Percutaneous Embolotherapy for Life-Threatening Hemoptysis*

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Study objectives: The management of life-threatening hemoptysis frequently poses a therapeutic dilemma because such patients are often poor surgical risks. Less often, patients refuse surgical intervention. The value of percutaneous embolotherapy, a useful alternative in these situations, was assessed.

Design, setting, patients, interventions: Sixteen consecutive patients who underwent percutaneous embolotherapy for life-threatening hemoptysis in a tertiary-care hospital were evaluated retrospectively. The bronchial arteries, as well as other intrathoracic arteries, were evaluated and selectively embolized if they were considered to supply the pathologic area from which the hemoptysis arose.

Results: The most common cause for hemoptysis was posttuberculous bronchiectasis (n = 12) with or without mycetomas. Ten patients required blood transfusions before embolotherapy. Pleural disease was noted on the chest radiograph in 13 patients and was generally associated with the presence of nonbronchial systemic collateral vessels. In three patients, arteries other than the bronchial arteries were the only source of hemoptysis. Percutaneous embolotherapy was successful in controlling the hemoptysis in all patients. The only complication documented was a transient paraparesis in one patient. Six patients did not return for follow-up. Of the remaining 10 patients, 3 patients had minor episodes of hemoptysis that were treated conservatively with success. One patient had significant recurrent hemoptysis that was managed with radiotherapy. One patient subsequently underwent a lobectomy.

Conclusion: Percutaneous embolotherapy is a useful therapeutic modality in the management of life-threatening hemoptysis. The contribution of nonbronchial systemic collateral vessels, particularly where there is evidence of coexistent pleural disease, should always be suspected. In experienced hands, this is a safe and potentially life-saving procedure.

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Key words: bronchial arteries; bronchiectasis; embolization; embolotherapy; hemoptysis; therapeutic

Treatment options for life-threatening hemoptysis are hampered by the need for swift diagnosis of the underlying pathology and by the urgency for an effective form of therapy that can be rapidly implemented. Such patients are often poor surgical candidates because of the presence of bilateral pulmonary disease or significantly impaired pulmonary function. Surgical management in the acute situation is associated with high mortality and high morbidity. The emergence of bronchial artery embolization as a therapeutic modality for life-threatening hemoptysis has revolutionized the management of these patients.

The term massive hemoptysis is loosely applied in the literature. Various authors use this phrase for the expectoration of an amount of blood varying from as little as 200 mL to as much as 1,000 mL over 24 to 48 h. The volume of blood loss is often underestimated because a significant amount may remain in the lung and may not be expectorated. Others have shown that the rate of blood loss is a better predictor of mortality.

We have used the term life-threatening hemoptysis to denote one of the following: (1) expectoration of at least 250 mL of blood in 24 h; (2) requirement for blood transfusion because of a significant decrease in hemoglobin level; or (3) failure or inade-
Materials and Methods

Patient Population

This is a retrospective study of patients presenting with life-threatening hemoptysis who underwent embolotherapy at Chris Hani Baragwanath Hospital, a tertiary-care institution in Johannesburg, South Africa. Embolotherapy was performed in 17 consecutive patients during an 18-month period (September 1996 to February 1998). All patients presented with a history of hemoptysis of at least 250 mL on the day of their hospital admission. In one of these patients (patient 4), embolotherapy was used as an interim measure before surgical resection was performed. In the other cases, embolotherapy was performed because the patients were deemed unsuitable for surgical intervention because of significant bilateral pulmonary disease (including active pulmonary tuberculosis in two patients), poor lung function, unresectable lung cancer, ongoing bleeding, or refusal of surgery (Table 1).

One patient was omitted from this study because the records of that patient were not available.

