Introduction

Spontaneous hemopneumothorax is a rare disease with a simultaneous accumulation of air and blood in the pleural cavity with no trauma or obvious causes. Usually, spontaneous hemopneumothorax occurred in 0.5%–12% of spontaneous pneumothorax. Its clinical feature is dramatic because of the hypovolemic shock by a rapid progress of symptoms unlike pneumothorax.1)

We report 17 patients of spontaneous hemopneumo-
rax out of 983 patients of spontaneous pneumothorax during a course of 12 years from March 1994 to February 2006 to find out the causes and proper treatments through reviews of the literature.

Patients and Methods

A retrospective study was done on 983 patients with spontaneous pneumothorax who had been treated from March 1994 to February 2006. In this study, spontaneous hemopneumothorax was defined as the accumulation of more than 400 mL of blood in the pleural cavity, based on the criteria of Ohmori and associates. And the bleeding volume of less than 400 mL was regarded as traumatic bleeding of the chest tube insertion, so it was exempted. Seventeen patients (1.7%) out of the 983 had spontaneous hemopneumothorax. We analyzed many factors such as the sex and age distribution, the affected site, clinical symptoms, the bleeding volume, causes, treatments, complications, and others.

Results

Sex and age distribution
All 17 patients with spontaneous hemopneumothorax were males. Except for an older patient of 60 years, all were of a young age; besides the elderly patient, their ages ranged from 16 to 33 years (the average age was 19.5 years). Five patients (29.4%) were teens, 10 patients (58.8%) were in their 20s, and only 1 (5.9%) was in his 30s.

Clinical symptoms and past history
Symptoms of spontaneous hemopneumothorax were similar to those of pneumothorax. Pain and discomfort of the chest were the initial symptoms in all 17 patients, and dyspnea also appeared in 8. With the progress of hemopneumothorax, 6 patients developed hypotension and became lethargic resulting from a loss of blood.

In all cases, none had a history of pneumothorax before the spontaneous hemopneumothorax attacked. Moreover, none had a history of pulmonary tuberculosis, or at least any evidence that the patient suffered from tuberculosis as shown in the chest radiograph or a lung high resolution computed tomography. Thirteen patients (76.5%) smoked about an average of 8.6 pack-years (Table 1).

Affected sites and degrees of affliction
The affected sites of 17 patients were on the right side in 6 patients and on the left in 11, so the occurrence on the left side is significantly dominant. A patient of hemopneumothorax (no.10) on the left side simultaneously had a mild degree of pneumothorax on his right side; the right-sided pneumothorax was treated by the oxygen inhalation method. According to classification by Rhea and associates, the degrees of collapsed lung are divided into mild (below 20%), moderate (20%–40%), and severe (more than 40%). All patients had severe degrees of pneumothorax, and the average collapsed rate was 64.1%. The bleeding amount was estimated by the drained volume of chest bottle combined with the accumulated and coagulated blood of the pleural cavity on the operating field, which ranged from 450 mL to 2,900 mL (average 1,308.8 mL). Eight patients needed the homologous blood transfusion; 2 (nos. 3 and 13) were transferred to our hospital after the transfusion. One patient (no. 13) had only a simple pneumothorax for which the pleural adhesion band was located between the lung apex and Sibson’s fascia in the first chest radiograph. But the massive bleeding was suddenly drained into the chest bottle after the chest catheter insertion (Fig. 1).

Treatment and management
Therapeutic policy was decided based on each individual patient’s condition and clinical features of hemopneumothorax. All patients were initially treated with closed thoracostomy: only closed thoracostomy in 5 patients (29.4%), and the closed thoracostomy combined with video-assisted thoracic surgery (VATS) or thoracotomy in 12 (70.6%). One patient (no. 4) was stanched after 450
Spontaneous Hemopneumothorax


mL bleeding. However, since large bullae were discovered in the chest radiograph and computed tomography on the next day, we performed VATS on the fourth day of admission. Another patient (no. 7) with giant bullae needed ventilator therapy for two days because of the reexpansion pulmonary edema after thoracostomy. After he got better, the giant bullae were removed on the seventh day by thoracotomy. There was one patient (no. 3) who was treated by the closed thoracostomy and blood transfusion, since he refused a surgical operation. But the coagulated blood got into the trapped lung, so he had to be treated with thoracotomy 10 days later. The average bleeding amount during the operation with a general anesthesia was 1,671 mL. In many cases of heavy bleeding, VATS or a thoracotomy was used.

