respiratory tract infection (n = 7). Other precipitating events included nausea and vomiting in three patients and physical sports (basketball and weight lifting) in two patients. SPM occurred after stretching and yawning in one patient. The remaining 11 patients reported no precipitating event.

Plain chest *x* ray was obtained for all patients, and an air shadow within the mediastinum or subcutaneous emphysema were found in all but one patient whose *x* ray displayed no specific abnormality. Chest CT scans were obtained for 22 patients that revealed pneumomediastinum in all cases. Oesophagography was performed in 14 patients, and 3 patients underwent oesophagoscopy. All oesophagographies and oesophagoscopies were clear. All patients were treated with oxygen treatment and prophylactic antibiotics. Nineteen patients fasted for a mean 2.2 (SD 0.8) days (range 1–4 days) during screening for oesophageal injury. Within the mean 2.8 (SD 0.9) days (range 2–5 days), symptoms subsided, and the air shadow in the mediastinum was almost absorbed, as seen on subsequent chest *x* ray. Mediastinitis or complications associated with oesophageal injury did not occur in any patient. The median hospital stay length was 4 days (range 2–41 days).

DISCUSSION

The important differential diagnosis in SPM is oesophageal perforation, which causes mediastinitis and requires prompt surgical intervention. The presentations of oesophageal perforation are vomiting, chest pain and subcutaneous emphysema, which is called the Mackler triad. In addition, oesophageal perforation may reveal fever, dyspnoea and dysphagia.[5] Because the symptoms are non-specific and similar to those of SPM, the diagnosis of oesophageal perforation is difficult and important. In this series, all oesophagography and oesophagoscopy results were normal, and all patients, including 15 patients with leucocytosis at presentation, recovered without complications associated with oesophageal injury. Accordingly, additional oesophageal evaluation such as oesophagography or oesophagoscopy might be not useful in SPM patients without a history suggestive of oesophageal injury or evidence of mediastinitis.

Table 2 Symptoms of SPM

Symptom	No. of patients	%
Subcutaneous emphysema	21	84.0
Chest pain	15	60.0
Dyspnoea	15	60.0
Persistent cough	8	32.0
Fever/chills	3	12.0

SPM was first described in 1939 by Hamman.[6] Macklin and Macklin[7] reported that SPM probably results from the rupture of terminal alveoli into the lung interstitium by a pressure gradient between the peripheral lung and hilum. Occurrence of SPM is known to be related to bronchial asthma,[8] severe cough or vomiting,[9] labour, the Valsalva manoeuvre,[10] diabetic ketoacidosis[11] and inhalation of illegal drugs.[12] In our patients, a history of bronchial asthma was found in seven cases and pulmonary tuberculosis in two patients. One patient had a history of spontaneous pneumothorax. Jougon and coworkers[9] reported that young, tall and thin men are at greatest risk for SPM. In our series, although SPM mainly occurred in young and male patients, the mean body mass index of the patients was within the normal range.

The symptom triad of SPM is chest pain, dyspnoea and subcutaneous emphysema.[9] All patients in our series presented with one or more symptoms related to pneumomediastinum. The most common symptoms were chest pain and dyspnoea. Subcutaneous emphysema was present in 84% of our patients.

Our diagnostic assessment consisted of chest *x* ray, chest CT scan and further evaluation such as oesophagography, oesophagoscopy or bronchoscopy, if needed. In our series, chest *x* ray revealed an air shadow within the mediastinum or subcutaneous emphysema in 24 (98%) patients. Twenty-two (88%) patients underwent a chest CT scan, which demonstrated pneumomediastinum in all cases. Oesophagography and oesophagoscopy were performed in 14 (56%) and 3 (12%) patients to exclude injury of the oesophagus. Oesophageal study was performed in all three patients who did not have a chest CT scan. Gerazounis *et al*[3] asserted that oesophageal studies with contrast material are essential in SPM to identify oesophageal disease. Koullias and colleagues[4] suggested that oesophageal tests should be performed in patients with fever, sweating or leucocytosis, which might represent a reasonable initial workup strategy for patients with SPM. In this series, however, oesophageal evaluation in patients without a history of oesophageal injury or evidence of mediastinitis revealed no pathology in the oesophagus. Furthermore, no patients developed delayed mediastinitis or complications related to oesophageal injury. Thorough history taking, physical examination, checks for objective signs of infection (such as fever and aggravation of symptoms) and serial measures of leucocytosis appear to be sufficient for the surveillance of oesophageal injury.

Chest *x* ray remains standard and important for the diagnosis of SPM. In contrast to previous reports,[13,14] chest radiography was diagnostic in 98% of our cases, although chest CT scan had a higher sensitivity. Chapdelaine *et al*[15] reported that the clinical benefit of a chest CT in benign conditions such as pneumomediastinum is questionable, considering the risk of radiation.

Treatments for SPM usually consist of hospitalisation, oxygen therapy, analgesics and prophylactic antibiotics. Moreover, in this series, 19 patients fasted until oesophageal perforation was excluded. Because oesophageal injury was not found by oesophagography or oesophagoscopy, fasting from the beginning of treatment was not necessary. In agreement with previous reports,[3,4,9] symptoms subsided within a mean 2.8 (SD 0.9) days (range 2–5 days), and the median length of hospital

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stay was 4 days (range 2–41 days). All patients were discharged without complications following conservative treatment.

CONCLUSION

SPM generally resolves spontaneously with conservative treatment. Chest *x* ray and chest CT scan are sufficient to diagnose SPM, and additional diagnostic assessments such as oesophagography and oesophagoscopy are not necessary in patients without an evident history of oesophageal injury or mediastinitis. Such patients can be identified by precise history taking, physical examination and laboratory tests.

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