Table 1—Underlying Causes of Hemoptysis*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Underlying Etiology of Hemoptysis</th>
<th>Indication for Embolotherapy†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>58</td>
<td>Female</td>
<td>Post-TB bronchiectasis LUL</td>
<td>Refused surgery</td>
</tr>
<tr>
<td>2</td>
<td>48</td>
<td>Male</td>
<td>Destroyed RML due to previous Klebsiella pneumonia, mild bilateral basal bronchiectasis</td>
<td>Refused surgery</td>
</tr>
<tr>
<td>3</td>
<td>68</td>
<td>Male</td>
<td>Post-TB bronchiectasis, LUL mycetoma</td>
<td>Bilateral disease</td>
</tr>
<tr>
<td>4</td>
<td>39</td>
<td>Male</td>
<td>LUL mycetoma, spontaneous left pneumothorax</td>
<td>Persistent significant hemoptysis not responsive to conservative management (subsequently underwent LUL lobectomy)</td>
</tr>
<tr>
<td>5</td>
<td>37</td>
<td>Male</td>
<td>Active TB (sputum positive)</td>
<td>Bilateral disease, newly diagnosed TB</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>Male</td>
<td>Primary adenocarcinoma lung</td>
<td>Inoperable lung carcinoma</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>Male</td>
<td>Post-TB bronchiectasis</td>
<td>Bilateral disease</td>
</tr>
<tr>
<td>8</td>
<td>27</td>
<td>Male</td>
<td>HIV positive, TB treatment for 1 wk</td>
<td>Bilateral disease, newly diagnosed TB</td>
</tr>
<tr>
<td>9</td>
<td>27</td>
<td>Male</td>
<td>HIV positive, TB treatment for 2 wk</td>
<td>Bilateral disease, newly diagnosed TB</td>
</tr>
<tr>
<td>10</td>
<td>48</td>
<td>Male</td>
<td>Post-TB bronchiectasis</td>
<td>Bilateral disease</td>
</tr>
<tr>
<td>11</td>
<td>72</td>
<td>Male</td>
<td>LUL bronchiectasis</td>
<td>Persistent hemoptysis not settling with conservative therapy</td>
</tr>
<tr>
<td>12</td>
<td>44</td>
<td>Female</td>
<td>Post-TB bronchiectasis, LUL mycetoma</td>
<td>Poor lung function</td>
</tr>
<tr>
<td>13</td>
<td>59</td>
<td>Male</td>
<td>Post-TB bronchiectasis</td>
<td>Bilateral disease</td>
</tr>
<tr>
<td>14</td>
<td>50</td>
<td>Male</td>
<td>Post-TB bronchiectasis</td>
<td>Bilateral disease</td>
</tr>
<tr>
<td>15</td>
<td>61</td>
<td>Male</td>
<td>Post-TB bronchiectasis, LUL mycetoma</td>
<td>Bilateral disease, poor lung function</td>
</tr>
<tr>
<td>16</td>
<td>44</td>
<td>Female</td>
<td>Post-TB bronchiectasis, large cavity RUL</td>
<td>Bilateral disease</td>
</tr>
</tbody>
</table>

*Post-TB = posttuberculous; TB = tuberculosis; LUL = left upper lobe; RML = right middle lobe; RUL = right upper lobe.
†Patients with radiologically significant bilateral disease were considered unsuitable candidates for surgical management.

Embolo therapy

Angiography and embolization were performed using a high-resolution digital subtraction angiography unit (Angiostar; Siemens; Erlangen, Germany). Fourteen of the 16 patients were treated by the same radiologist (P.S.); two other radiologists were involved in the management of the remaining two patients. None of the patients required mechanical ventilation before, during, or after the embolization procedure.

Diagnostic thoracic aortography was performed via cannulation of the femoral artery in all patients. The number, location, and degree of pathologic enlargement of the bronchial arteries, as well as the presence of nonbronchial systemic collateral vessels (in particular, intercostal arteries from T4 to T7, subclavian arteries, and internal mammary arteries) were evaluated in all patients. After aortography, selective catheterization of all arteries responsible for hemoptysis, if technically feasible, was performed using 4F to 5F H1, C2, or Simmons catheters (Cordis; Miami, FL). Omnipaque (Nycomed Imaging; Oslo, Norway) was the contrast agent used.

