

**CRYPTOGENIC HEMOPTYSIS IN SMOKERS: ANGIOGRAPHY AND RESULTS OF  
EMBOLIZATION IN 35 PATIENTS**

**CRYPTOGENIC HEMOPTYSIS IN SMOKERS: ANGIOGRAPHY**

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## **ABSTRACT :**

**Objectives:** To describe the angiographic findings and embolization results in smokers with hemoptysis.

**Methods:** We retrospectively reviewed the clinical data and angiographic findings of 35 patients with smoking-related bronchopulmonary disease and no associated comorbidity, referred for embolisation for mild (n=6), moderate (n=14) and severe (n=15) hemoptysis. Spirometric classification subdivided our population in: (a) 16 patients with chronic bronchitis but no airflow limitation; (b) 19 patients with COPD (stage I: n=12; stage II: n=5; stage III: n=2).

**Results:** Bronchoscopy depicted focal submucosal vascular abnormalities in 3 patients and only endobronchial inflammation in 32 (91%) patients. Bronchial artery angiography revealed moderate (n=18) or severe (n=10) hypervascularisation in 28 patients (80%), and normal vascularization in 7 (20%). No statistically significant difference was observed between the angiographic findings and the severity of COPD, tobacco consumption or the amount of bleeding. Cessation of bleeding was obtained by embolisation in 29 out of the 34 technically successful procedures (85%), requiring surgery in 3 out of 5 patients with recurrence. Follow-up (mean duration: 7 years) demonstrated no recurrence of bleeding in 32 patients (32/34; 94%) and excluded late endobronchial malignancy.

**Conclusions:** Smokers with various stages of COPD severity may suffer from hemoptysis, efficiently treatable by endovascular treatment.

## **INTRODUCTION:**

Hemoptysis is a frequent symptom in pneumology and requires rapid and careful investigation. The changing spectrum of causes of hemoptysis was first acknowledged in the late eighties by the study of Johnston and Reisz [1]. Comparing their data with older studies, they underlined that hemoptysis was less likely to be caused by tuberculosis and bronchiectasis, while hemoptysis caused by bronchitis had increased proportionately, subsequently confirmed in the literature [1-6]. The second striking feature regarding the revised prevalence of the etiologies of hemoptysis is that, despite modern diagnostic means, nearly a third of hemoptysis remains classified as cryptogenic [1, 3, 7-10]. However, the populations reported in this category are quite heterogeneous, gathering patients with no identifiable causes of hemoptysis, but also patients in whom an underlying disease was finally recognized such as “inactive tuberculosis” or “occult bronchiectasis”. This apparent contradiction can be explained by the various criteria retained by the authors to assess this diagnosis. If fiberoptic bronchoscopy was systematically included [1, 3, 7-10], patients’ follow-up and imaging studies, in particular chest CT, have been only integrated in the most recent reports on cryptogenic hemoptysis [3, 6, 9, 10].

When no associated comorbidity can be confidently excluded, the subgroup of cryptogenic hemoptysis shares a common risk factor with patients in whom hemoptysis is explained by chronic bronchitis, namely the patient’s tobacco consumption. Careful reading of studies on cryptogenic hemoptysis in which detailed patient’s history is available reveals a high proportion of smokers, varying between 42% and 79% of the studied populations [2, 8-10]. However, these authors did not discuss the causal relationship between cryptogenic hemoptysis and smoking as did Hiyama et al in 2002 [11]. In our experience of a referral center for embolization of hemoptysis, we encountered similar situations in which the patient’s smoking history was the only potential cause for bronchial bleeding after a thorough search for an underlying disease. The purpose of this

study was to report the angiographic aspects of the bronchial circulation and the long-term results of bronchial artery embolization in this subset of patients with various stages of smoking-related disease and no other comorbidity.

## **MATERIALS AND METHODS:**

### **1- Study population**

The study population was selected from the data base of 318 consecutive patients who had been referred to the Department of Radiology for endovascular treatment of hemoptysis over a period of 22 years (November 1983 - February 2006). The criteria for selecting this population included: (a) the absence of any comorbidity assessed by fiberoptic endoscopic examinations and imaging studies at the time of admission; (b) clinical symptoms of chronic bronchitis in a context of smoking-history; (c) no late diagnosis of bronchial carcinoma confirmed by patients' follow-up. Thirty-five (35/318; 11%) fulfilled these criteria. None of these patients showed clinical and/or biological features of concomitant infection at the time of their referral for hemoptysis. Except for two patients for whom the information was not retrospectively accessible, our population did not received aspirin or coumadin treatment when evaluated for bronchial bleeding.

