Massive hemoptysis: what place for medical and surgical treatment

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Abstract

Objective: The objective of the study was to define timing of surgical treatment in management of massive hemoptysis. Methods: We performed a retrospective review of all patients admitted for massive hemoptysis in the intensive care unit of our thoracic surgery department. Treatment was managed according to the patient’s status, the etiology of bleeding, the findings of bronchoscopy and computed tomographic scan. Therapeutic measures available were medical treatment, tracheal intubation (single or double lumen tube), interventional endoscopy, arterial embolisation and surgical treatment. Results: Between September 1996 and December 2001, 43 patients were treated (nine females and 34 males with mean age of 54 years, range from 32 to 79). The mean red cell blood transfusion per patient was 1.57 Units. The patients were classified into three groups: Group 1, 11 patients were operated on immediately close to the bleeding crisis (five lobectomy and six pneumonectomy); Group 2, five patients for whom operation was delayed from the 7th to the 22nd day after cessation of bleeding (five lobectomy); Group 3, 27 patients were treated by non-surgical methods (medical treatment, endobronchial treatment, percutaneous embolisation). Fifteen patients underwent an arterial embolisation, which was complete in 13 cases. Among the five patients of group 2, cessation of bleeding was obtained by bronchial embolisation in four cases. Considering the whole series, 10 (23%) patients died: three (19%) patients in group 1, zero in group 2, seven (26%) in group 3. In two patients who were suffering from tumor necrosis, hemoptysis relapsed leading to death. Conclusion: Emergency thoracotomy for massive hemoptysis is at high risk. In case of bleeding from the arterial bronchial vessels, embolization may enable to postpone surgery and operate secondarily. In case of bleeding from the pulmonary vessels (tumor necrosis), surgical treatment must be immediate. An algorithm for management is proposed. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Hemoptysis; Respiratory distress; Bronchial carcinoma; Tuberculosis; Bronchectasis; Bronchial arteries

1. Introduction

In the beginning of last century, hemoptysis was pathognomonic of advanced phthisis [1,2]. Nowadays, chronic inflammatory pulmonary diseases and lung cancer are the most frequent causes in our country [3,4]. For majority of the patients, blood spitting or blood present in the sputum constitutes a worrying symptom rarely taken lightly. Hemoptysis can be a ‘lucky’ symptom allowing diagnosis of small bronchial carcinoma. However, when it is massive, patient’s life is threatened and emergency admission in intensive care unit must be achieved. The threat lies in asphyxiation by flooding of the tracheobronchial tree [5]. Before definitive treatment, the risk remains even after cessation of an episode of massive hemoptysis because relapses are unpredictable. It is a medical emergency associated with a 30–50% rate of mortality reported in the last 20 years [6]. The surgical treatment in the management of these patients has been reduced since the first report of bronchial artery embolization by Remy et al. [7]. A new multidisciplinary approach associating the intensive care physician, the endoscopic pneumologist, the radiologist and the thoracic surgeon enables to lower the death rate. In our department, the included intensive care unit is the major referral center intended to manage massive hemoptysis. In this multidisciplinary approach, the place of surgical treatment between medical treatment and bronchial artery embolization is not obvious. We retrospectively reviewed the files of 43 patients treated for massive hemoptysis in the last 4 years to try to answer to that question.
2. Patients and methods

2.1. Selection of patients and management

We considered the hemoptysis massive when the patient’s life was threatened at short term or, as commonly defined, the expectoration of 600 ml or more of blood over 24 h. All patients included in this study satisfied those criteria.