Arteries were considered pathologic if they were hypertrophied, or demonstrated vascular blush or arteriovenous shunting. Extravasation of the contrast agent was not visualized in any patient. Embolization was performed using 250- to 750-μm polyvinyl alcohol particles (Trufill; Cordis). In one patient, standard steel coils (Cook; Bloomington, IN) were used to embolize the internal mammary artery. Diagnostic angiography was routinely performed during the embolotherapy procedure to exclude the presence of spinal arteries arising from the bronchial or intercostal arteries. Spinal arteries, if found before or after the angiogram and bronchial embolization procedure were lost.

Sixteen patients (13 men and 3 women) were therefore analyzed in this study. The age of the study population ranged from 27 to 72 years (median, 49 years). Two patients were HIV seropositive. The HIV status was not determined in five patients.
partial embolization, were not considered a contraindication to embolization. In these cases, the catheter was positioned so that the tip was distal to the origin of the spinal artery, and larger particles were then used. The rationale for this technique is that these larger particles will occlude the abnormal hypertrophied bronchial or intercostal arteries, but will be too large to pass into the smaller spinal arteries, thus avoiding occlusion of the latter.

A coaxial microcatheter system was not used in any patient. Pulmonary angiography was not performed in any patient.

Analysis

The following factors were examined in the study population: the underlying etiology for the hemoptysis, the presence or absence of pleural thickening (assessed on plain chest radiography) in the area of lung from which the bleeding had arisen, the arteries that required occlusion by embolotherapy, the lowest hemoglobin concentration measured before embolotherapy, and the incidence of complications consequent to the embolization procedure. Outcome at follow-up visits was also assessed.

RESULTS

Percutaneous embolotherapy was successful in aborting the acute episode of hemoptysis in all patients.

Underlying Cause of Hemoptysis

Twelve patients had underlying bronchiectasis, the etiology of which was posttuberculous in all patients except one (Table 1). Four of these patients also had mycetomas. Three patients had active pulmonary tuberculosis. One patient had inoperable primary bronchogenic adenocarcinoma.

Severity of Anemia Before Embolotherapy

An estimate of the volume of blood expectorated before embolization was documented in 11 of the 16 patients. In all patients except for one, the volume of blood expectorated was > 500 mL in a 24-h period. The lowest hemoglobin concentration recorded for each patient before embolotherapy varied from 4.4 to 12.3 g/dL (mean ± SD, 8.6 ± 2.7 g/dL). Ten patients required blood transfusions. One of these patients (patient 9) received > 10 U of packed RBCs during his hospital admission.

Presence of Pleural Disease

Pleural disease (thickening) in the region from which the hemoptysis originated was visible on the chest radiograph in all but three patients (Table 2).

Arteries Occluded

The arterial sources of hemoptysis were identified during angiography in each patient by the presence of abnormal vessels in the area of radiologically apparent parenchymal disease (Table 2). The chest radiograph and pre-embolotherapy and post-embolotherapy angiograms of patient 7 are shown in Figures 1–3.

In three patients, the source of bleeding was not from the bronchial artery. In these patients, the internal mammary or intercostal arteries supplied the abnormal vascular network.

