Introduction

Pneumomediastinum (PM) is an uncommon event which defined as the presence of free air in the mediastinum. Its clinical picture includes retrosternal chest pain, subcutaneous emphysema, dyspnea, dysphagia and asthenia. PM is further divided into two groups of patients: spontaneous PM, without any obvious primary source, and secondary PM, with a specific responsible pathologic event, such as trauma, intrathoracic infections and violation of the aerodigestive track or others.

Spontaneous PM was originally described in 1939; thus, the crepitus heard with the heartbeat on chest auscultation is known as the Hamman sign [1]. Spontaneous PM is generally described as a benign condition, presenting in young adults exposed to a sudden pressure change within the intrathoracic cavity. According to first report of pathogenesis of spontaneous PM, a sudden increase in intrathoracic pressure results in increased intra-alveolar pressure [2]. The pressure differential created within the pulmonary parenchyma leads to alveolar rupture with further leakage of air throughout the interstitium and bronchovascular tissue sheath following a centripetal pattern toward the mediastinum. In clinical practice, spontaneous PM often develops as a result of various precipitating events triggering a strong Valsalva maneuver, creating the pressure differential needed for its genesis. In respiratory PM, released alveolar air centripetally dissects through the pulmonary interstitium along the bronchovascular sheaths toward the pulmonary hila, into the mediastinum [3]. This pathophysiological mechanism was described by Macklin and Macklin [4], and is known as the Macklin effect. This effect appears on thoracic computed tomography (CT) as linear collections of air contiguous to the bronchovascular sheaths [5-7]. The air dissects into the pulmonary hila and from there enters the mediastinum.

Secondary PM comprises the bulk of experience with PM. In the majority of cases, it is initiated by blunt or penetrating trauma, recent interventions in the esophageal or tracheobronchial tree, rupture of a hollow viscus, tissue dissection originating from spontaneous pneumothorax, pulmonary or mediastinal infection by gas-forming organisms [8]. Spontaneous PM is often difficult to differentiate form more subtle causes of secondary PM, such as contained esophageal perforation, minor tears in the central tracheobronchial tree, and smoldering pulmonary and mediastinal infections. It is essential to confirm the absence of a secondary pathological event responsible for
the presence of mediastinal air.

In this retrospective study, we examined backgrounds, onset, treatments and outcomes of PM and compared spontaneous and secondary PM.

Patients and Methods

Patients who diagnosed as PM on CT in Kanagawa Prefectural Ashigarakami Hospital were entered in this retrospective study. Patients who experienced recent aero digestive tract interventions, recent thoracic, cardiac, abdominal, or cervical surgical interventions, cervical soft tissue or intrathoracic infection and esophageal perforation were excluded. Trauma-related patients were also excluded. They were divided two groups; spontaneous and secondary PM. Secondary PM was defined who have other pulmonary lesions. We examined their backgrounds such as age, weight, height, smoking history, and coexisting illness, onset of the PM, treatments and outcomes. We compared these factors in each groups.

Results

Between January 2011 and February 2016, fifteen patients were detected PM on Chest CT (Figure 1). There were ten patients in spontaneous PM (Table 1). Nine were male and another was female. Their age range was 16 to 25 years old. Their heights were various, but weights of them were 46.6 kg to 62.0 kg and body mass index (BMI) was low such as median 18.1 and range 15.8 to 22.6. Smoker was only 2 patients. Nine patients visited our hospital because their cervical to anterior chest pain continued for several to 12 hours (Table 2). One patient was discovered at unconsciousness status and was transferred by emergency car. Five of 10 patients experienced pain attack at rest or desk working. Only one of 10 patients showed mild tachycardia and hypotension. Vital signs of other nine patients were normal. Nobody showed hypoxia. Besides, five patients of secondary PM were elderly aged 76 to 91 years old (Table 1). Their BMI was low such as median 17.1 and range 14.2 to 24.2. All 5 patients could not remember onset of their condition and visited our hospital because of vague cervical or chest pain or dyspnea continuing for several days (Table 2). Three of them showed tachycardia with low grade fever at administration and 1 showed hypoxia. Laboratory data in peripheral blood and biological examination revealed almost normal in every 15 patient. Every patient of both groups kept rest in bed. Seven and 3 patients received oxygen

Table 1: Patient characteristics.