Patients’ assessment was conducted using the same procedure but was guided by seriousness of the situation. The main objectives of treatment were to prevent asphyxiation, to localize the site of bleeding, to stop the hemorrhage, to determine the etiology of hemoptysis, and to avoid the recurrence of hemoptysis definitively. Therapeutic means used were: pulmonary isolation, surgical treatment, interventional bronchoscopy, arterial embolization, and medical therapy. Those means were adapted to the cause of the hemoptysis, which was supposed by the past medical history and clinical examination of the patient, and confirmed by chest X-ray, bronchial endoscopy and computed tomography (CT) scan. Medical treatment included rest in bed, insertion of a wide-bore intravenous cannula, monitoring of arterial blood gases, aerosoltherapy of adrenaline, adapted antibiotic therapy if needed, and correction of clotting disorder if associated. An intravenous infusion of vasopressin (20 units over 15 min continued by 0.2 units/min for 36 h) was prescribed in the absence of contra-indication. Baseline hematology, biochemistry, and clotting test were obtained. Collected sputa were stained for bacteria, acid-fast bacilli and fungi. A chest X-ray was the first morphologic examination performed. A bronchial endoscopy was subsequently done. Its objective was threefold: to see the cause of bleeding if possible; to localize the site of bleeding; and to carry out endobronchial control measures [8]. These consisted in adenine serum lavage (adenine-saline solution 1‰) and laser YAG electocotery. A CT-scan was then achieved. It was always performed in case of arterial embolisation or surgical treatment. When an arterial embolisation was achieved, infusion of vasopressine was carried on until the arteriography. Arterial embolisation was routinely performed by a Seldinger technique through femoral access. An emergency surgical treatment was applied when the site of bleeding was localized, the indication of pulmonary resection justified and the other means of treatment having failed. The surgical treatment was postponed as far as possible after cessation of bleeding using the other means of treatment. It was only considered when the patient had sufficient pulmonary reserve and when the bleeding source was clearly identified. When the surgical treatment was not possible (leak pulmonary function or diffuse lesions), the patient was informed about the possible recurrence of hemoptysis even after a successful arterial embolisation [8,9]. In case of massive acute bleeding, isolation of the bleeding lung from the healthy one was achieved by the use of a double lumen endotracheal tube. In that case, the choice between operation or non-surgical treatment was taken after the CT-scan.

2.2. Population

Between September 1996 and December 2001, 43 patients with massive hemoptysis were treated in the intensive care unit of the thoracic surgery department. There were 34 men and nine women aged 32–79 years (mean 54). All of them underwent a bronchoscopy and a CT scan. The causes of hemoptysis (Fig. 1) were chronic pulmonary inflammatory diseases in 28 cases (bronchiectasis 12 cases, tuberculosis sequelae four cases, atypic mycobacteriosis one case, aspergillosis one case, unknown etiologies 10 cases), lung cancer in 12 cases, necrotizing pneumonia in three cases. Additional factors to bleeding included loss of anticoagulant control in six patients (coumarinic in four cases and ticlopidine in two cases). Blood transfusions were achieved in 21 patients (1–6 units of red blood cell, mean 1.57 unit per patient). According to the treatment applied, patients were classified in three groups:

2.2.1. Group 1: Immediate operation, close to the bleeding crisis.

A surgical treatment was performed close to the bleeding crisis in 11 cases (see Table 1, part A). All patients had a bronchoscopy and a CT scan before being operated, but only two underwent a lung function test performed previously by their general physician. In nine cases, immediate operation was performed because of persistence of bleeding in spite of medical treatment. So, a pulmonary isolation by insertion of a double lumen endotracheal tube was necessary in three of these nine patients. The operation was performed after hemodynamic stabilization of the patient and after confirmation of a localized lesion.

In two cases (case # 4 and # 8), bleeding was stopped, but operation was nevertheless immediately achieved to avoid fatal recurrence of hemoptysis coming from the pulmonary artery: In case # 8, the patient had been treated since 4 months for a cavitating asymptomatic lesion (Fig. 2) suspected to be sequella of tuberculosis. A bronchial arterial embolisation was attempted. As it was normal embolisation was not performed, but a pulmonary angiography showed that bleeding was coming from the left pulmonary artery (Fig. 3) ulcerated. The patient was immediately operated as it was a lung cancer. Cause of bleeding and type of operation performed are presented in Table 1, part A.

2.2.2. Group 2: delayed surgical treatment

A delayed surgical treatment was carried on in five cases after a second hospitalization respectively at the 7th, 9th, 16th, 17th and 22nd day after acute massive hemoptysis. Cessation of hemorrhage was obtained by medical treatment in one case and by arterial embolisation in four cases. In all five cases, this stopped the bleeding and pulmonary isola-
tion was useless. The lesions responsible for hemoptysis have always been localized and cardiopulmonary function was prepared for planned surgery. In all cases, a selected pulmonary resection was performed (see Table 1, part B).

2.2.3. Group 3: non-surgical treatment

A non-surgical treatment was achieved in 27 cases. Those patients had either too diffused lesions or too weak cardiopulmonary function to undergo a surgical treatment. All of them received the medical treatment described above associated with local therapy or arterial embolisation.

Local therapy (adrenaline serum lavage) was applied during bronchoscopy when pulmonary isolation was not necessary. Laser electrocoagulation was achieved in one case of inoperable lung cancer recurring after medical treatment.

Arterial embolisation was attempted in 11 cases but successfully achieved in nine cases leading to cessation of bleeding: In one case the vasoocclusion was not completely achieved because of arising of an anterior spinal artery; the other case, above described (case # 8), bronchial arteries were normal (Fig. 2).