In 13 patients, bronchial arteries supplied the radiographically abnormal area from which the he-

| Table 2—No. of Arteries Successfully Occluded by Embolotherapy, and Presence of Pleural Disease* |
|---|---|---|---|---|---|
| Patient No. | Side of Embolotherapy | Bronchial | Intercostal | Internal Mammary | Pleural Disease† |
| 1 | Left | 1 | 2 | 0 | Yes |
| 2 | Right | 0 | 0 | 1 | No |
| 3 | Left (ICBT) | 1 | 0 | 0 | Yes |
| 4 | Left | 1 | 0 | 0 | Yes |
| 5 | Right | 1 | 0 | 2 | Yes |
| 6 | Right | 1 | 0 | 0 | Yes |
| 7 | Right and left | 2 | 1 | 0 | Yes |
| 8 | Right | 1 | 0 | 0 | No |
| 9 | Right | 1 | 1 | 0 | Yes |
| 10 | Right and left (CBT) | 2 | 1 | 0 | Yes |
| 11 | Left | 1 | 3 | 0 | Yes |
| 12 | Left | 2 | 2 | 1 | No |
| 13 | Left | 1 | 0 | 1 | Yes |
| 14 | Right | 0 | 2 | 0 | Yes |
| 15 | Right and left (CBT) | 2 | 1 | 0 | Yes |
| 16 | Right | 0 | 1 | 0 | Yes |

*CBT = common bronchial trunk; ICBT = intercostobronchial trunk.
†Presence of pleural thickening as assessed on chest radiograph.
‡Small collateral vessels from the left lateral thoracic artery could not be cannulated.
§One other branch of the subclavian artery also embolized.
Hemoptysis had most likely arisen. However, in 11 of these patients, other arteries (intercostal arteries, the internal mammary artery, branches from the subclavian artery) also fed the abnormal network of vessels. We can only surmise that these nonbronchial collateral vessels played a role in the hemoptysis.

Of the 13 patients with pleural thickening adjacent to the radiologically abnormal area of lung parenchyma on the chest radiograph, all patients except three had vessels other than the bronchial arteries also involved in the abnormal vascular network. Of the three patients without obvious pleural thickening, two patients had vessels other than the bronchial arteries involved. It is possible that pleural disease may have been detected in these patients, had a more sensitive radiologic technique, such as CT, been used.

Three of the 13 patients with radiologic evidence of pleural disease did not undergo embolization of any nonbronchial systemic vessels. In patient 3, no systemic collateral supply was documented. Small collateral vessels were noted to originate from the left lateral thoracic artery in patient 4; these were too small to be selectively cannulated for embolization. Patient 6 was found to have a very tortuous right subclavian artery that prohibited cannulation and examination of the internal mammary artery and other branches.

Complications of Embolotherapy

One patient (patient 1) experienced transient paraparesis (previously documented) but subsequently recovered fully. Another patient who had active pulmonary tuberculosis and was HIV seropositive with a CD4 cell count of 184/µL developed disseminated herpes zoster 3 days after embolotherapy. A large right hemopneumothorax developed that required tube thoracostomy. This was consid-
ered an incidental adverse event unrelated to the embolization procedure. All other patients had an uneventful course.

Follow-up

The duration of follow-up for the 16 patients ranged from 0 to 28 months (median, 1.5 months). Six patients did not return for any follow-up visits. Patient 1 presented with hemoptysis (100 mL) 10 months after she underwent embolotherapy. This subsided with antibiotic therapy. Patient 4 underwent a left upper lobectomy 6 weeks after embolization for ongoing minor hemoptysis and a recurrent left pneumothorax. He was well when last seen 2 years postoperatively. Patient 6 was referred to the oncology service for palliative radiotherapy for his lung tumor. Patient 10 had three further hospital admissions for recurrent hemoptysis, all of which were resolved with conservative therapy (supplemental oxygen, morphine, and antibiotics). A CT scan performed after his last hospital admission demonstrated a left upper lobe mycetoma that had not been detected on his chest radiograph. Patient 12 was readmitted for significant hemoptysis 27 months after bronchial artery embolization. A repeat procedure was planned but was cancelled because of technical reasons. She was discharged after the hemoptysis resolved with conservative therapy. Patient 15 required two hospital admissions at 7 weeks and 12 weeks, respectively, after the embolization procedure. On the first occasion, he presented again with significant hemoptysis and was successfully treated with radiotherapy. On the second occasion, minor hemoptysis responded to antibiotic therapy. Ten weeks later, primary carcinoma of the esophagus was diagnosed and he was referred to the surgical department, which determined that the tumor was inoperable. An esophageal stent was inserted. After his third hospital admission for hemoptysis 15 months after embolotherapy, he underwent a second course of radiotherapy. He died as a result of the esophageal malignancy 7 months later.

