Control of Hemoptysis by Bronchial Artery Embolization*

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Control of massive hemoptysis by embolization of bronchial arteries was achieved in two patients with bronchopleural fistula. Both patients would have been prohibitive risks for thoracotomy. The indications, contraindications, and technique of the procedure are presented as well as a review of the literature.

Hemoptysis may present a life-threatening situation, especially in a patient who has chronic lung disease and is a poor candidate for pulmonary resection. While bronchoscopy followed by thoracotomy and pulmonary resection is the customary means of managing massive hemoptysis,1,2 other methods of controlling hemorrhage must be employed in the face of advanced pulmonary insufficiency or inability to localize the site of bleeding by bronchoscopy.3 The mortality rate in patients who are incapable of tolerating surgical procedure is 80 percent.4

Catheterization and arterial embolization have been used successfully to arrest massive and recurrent hemoptysis.5,6,7 In the cases under consideration, bronchial and intercostal artery embolization was employed for control of massive hemoptysis complicating two cases of bronchopleural fistula.

METHOD

Prior to the embolization procedure, it is essential to determine the site of bleeding. This is usually accomplished by bronchoscopy. In one of our patients, the site was determined by obvious bleeding through the thoracotomy tube.

The angiograms obtained by the femoral route with appropriately preshaped catheters of F5-F6 size should be of excellent quality in order to visualize important vessels supplying the spinal cord. If the spinal artery is not present, the catheter tip is selectively advanced distally and small pieces of gelfoam (2 x 2 mm) are introduced into the artery. At the very beginning, they are injected, but as soon as the flow becomes sluggish, we prefer to place the material into the catheter and push it into the vessel by advancing the guide wire. At the end of our procedure, we obtain the postemboli-

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CASE REPORTS

CASE 1

A 72-year-old white man with chronic obstructive pulmonary disease developed an abscess involving the superior segment of the lower lobe. This became an empyema necessitating tube thoracotomy for adequate drainage.

Over the next two and one-half year period, the patient’s condition remained reasonably stable with chest drainage until the development of massive hemoptysis associated with bloody purulent discharge from the thoracotomy tube. Injection of propyliodone (Dionosil) into the thoracotomy tube revealed bronchopleural fistula. Because of markedly depressed pulmonary function, the left bronchial artery was catheterized and an angiogram obtained showing markedly increased vascularity in the area of the cavity (Fig 1A [upper]). The control of hemorrhage was achieved by embolizing small particles of absorbable gelatin sponge (Gelfoam) into the involved bronchial artery (Fig 1B [lower]). Cultures from the chest tube at the time of hemoptysis were positive for *Mycobacterium avium*, *Proteus mirabilis*, and *Escherichia coli*. In addition to cefalosporins, the patient was placed on a regimen of rifampin and ethambutol for which the tuberculous organism was later shown to be resistant. The patient stopped all medication including antituberculosis drugs two months after embolization because of a sense of well-being. The purulent drainage and air leak diminished. Six months after embolization, a bronchogram performed through the chest tube showed virtual obliteration of the cavity, and the chest tube was removed. The patient was doing well without any drugs at the time of this writing two years following embolization.

CASE 2

A 63-year-old white man underwent right upper lobectomy and chest wall resection for bronchogenic carcinoma. This was followed by radiation therapy. On a routine chest x-ray film 14 months after operation, the patient was found to have an apical bronchopleural fistula on the right with an air fluid level.

One month after this x-ray film, he developed massive hemoptysis. The site of hemorrhage and air leak were located by flexible bronchoscopy at the right upper lobe bronchial stump. Arteriography was performed. A bronchial artery on the right was not located, but injection of a right intercostal artery showed irregular vessels, multiple collaterals, and a point of hemorrhage in the area of the right upper lobe bronchial stump (Fig 2A [upper]). Embolization was carried out with small particles of Gelfoam (Fig 2B [lower]). Hemoptysis immediately stopped. There was adequate dependent drainage from the bronchopleural fistula. The patient was asymptomatic and at work at the time of this writing six months later.

DISCUSSION

Since Remy and associates introduced the embolization of bronchial arteries in 1974, there have been several reports in the world literature including one by Remy et al in 1977 reviewing their experience with 104 cases. Recently advocated endobronchial tamponade therapy with Fogarty balloon for intractable hemoptysis would not be an applicable procedure in our patients because of free bronchopleural fistula.