3. Results

Altogether, 16 patients were operated (group 1 and group 2), out of which four were embolised before being secondarily operated. The other patients were treated by others non-surgical methods. None of the patients who underwent arterial embolisation alone or surgical treatment had recurrent hemoptysis at the time of follow-up extended from 3 months to 4 years (mean 1080 days). The hospital mortality was 23% (ten patients) in all the series. The mortality rate group wise was 27% (three patients) in group 1, zero in group 2, and 26% (seven patients) in group 3; by type of treatment applied it was: 19% (three patients) after surgical treatment and 26% (seven patients) after non-surgical treatment.

Two patients who suffered from a necrotic lung cancer died from a cataclysmic relapse hemoptysis. In one case, it was an inoperable central bronchial carcinoma in the course of radiotherapy, and in the second the patient suffered from an operable necrotic lung cancer. Medical treatment was applied and operation was planned the day after. A cataclysmic massive relapse led to the patient’s death before the operation in spite of an immediate selective intubation.

One patient died from acute arrhythmia attributed to an overflow of intravenous glypressin. Other deaths were consecutive to bacterial pneumonia and acute respiratory distress syndrome.

4. Discussion

Blood flooding into the tracheobronchial tree may arise from two vascular networks spreading into the pulmonary
Table 1
Demography of patients operated on Part A: (group 1) operation performed close to the bleeding crisis; and Part B: (group 2) operation delayed after cessation of bleeding

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Sex/age</th>
<th>Cause of bleeding</th>
<th>Localization</th>
<th>Embolisation</th>
<th>Surgery</th>
<th>Delay (days)</th>
<th>Outcome</th>
<th>Follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Part A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>M/50</td>
<td>Abscess</td>
<td>Left lower lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>0</td>
<td>ALI</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>M/73</td>
<td>Tuberculosis</td>
<td>Left upper lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>1</td>
<td>Uneventful</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>F/62</td>
<td>Lung cancer</td>
<td>Right lower lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>2</td>
<td>Uneventful (cancer recurrence)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M/51</td>
<td>Lung cancer</td>
<td>Right lower lobe</td>
<td>Pulmonary angiography</td>
<td>Lobectomy</td>
<td>3</td>
<td>Uneventful</td>
<td>32</td>
</tr>
<tr>
<td>5</td>
<td>M/59</td>
<td>Lung cancer</td>
<td>Left upper lobe</td>
<td>No</td>
<td>Pneumonectomy</td>
<td>1</td>
<td>Uneventful</td>
<td>24</td>
</tr>
<tr>
<td>6</td>
<td>M/57</td>
<td>Lung cancer</td>
<td>Right upper lobe</td>
<td>No</td>
<td>Lobectomy + decortication</td>
<td>1</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>M/52</td>
<td>Necrotizing pneumonia</td>
<td>Right lower lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>0</td>
<td>Death</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>M/58</td>
<td>Lung cancer</td>
<td>Left upper lobe</td>
<td>Pulmonary angiography</td>
<td>Pneumonectomy</td>
<td>0</td>
<td>Uneventful</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>F/51</td>
<td>At. Myco.</td>
<td>Right upper lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>0</td>
<td>Uneventful</td>
<td>5</td>
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<tr>
<td>10</td>
<td>F/72</td>
<td>CPID</td>
<td>Left upper lobe</td>
<td>Unsuccessful embolisation</td>
<td>Pneumonectomy</td>
<td>1</td>
<td>Death</td>
<td></td>
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<tr>
<td>11</td>
<td>M/63</td>
<td>Necrotizing pneumonia</td>
<td>Left upper lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>0</td>
<td>Uneventful</td>
<td>8</td>
</tr>
<tr>
<td>Part B</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>F/54</td>
<td>Bronchiectasis</td>
<td>Left lower lobe</td>
<td>Uncompleted embolisation</td>
<td>Lobectomy</td>
<td>9</td>
<td>Uneventful</td>
<td>40</td>
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<td>13</td>
<td>M/63</td>
<td>CPID</td>
<td>Left lower lobe</td>
<td>Yes</td>
<td>Lobectomy</td>
<td>17</td>
<td>Uneventful</td>
<td>36</td>
</tr>
<tr>
<td>14</td>
<td>M/76</td>
<td>CPID</td>
<td>Left lower lobe</td>
<td>Yes</td>
<td>Lobectomy</td>
<td>7</td>
<td>Uneventful</td>
<td>33</td>
</tr>
<tr>
<td>15</td>
<td>M/42</td>
<td>CPID</td>
<td>Right upper lobe</td>
<td>Yes</td>
<td>Lobectomy</td>
<td>16</td>
<td>Uneventful</td>
<td>54</td>
</tr>
<tr>
<td>16</td>
<td>M/78</td>
<td>Lung cancer</td>
<td>Right lower lobe</td>
<td>No</td>
<td>Lobectomy</td>
<td>22</td>
<td>Uneventful</td>
<td>45</td>
</tr>
</tbody>
</table>