### **2- Fiberoptic bronchoscopy**

Fiberoptic bronchoscopy was performed within 48 hours after admission in cases of mild to moderate hemoptysis or as an emergency in cases of massive hemoptysis. The bronchoscopic report stated on (1) the presence of active bleeding, (2) the site of bleeding, and whenever possible the lobar origin of bleeding, and (3) the presence of endobronchial abnormality. Endobronchial and transbronchial biopsies were performed when indicated, and all specimens were routinely examined for cytology and microbiology.

### **3- Imaging studies**

At the time of admission, each patient underwent a radiographic examination of the chest. To exclude an underlying lesion potentially responsible for bronchial bleeding, patients underwent additional imaging studies, consisting of conventional bronchography (n=6) and/or tomograms (n=4) in the 9 patients (26%) referred prior to the advent of CT, and chest CT in the remaining 26 patients (74%). Depending on the CT technology available, high-resolution chest CT was obtained using a single- (n=12), 4- (n=5), 16- (n=4) and 64- (n=5) slice CT scanner. Initial and follow-up CT scans were reviewed to exclude any specific anatomic lesion as the source of bleeding and to describe the presence of alveolar infiltration and/or endobronchial nodules, reflecting the filling of the alveolar spaces and airways with blood, respectively. No attempt was made to describe the CT appearance of bronchial arteries in our study group because CT was not always available (9/35 studied patients) or consisted of a noncontrast CT scan (13/26 patients with CT).

### **4- Angiography**

In all patients, conventional angiography, with a Seldinger technique, was performed with a conventional, then digital subtraction technique within one week after admission (range: 1-7 days). Management of hemoptysis followed the general recommendation according to which bronchial arteries ipsilateral to the side of bronchial bleeding should be embolized first (**12**). Before the embolization procedure, a bronchial angiogram was systematically performed with manual injection of contrast medium in the catheterized vessel, enabling analysis of the diameter of bronchial artery (ies) and depiction of angiographic features of hypervascularization on the side of bleeding. The diameter of bronchial arteries was coded as normal when less than 2 mm, moderately enlarged when the diameter was between 2 and 4 mm and severely dilated when the diameter was more than 4 mm. The additional angiographic features of hypervascularization included the

depiction of: (a) tortuous enlargement of bronchial artery (ies) that supplied an area of parenchymal staining; and (b) a shunt into pulmonary vessels. Bronchial vascularization was coded as normal, moderately or markedly increased. Endobronchial bleeding occurring during the embolization procedure was systematically recorded. The angiographic appearance of contralateral bronchial arteries was available in two conditions: (a) when the side of bleeding was supplied by a common trunk for the right and left bronchial arteries; and (b) when contralateral bronchial arteries were incidentally catheterized during the angiographic procedure. When contralateral bronchial angiograms were available, the diameter of the bronchial artery(ies) and the presence of angiographic features of hypervascularization were coded using the same criteria as those used for the analysis on the side of bleeding.

Persistent hemoptysis after a technically successful procedure required to verify the status of previously embolized vessel(s): (a) in case of partial recanalization, the artery(ies) was (were) again embolized; (b) in case of persistent adequate obstruction, then recurrence of bleeding constituted an indication for evaluation of nonbronchial systemic arteries on the side of bleeding. This procedure was always considered at second intention because of the potential neurologic iatrogenic risks when embolizing these vessels and the lack of constant relationship between arterial hypervascularity and bronchial bleeding. The immediate and long-term results of embolization were systematically recorded.

## **5- Imaging study interpretation**

The imaging studies undertaken at the time of the initial management were reviewed by two radiologists (LM; MRJ), with 5 and 20 years of experience in thoracic imaging respectively, both unaware of the patient's clinical condition at the time of their reading. When initial and follow-up CT scans were available, they were analyzed consecutively by the two readers to assess the outcome of CT findings.