Posthistory

One patient (no. 2), treated only with closed thoracostomy, had relapsed into ipsilateral pneumothorax 1.5 months later, so he needed to be operated with VATS after the thoracostomy. In 2 other patients (nos. 6 and 16), they were operated on with VATS after the thoracostomy because of the recurrence of contralateral pneumothorax 1 year later and 8 months later, respectively.

Discussion

Spontaneous hemopneumothorax is known as a disease that has occurred in young males from their 20s to their 40s more than in females, according to the description by Laennec, since 1829. Therefore the occurrence of male spontaneous hemopneumothorax is much higher than that of spontaneous pneumothorax. Males dominated 100% of spontaneous hemopneumothorax in our study.

Also, spontaneous hemopneumothorax is a comparatively uncommon disease; previous reports state that it occurs in about 0.5%–12% of spontaneous pneumothorax patients. In our study, it was 1.7% in 17 patients from among 983 total patients.

The mechanisms of spontaneous hemopneumothorax can be caused by a torn pleural adhesion between the parietal and visceral pleura as a result of the torn congenital aberrant vessels between the parietal and visceral pleura, or because of the rupture of the vascularized bullae and underlying lung parenchyma. But there is a ruptured arterial stalk in the bleeding part of the pleural cavity in the operating field in all patients of our study.

We could make certain of the arterial stalk of the pleural

Table 1. Summary of preoperative data and treatments of 17 patients

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Sex/age</th>
<th>Symptoms</th>
<th>Tbc history/smoking (pack-year)</th>
<th>Collapse (%)(R/Lt)</th>
<th>Loss volume (mL)</th>
<th>Transfusion (pint)</th>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/19</td>
<td>pain³</td>
<td>–/–</td>
<td>–/+ (50%)</td>
<td>1,800</td>
<td>2</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>2</td>
<td>M/26</td>
<td>pain, dyspnea</td>
<td>–/–</td>
<td>+ (50%)/–</td>
<td>1,200</td>
<td>4</td>
<td>CTD</td>
</tr>
<tr>
<td>3</td>
<td>M/25</td>
<td>pain, lethargy</td>
<td>–/–</td>
<td>–/+ (60%)/–</td>
<td>2,200</td>
<td>3</td>
<td>CTD+T³</td>
</tr>
<tr>
<td>4</td>
<td>M/18</td>
<td>pain, dyspnea</td>
<td>–/–</td>
<td>–/+ (80%)/–</td>
<td>450</td>
<td>–</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>5</td>
<td>M/22</td>
<td>pain, dyspnea</td>
<td>–/–</td>
<td>–/+ (80%)/–</td>
<td>1,500</td>
<td>–</td>
<td>CTD</td>
</tr>
<tr>
<td>6</td>
<td>M/16</td>
<td>pain</td>
<td>–/–</td>
<td>+ (80%)/–</td>
<td>1,200</td>
<td>–</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>7</td>
<td>M/26</td>
<td>pain, lethargy</td>
<td>–/–</td>
<td>+ (100%)/–</td>
<td>2,900</td>
<td>6</td>
<td>CTD+T</td>
</tr>
<tr>
<td>8</td>
<td>M/20</td>
<td>pain, dyspnea</td>
<td>–/–</td>
<td>–/+ (70%)/–</td>
<td>500</td>
<td>–</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>9</td>
<td>M/24</td>
<td>pain</td>
<td>–/–</td>
<td>–/+ (70%)/–</td>
<td>1,200</td>
<td>2</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>10</td>
<td>M/18</td>
<td>discomfort³</td>
<td>–/–</td>
<td>+ (10%)³/+ (60%)</td>
<td>500</td>
<td>–</td>
<td>O₂/CTD</td>
</tr>
<tr>
<td>11</td>
<td>M/21</td>
<td>pain, lethargy</td>
<td>–/–</td>
<td>+ (60%)/–</td>
<td>1,300</td>
<td>–</td>
<td>CTD</td>
</tr>
<tr>
<td>12</td>
<td>M/60</td>
<td>pain, dyspnea</td>
<td>–/48</td>
<td>–/+ (50%)</td>
<td>500</td>
<td>–</td>
<td>CTD</td>
</tr>
<tr>
<td>13</td>
<td>M/27</td>
<td>pain, dyspnea, lethargy</td>
<td>–/10</td>
<td>–/+ (70%)/–</td>
<td>2,700</td>
<td>2</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>14</td>
<td>M/24</td>
<td>pain, lethargy</td>
<td>–/7</td>
<td>–/+ (50%)</td>
<td>1,500</td>
<td>2</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>15</td>
<td>M/23</td>
<td>pain, dyspnea</td>
<td>–/3</td>
<td>–/+ (60%)/–</td>
<td>600</td>
<td>–</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>16</td>
<td>M/15</td>
<td>pain, lethargy</td>
<td>–/–</td>
<td>–/+ (50%)/–</td>
<td>600</td>
<td>–</td>
<td>CTD+VATS</td>
</tr>
<tr>
<td>17</td>
<td>M/33</td>
<td>pain, dyspnea</td>
<td>–/17</td>
<td>–/+ (50%)</td>
<td>1,600</td>
<td>3</td>
<td>CTD+VATS</td>
</tr>
</tbody>
</table>