The indication for embolization in all cases has been massive or recurrent hemoptysis with tuberculosis, bronchiectasis, aspergilloma, and pneumoconiosis being underlying disease in a great majority of patients. The procedure is a palliative one with the main goal to arrest the hemoptysis with the absence of relapse during the following months. The long term success depends, however, on efficacy of medical treatment because even with complete occlusion of the bleeding artery, the possibility of recanalization or the development of collateral blood supply to the lesion may occur and cause the relapse.

Complications have included paraplegia, severe respiratory infection, esophageobronchial fistula, and ischemic colitis. Paraplegia has resulted from the presence of spinal artery branching from the bronchial artery undergoing embolization. Paraplegia was also a result of the simple angiography of the right fifth
intercostal artery.\(^9\)

Massive hemoptysis has been controlled by surgical ligation of bronchial arteries without pulmonary resection in a few reported instances.\(^6\)\(^-\)\(^8\)

To avoid complications, the catheter should be advanced selectively into the bleeding vessel and Gelfoam particles injected very slowly in order to prevent inadvertent embolization of other organs. The presence of a major spinal artery is an absolute contraindication for an embolization procedure.\(^6\)

In the largest reported series, successful control of hemoptysis by embolization procedure without relapse within two months was achieved in approximately 90 percent of patients with the exception of those with aspergillosis.

The possibility of controlling hemoptysis by bronchial artery ligation with the aid of preoperative arteriograms may be worth consideration, especially in the patient with advanced pulmonary insufficiency who is incapable of tolerating pulmonary resection.

The bronchial circulation becomes hyperplastic with an inflammatory process in the lungs and subsides to a normal state when the process is brought under control.\(^6\) Whether interruption of a hyperplastic circulation alters the course of an inflammatory process or the milieu for growth of tuberculosis organisms is a matter of speculation. In our case 1 and in two other cases of tuberculosis in the literature,\(^5\)\(^10\) the course of the patients following interruption of the bronchial circulation was one of rapid recovery.

**References**


**Late, Late Doxorubicin Cardiotoxicity**

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**Cardiac toxicity is a major complication which limits the use of Adriamycin as a chemotherapeutic agent. Cardiomyopathy is frequent when the total dose exceeds 600 mg/m² and occurs within one to six months after cessation of therapy. A patient is reported who developed progressive cardiomyopathy two and one-half years after receiving 580 mg/m² which apparently represents late, late cardiotoxicity.**

Cardiac toxicity has been the major factor limiting the use of doxorubicin hydrochloride (Adriamycin) as an effective antineoplastic agent.\(^1\) Cardiovascular effects of doxorubicin are manifested by acute, transient and usually benign arrhythmias and by a late dose-dependent cardiomyopathy.\(^2\) The incidence of cardiomyopathy is greater than 30 percent among patients who receive a total dose of more than 600 mg/sq m and usually occurs within one to six months after completion of therapy.\(^3\)\(^,\)\(^4\) In this report, we describe a patient with progressive heart failure 2½ years following completion of doxorubicin chemotherapy. We believe this represents a case of late, late doxorubicin cardiotoxicity.

**Case Report**

A 48-year-old white woman was hospitalized for progressive biventricular failure and eventually died from the severe low output state. Breast cancer had been diagnosed 16 years previously, and she was treated with surgery and nonmediastinal radiotherapy. Recurrence two years later was treated with excision, local radiotherapy, and oophorectomy. A right axillary ulcer was noted ten years later, and chemotherapy, consisting of a six-month course of cyclophosphamide (Cytoxan), methotrexate, and 5-fluorouracil, was given. Because of bone metastasis, doxorubicin alone was then given at three-week intervals for six months. A total dose of 580 mg/sq m was given. Upon completion of doxorubicin therapy, an infusion of 30 mg/kg/day of 5-fluorouracil was given for five days at monthly intervals for a total of 16 months. Finally, 400 mg of megestrol acetate (Megace) was given four times a day for eight months. The entire course of doxorubicin therapy was completed three years prior to the hospitalization for congestive heart failure (Table 1). The symptoms of congestive heart failure began six months prior to the final hospitalization. Serial chest x-ray films to follow the course of her malignancy had not shown cardiomegaly until the time of onset of symptoms. The patient had not been hypertensive.

Physical examination at the time of the last admission revealed a cachectic, dyspneic, white woman with evidence of severe biventricular failure. She had blood pressure of 90/