## **6- Statistical analysis**

Statistical analysis was performed with a commercially available software (SAS Institute, Cary, N.C. 25513). Results were expressed as means, standard deviations, and ranges for continuous variables and as frequencies and percentages for categorical variables. Comparative analyses were obtained using the chi-square or the Fisher exact test for categorical data. For numerical variables, group comparisons were performed using either the student t-test (if the sample was larger than 30) or the U-Mann Whitney test (if the sample was smaller than 30). A p value lower than 0.05 was considered to be statistically significant.

## **RESULTS:**

### **1- Characteristics of the population studied:**

#### **a- Clinical data:**

The investigated cohort included 33 males and 2 females (mean age: 57.08 +/- 10.6 years; range: 34-76) and a mean cigarette consumption of 33.4 pack-years (+/-14.54) (range: 12 – 80). The absence of underlying anatomic lesion as the source of bleeding was assessed in all patients by the review of imaging findings (chest films and CT scans: n= 26 patients, 74%; chest films, conventional bronchography and/or tomograms in 9 patients, 26%). The mean follow-up was 7.01 years (+/-6.8; range: 1.6-26.8 yr). The absence of underlying lung cancer was assessed on the basis of: (a) an uneventful mean follow-up period of 9.72 years in the 9 patients who did not undergo chest CT at the time of initial management; (b) an uneventful mean follow-up period of 6.07 years in 26 patients who underwent chest CT at the time of initial management; among them, 24 patients were followed-up by fiberoptic bronchoscopy and chest CT which did not reveal any tumoral lesion.



Applying the revised criteria of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) strategy (13), our population included: (a) 16 patients with symptoms of chronic bronchitis but no airflow limitation (i.e., healthy smokers); (b) 19 patients with a chronic obstructive pulmonary disease (COPD) (stage I: n=12; stage II: n=5; stage III: n=2). The amount of hemoptysis was mild (<100 ml/day; n=6), moderate (100-200 ml/day; n=14), and massive (> 200 ml/day; n= 15). In all patients, bronchial embolization was indicated because of the amount and/or recurrence of bleeding despite medical treatment. **Table 1** summarizes the amount of bleeding in the 4 categories of patients classified according to the spirometric data. For the purpose of statistical analysis, we gathered the 5 patients with a COPD stage II and the 2 patients with a COPD stage III into the same subgroup; no statistically significant difference was observed in the distribution of the amount of bleeding between the 3 groups of smokers (p=0.74). For 21 patients, it was the first episode of hemoptysis, while 14 patients had already been admitted to our hospital for the treatment of mild (n=9), moderate (n=4) and severe (n=1) bleeding, all stopped successfully with medical treatment.

#### **b- Fiberoptic bronchoscopic findings**

Active bleeding at the time of the endoscopic procedure or the focal location of blood clots led to the identification of the site of bronchial bleeding during fiberoptic bronchoscopy in all patients. The sites of bronchial bleeding were the upper lobes (25/35; 71%), with a right-sided predominance of upper lobe bleeding (18/25; 72%), the right middle lobe and lingula (2/35; 6%) and the lower lobes (8/35; 23%). Fiberoptic bronchoscopy depicted the presence of mucosal inflammation in 32 patients (91%), localized to the area of bleeding in 20 patients (20/32; 62.5%) or diffusely observed in the bronchial tree in 12 patients (12/32; 37.5%). In 3 patients (9%), fiberoptic bronchoscopy revealed the presence of submucosal vascular abnormalities in the area of bronchial

bleeding described as nonpulsating submucosal vascular nodules (2 patients) or dilated submucosal vessels (1 patient). In the overall population, no additional endobronchial abnormality, and in particular, no endobronchial malignancy was found during the endoscopic examination.

### **c- Chest X-ray and CT findings**

Chest X-rays were normal in 17 patients (54%) and abnormal in 16 patients (46%), showing alveolar infiltrates in 12 patients and emphysematous changes in 4 patients. Among the 26 patients who underwent chest CT at the time of referral for hemoptysis, CT was normal in 2 patients and depicted features of alveolar and/or endobronchial bleeding in 24 patients, which resolved on follow-up CT scans in all patients. An endobronchial nodule was found on the initial CT scan in 12 patients which resolved on the follow-up examination in all patients.