(1) history of pulmonary tuberculosis; (2) affected sites; (3) chest pain or discomfort; (4) thoracotomy; (5) simultaneous pneumothorax; (6) nasal O₂ inhalation therapy.
adhesion band in the removed bullae by VATS (Fig. 2).

There was no case of spontaneous hemopneumothorax by a sequela of tuberculosis in our study. It is thought that this is why a pleural adhesion band is not easily torn by the lung collapse, since tuberculosis forms a thick and dense pleural adhesion. Also, the smoking ratio of patients with spontaneous hemopneumothorax, 13 (76.5%) of 17 patients, was higher than that of spontaneous pneumothorax patients in general. It is thought that light respiratory inflammation by smoking forms more adequate pleural adhesion causing spontaneous hemopneumothorax than tuberculosis is expected to form.

The vessels of adhesion normally do not contract because of the thin vessel wall between intimal and medial fibrosis as well as lack of the muscle layer. In addition, the negative pressure in the pleural cavity causes a continuous bleeding. An active bleeding tends to continue without being stanched easily; hemostasis does not depend on vasoconstriction, but on the diameter of the vessel and on the declined blood pressure resulting from blood loss. Therefore the gravity of symptoms of spontaneous hemopneumothorax depends mainly on the volume of the blood loss rather than on the degree of lung collapse as the result of an air leak. And the clinical feature of spontaneous hemopneumothorax was dramatic because of the emergency circumstances by hypovolemic shock, so it needs an intensive treatment.

The treatment of spontaneous hemopneumothorax is similar to that of spontaneous pneumothorax. The goals of treatment include hemostasis and reexpansion of the lung by a rapidly closed thoracostomy. The reexpansion of an affected lung allows an estimation of the volume of blood loss by evacuated blood from the pleural cavity and makes it easy to stanch the bleeding to compress the torn vessels. Next, the treatment of spontaneous hemopneumothorax must be followed by fluid resuscitation and the blood transfusion. In case of persistent bleeding, an urgent VATS or thoracotomy is necessary. But in the process of treatment, most patients do not need a transfusion because spontaneous hemopneumothorax appears mostly at a young age, and patients in this age group are able to recover from the postoperative anemia in spite of a large amount of bleeding.

The indications for VATS or thoracotomy are the hypovolemic shock, continuous bleeding (100/h), persistent air leak, impaired lung expansion, pachypleuritis, recurrent pneumothorax. A VATS or thoracotomy makes it possible to stanch the bleeding and evacuate coagulated blood from the pleural cavity, seal the air-leak spot on the lung surface by resecting the areas of emphysematous bullae, and secure effective drainage by chest tube placement under direct vision. In our hospital, VATS is preferred to axillary thoracotomy. There are two advantages to VATS for fast recovery: one is the shortened operation time that lessens the loss of bleeding, and the other is the easier access to the bleeding points near the Sibson’s fascia and the first rib.

Conclusions

We report 17 patients with spontaneous hemopneumothorax out of 983 patients of spontaneous pneumothorax in the Thoracic and Cardiovascular Surgery of Hanil General Hospital from March 1994 to February 2006. In the 17, spontaneous pneumothorax was dominant on the left side rather than the right side. It was caused by the torn pleural adhesion band according to the progress of lung collapse. The mechanisms of hemopneumothorax were mainly an air leak on the lung surface from ruptured bullae, and rarely an additional external pressure during drainage procedure. Spontaneous hemopneumothorax is different from pneumothorax in the aspect of urgency with hypovolemic shock by a rapid progress of symptoms. So we emphasize the importance of early recognition and prompt surgical intervention by VATS or axillary thoracotomy. We preferred VATS to axillary thoracotomy because VATS allows for lesser blood loss in a relatively shorter operation time than required by thoracotomy and also easy accessibility to the bleeding point near the Sibson’s fascia.
References