CPID, chronic pulmonary inflammatory disease; M, male; F, female; ALI, acute lung injury; At. myco., atypical mycobacteriosis.
tissue: the bronchial and the pulmonary arterial systems. Bleeding coming from the former, results from neovascularisation of the lung systemic vessels, which quite often is induced by an inflammatory pulmonary disease or defect of the pulmonary arterial system [10]. Many inflammatory pulmonary diseases are able to produce such neovascularisation: bronchiectasis, suppurative lung disease, mycobacteriosis… Blood irruption stems from an erosion or a breaking of this hypertrophic neovascularisation. These vessels are endowed with a mural musculature wall, which is able to contract (arteriolar smooth muscle). Vasospasm of this network may be produced either by pharmacological methods (vasoactive drug as vasopressin or aerosolized adrenalin) or by physical methods (bronchial ice-cold saline lavage) able to produce temporary slowing or cessation of the bleeding.

Percutaneous embolisation is also a reliable method to eliminate this hypertrophic neovascularisation [4,5,7]. However, results are not stable and relapses are possible if the underlying disease responsible for promotion of neovascularisation is not removed [8,9]. None of the patients in our series treated by arterial embolisation without surgery had relapses. It is therefore, a temporary treatment, which stops acute bleeding in order to prepare the patient to a delayed surgery if indicated. Absence of tracheobronchial hemorrhage allows for safe operative intervention, with better delineation of the pulmonary disease and selection for the best economic pulmonary resection. Operation during bleeding crises may indeed precipitate emergency pneumonectomy. It is also better to operate once the bronchial tree has been effectively cleared and the pulmonary parenchymal and pulmonary vasculature reserve recovered. This explains the difference of death between group 1 (27%) and 2 (no death). Nevertheless, this difference is not statistically significant due to the number of patients included in the series.

Unlike bronchial vessels network, the pulmonary arterial one is not able to vasospasm as powerfully as bronchial vessels. The wall of these vessels is thin and there is no active contraction. Vasoactive drugs or physical agents as ice-cold saline lavage have mild effects. Bleeding from these vessels generally comes through an ulceration of the vascular wall caused by destructive processes of the lung whatever the pathogenesis could be, such as, for instance, necrotizing bacterial pneumonia, necrotizing lung cancer, or aspergillus cavitizing infection. In such cases, the arrest of bleeding is due to a temporary sealing of the vascular tear by a clot. Smelting of the clot or progression of the vascular tear leads to relapse of an even more massive and usually fatal hemoptysis: this concerned two patients in our series. So, in case of bleeding coming from the pulmonary vessels, operation, if possible, must be performed immediately without any...
need of bronchial arteriography. Unfortunately, selection of the patients in one of the two categories, that is bronchial or pulmonary bleeding, is not always so easy. However, some clues may predict the pulmonary artery vessels origin: fungal ball, lung abscess, presence of a cavity with emptying and refilling aspect (Figs. 4 and 5), cavity with an air–fluid level as defined by Thoms et al. [11] are predicting element. Without clues of pulmonary artery bleeding, a bronchial arteriography is indicated. It will be completed by a pulmonary angiography in case of normal arteriography.

Therefore, treatment of massive hemoptysis is not over, even when acute bleeding has been stopped by conservative treatment. A complete and, if necessary, invasive assessment for a precise diagnosis must always follow. In spite of this multidisciplinary approach associating the intensive care physician, the radiologist and the thoracic surgeon, massive hemoptysis is still an imminent threat to life. The in-hospital mortality rate in our series (25%) is not very different from those reported in literature (22.7% for Conlan et al. [3], 15% for Lee et al. [4]).

Surgical treatment is best performed in postponing date in case of bronchial vessels hemorrhage. In case of pulmonary vessels hemorrhage, the operation must be achieved immediately. Similarly, in case of massive bleeding requiring pulmonary isolation, immediate operation must be performed in case of localized lesion. This attitude is summarized in Fig. 6. Physiological lung exclusion may be an alternative technique [12] in case of dense vascular adhesion and pleural fibrosis, but, according to our experience, elective pulmonary resection is the optimal treatment, which was always possible in our series.

References


Fig. 5. Emptying and refilling mass. Chest X ray of the same patient as Fig. 4 performed after a hemoptysis crise (arrow).

Fig. 6. Algorithm of management for massive hemoptysis.