Discussion

Hemoptysis may be a life-threatening condition with a propensity to recur if definitive therapy is not instituted. In a study by Knott-Craig et al., 36.4% of patients presenting with massive hemoptysis (defined as > 200 mL blood per 24 h) who underwent medical therapy had a recurrent episode of hemoptysis within 6 months of hospital discharge. Almost one half of these recurrent episodes (45%) proved fatal.

In situations in which pulmonary resection will eliminate the cause for the hemoptysis, this is the treatment of choice. Unlike other modalities, surgical resection is curative. In patients who are unsuitable surgical candidates, who refuse surgical treatment, or for whom appropriate medical therapy will ultimately lead to a cure, control of the hemorrhage may be achieved by various other options. These include cold saline solution lavage via bronchoscopy,7,8 endobronchial balloon tamponade with or without endobronchial instillation of epinephrine,9,10 instillation of thrombin or fibrinogen-thrombin infusions endobronchially via fiberoptic bronchoscopy,11 laser photocoagulation,12 radiotherapy,13 or bronchial artery embolization.14,15 Selection of the modality of treatment will be determined by the underlying etiology for the hemoptysis and the available expertise in the center to which the patient presents. All of these procedures should be regarded as temporizing measures before more definitive management. Unfortunately, these are the only options available for functionally compromised patients.

The etiology for massive hemoptysis in various parts of the world reflects the socioeconomic development of the geographic location.5 In South Africa, where the prevalence of tuberculosis is one of the highest in the world, most patients presenting with massive hemoptysis either have active tuberculosis or have the sequelae of this disease, such as bronchiectasis or colonization of old tuberculous cavities by Aspergillus. These patients frequently have bilateral disease and significantly compromised pulmonary function. Bronchial artery embolization has been demonstrated to be an effective means of managing these patients.4,16

Although deaths related to massive hemoptysis are most commonly caused by asphyxiation, acute loss of blood volume leading to shock may also occur. It has been shown that the bronchial circulation, rather than the pulmonary arterial circulation, is the usual source of bleeding from areas of acute and chronic lung inflammation and scarring. This vascular system can increase to more than one third of the systemic flow in chronic pulmonary disease.17

Bronchial artery anatomy varies greatly. In the majority of patients, the bronchial arteries arise directly from the aorta or from the intercostal arteries. Uflacker et al18 identified as many as 10 different patterns in a group of 72 patients who underwent bronchial artery embolization. However, bronchial arteries have also been described to arise from the subclavian, brachiocephalic, internal mammary, phrenic, and coronary arteries. The interventional radiologist performing embolotherapy needs to have a good working knowledge of these variants. Because catheterization of the bronchial and other arteries
requires expertise and skill, the success of this procedure is dependent on the experience of the radiologist.

The parietal pleura is supplied by the intercostal, internal mammary, and musculophrenic arteries, whereas the visceral pleura derives its arterial supply from the bronchial arteries.19 Patients who have pleural disease in association with parenchymal abnormalities are more likely to have nonbronchial systemic collateral vessels supplying the bleeding area. For this reason, it is prudent to actively exclude such vessels as contributors to the origin of hemoptysis in cases where there is associated pleural disease. Indeed, in three of our patients, bleeding from systemic vessels other than the bronchial arteries was the only source of hemoptysis. In 9 of the remaining 13 patients, these vessels provided a significant contribution toward the hemoptysis.