<table>
<thead>
<tr>
<th>Total</th>
<th>Spontaneous number of patients</th>
<th>Secondary number of patients</th>
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<tbody>
<tr>
<td>Age (years)</td>
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<tr>
<td>Gender</td>
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<td>Height (cm)</td>
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<td>Weight (kg)</td>
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<td>BMI</td>
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<td>Complication</td>
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<td>Smoking</td>
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Table 2: Patient complaints and vital signs.

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<tr>
<th>Onset</th>
<th>Spontaneous number of patients</th>
<th>Secondary number of patients</th>
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<tbody>
<tr>
<td>Symptoms*</td>
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<tr>
<td>Duration (onset to visit)</td>
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<tr>
<td>Fever</td>
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* Some patients complained several symptoms.
treatment in spontaneous and secondary group, respectively (Table 3). Two patients in spontaneous group required pain reliever drugs. One patient with secondary PM received corticosteroid therapy against his complicated interstitial pneumonitis. Spontaneous PM improved after administration in 2 to 9 days. PM recurred after 6 and 7 months in 2 patients but improved at rest. On the other hands, secondary PM of 3 patients improved in 6 to 15 days, however other 2 patients did not show the recovery because they died at day 6 or at day 30. Every 5 patients complicated secondary PM died within 3 months because of coexisting pulmonary illness.

Discussion

The diagnosis of PM is revealed by radiographic examination. Caceres et al. [9] described that only 69% of cases were discovered by plain chest X-ray. It was difficult to detect PM with only a plain chest X-ray film and required CT for its diagnosis in every patient in our study. The similar data was described in previous report [10]. CT has the gold standard for diagnosing this lesion. Respiratory PM is a result of rupture along the alveolar tree, which leads to an abrupt increase in the intra-alveolar pressure. Released alveolar air centripetally dissects through the pulmonary interstitial along the bronchovascular sheaths toward the pulmonary hila, into the mediastinum [3,11], CT scans of our patients did not show these signs, who admitted after several hours passed from onset, it is possible these signs on CT disappear within several hours from its onset.

Most series describe the presence of a triggering event before the development of spontaneous PM. Numerous immediate preceding events have been noted, including forceful emesis, intense coughing, inhalational drugs, physical activity, intense screaming, spirometry, childbirth, bronchospasm, and playing of wind instruments [12–17]. There are variable data in the literature regarding triggering events responsible for the origin of the spontaneous PM; however, reported triggering events lists the most frequent ones as emesis, intense physical activity, coughing, and intense screaming. The trigger of our defined secondary PM was considered intense coughing and intense screaming, however 5 of 10 spontaneous group experienced at rest or desk work. They did not remember trigger of PM, they might be caused as a result of a mild to moderate Valsalva maneuver at desk work.

Nine of ten spontaneous PM in our study occurred in young male adults. Their median BMI was 18.1. This showed the lesion occurred in patients with slender body which is similar to spontaneous pneumothorax. Pulmonary organ might develop gradually and complete in 20 to 30 years old. Tracheobronchial tree might be weak at end-stage of growth phase. Muscle or fat tissues of slender body are considered less. Thus this PM may beoccur in young slender adult. Otherwise, secondary PM without traumatic events occurred as a result of fibrotic destroyed lung. Secondary pneumothorax occurs in pulmonary lesions to induce fibrotic change. Both PM and pneumothorax is terminal phenomenon of destroyed lung disease. Thus, we agreed all patients experienced secondary PM died within 3 months.

Although chief complaint was chest pain, pain reliever was required in only 2 patients. This might suggest PM recovery initiated from onset of the lesion. Both spontaneous and secondary PM in our study did not impair cardiac and pulmonary functions at admission. Improvement of PM was rapid in several days by rest and special treatment such as pneumothorax is not required. PM in our study, whichever spontaneous or secondary coexisting pulmonary lesions, were improved within 2 weeks and are considered a benign condition.

In conclusion, we compared PM between spontaneous and secondary with coexisting pulmonary illness. Both occurred in patients with slender body and improved in several days to 2 weeks by rest with or without oxygen therapy. Patients of spontaneous PM have well prognosis, however those of secondary PM showed poor because they die with coexisting pulmonary disease.

References