Embolization of the bronchial arteries alone may achieve control of bleeding in a significant number of patients. Because it is not always routine to then exclude additional nonbronchial systemic collateral vessels, their presence is probably frequently missed and their importance difficult to estimate. Nonbronchial systemic collateral vessels may arise from the axillary, subclavian, thyrocervical, lateral thoracic, internal mammary, and phrenic arteries, as well as from the chest wall.20–22 Brinson et al23 reported 18 patients with cystic fibrosis and hemoptysis who underwent a total of 36 bronchial artery embolization procedures. The incidence of hemorrhage from nonbronchial systemic collateral vessels was 75% in patients with recurrent bleeding but only 8% in patients who underwent their first embolization procedure. In the series published by Keller et al,24 45% of 20 patients with massive or recurrent hemoptysis had a significant blood supply from nonbronchial collateral vessels. Vujic et al25 described three patients in whom hemoptysis was controlled by embolization of the intercostal arteries only. In another study,26 the internal mammary artery was demonstrated to be an important collateral supply in 11 of 23 patients with hemoptysis. It has been shown that both short-term and long-term outcomes are less successful in patients with pleural abnormalities,21 presumably because it may be impossible to occlude all significant collateral vessels supplying the diseased area.

Complications from embolotherapy in patients with hemoptysis are uncommon but have been described. These include spinal cord syndromes,6,25,27,28 bronchial stenosis,29 bronchoesophageal fistula,30 infarction of a bronchus,31 mediastinal hemATOMA after subintimal aortic dissection,28 and transient cortical blindness.32 Some of these complications may be attributable to the use of older ionic contrast agents23 or the use of alcohol as an embolic agent,31 rather than inadvertent embolization of important vessels. The most dreaded consequence of bronchial artery embolization is inadvertent occlusion of spinal arteries. In a study of 75 patients,18 in no patient was the anterior spinal artery demonstrated to arise from a bronchial artery or an intercostobronchial trunk. Cohen et al34 found a spinal artery branching from a vessel that also supplied the bronchial artery circulation in as many as 55% of 20 patients with cystic fibrosis. Interestingly, all were branches of right-sided bronchial arteries or right-sided head and neck vessels.

Although many authors feel that identification of the anterior spinal artery or spinal radicular arteries arising from the bronchial arteries is an absolute contraindication to proceed with embolization, others do not.4,14,18,23 It is noteworthy that in instances in which spinal cord injury followed embolotherapy, branches to the spinal cord were not identified at angiography.6,25,27,28 It is possible that arteries to the spinal cord may become angiographically visible only once embolization of larger hypertrophied bronchial vessels, into which flow will be preferentially directed, is achieved. In our series, a spinal artery was visualized in only one patient (patient 5). Cowling and Belli35 have also warned of potential complications related to the passage of particles from the bronchial circulation to the subclavian artery or its branches via collateral vessels.

Unless the underlying disease giving rise to hemoptysis can be cured by appropriate medical therapy, embolotherapy should be regarded as an interim measure to stabilize the patient before definitive management is instituted. However, many of these patients are not fit for lung resection surgery. In the study by Uflacker et al,18 bronchial artery embolization was successful in treating hemoptysis in 72.7% of 33 patients who underwent this procedure as the sole form of treatment.

Approximately 20% of patients who undergo embolotherapy will develop recurrent hemoptysis, usually within 6 months.36,37 In one study28 of 43 patients who underwent bronchial artery embolization, 7 of these patients experienced a recurrence of hemoptysis within 30 days of undergoing the procedure. Rebleeding may be caused by incomplete occlusion of vessels feeding the area (including bleeding from the pulmonary arterial circulation, which accounts for < 10% of patients with massive hemoptysis).20 recanalization of previously embolized vessels, the development of new collaterals, or inadequate treatment of the underlying disease. Repeat embolization may be successful in those patients who rebleed and in whom surgical intervention is not feasible.37 Where subsequent pulmonary resection is an alternative, the timing of surgery is a
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