## **2- Angiographic findings**

Whereas bronchial artery catheterization was technically successful in 34 patients, it failed in 1 patient owing to the ectopic origin of the right bronchial artery, found to originate from the concavity of the aortic arch on the aortogram obtained at the end of the angiographic procedure. The angiographic findings on the side of bronchial bleeding (23 on the right side; 12 on the left side) were available from selective arteriograms in 34 patients and from an aortogram in 1 patient.

On the side of bronchial bleeding, a total of 43 bronchial arteries were depicted in the 35 patients, including 16 intercostobronchial trunks, 14 common trunks from the right and left bronchial arteries and 13 isolated bronchial arteries. Twenty-nine patients (83%) showed abnormally dilated bronchial arteries, rated as moderately dilated in 19 patients (**Figure 1**) and markedly dilated in 10 patients (**Figure 2**); 6 patients (17%) had

angiographically normal bronchial arteries on the side of bronchial bleeding (**Figure 3**). Enlarged bronchial arteries were seen with the concurrent presence of bronchial artery tortuosity in 15 patients and/or parenchymal staining with retrograde bronchial-to-pulmonary shunting in 28 patients. Angiographic features of hypervascularization, present in 28 patients (80%) were rated as moderate (n=18), and marked (n=10). Endobronchial bleeding during the embolization procedure was observed in 6 patients.

In 25 patients, bronchial artery angiography was also obtained on the controlateral side, enabling us to compare the morphological characteristics of bronchial arteries on the ipsilateral and controlateral side of bleeding (**Table 2**). In 19 patients (19/25; 76%), the angiographic findings were symmetrical; in 6 patients (6/25; 24%), abnormalities were more severe on the side of bronchial bleeding.

**Table 3** summarizes the angiographic findings in our cohort of patients according to the spirometric data. There was no statistically significant difference in the distribution of bronchial artery diameters (Fisher's exact test;  $p=0.27$ ) and degree of hypervascularization (Fisher's exact test;  $p=0.18$ ) according to the pulmonary functional test results. No statistically significant difference was observed between the angiographic findings and the amount of hemoptysis ( $p=1$ ) (**Table 4**) nor between the angiographic findings and the tobacco consumption of patients ( $p=0.34$ ). Among the 3 patients in whom fiberoptic bronchoscopy had revealed endobronchial vascular abnormalities, the bronchial arteriogram was normal in 1 patient and showed moderate bronchial hypervascularization in 2 patients.

### **3- Results of the embolization procedure**

Embolization was technically successful in 34 patients and failed in one patient owing to the impossibility to catheterize a bronchial artery with an ectopic origin. In this latter case, bronchial bleeding stopped under medical treatment. Early results of embolization in

34 patients were classified as follows: (a) immediate cessation of bleeding was achieved in 29 patients (29/34; 85%); (b) “immediate recurrence” of hemoptysis (within 48 hours) was observed in 5 patients (5/34; 15%). In these 5 patients, cessation of bleeding was obtained after a second embolization procedure of previously occluded bronchial arteries. (a) “early recurrence” of bleeding (within 3 months after endovascular treatment) was observed in 5 patients (5/34; 15%); (b) bleeding cessation was obtained by medical treatment alone (n=2) or bronchial artery embolization followed by surgery (n=3) because of hemoptysis recurrence. Surgery consisted of a right upper (n=1), left upper (n=1) and right middle (n=1) lobectomy. Two patients (2/34; 6%) presented a late recurrence of hemoptysis, 2 and 12 years after the initial embolization procedure, respectively, successfully treated by medical treatment. From the initial study group, a total of 23 patients had no recurrence of bronchial bleeding. No statistically significant relationship was found between the recurrence of bleeding and the severity of the underlying smoking-related bronchopulmonary disease and the angiographic aspects at the time of the embolization procedure (**Table 5**).

Two of the 3 patients with submucosal vascular abnormalities were treated by embolization with a follow-up endoscopic examination within 48 hours after the endovascular procedure. Fiberoptic bronchoscopy showed a complete (n=1) or partial (n=1) resolution of the endobronchial abnormality; in the latter case, a biopsy of the right upper lobe bronchus was obtained after the embolization procedure, responsible for mild hemoptysis.

#### **4- Pathologic findings**

Pathologic data were available in 4 patients, obtained after biopsy (n=1) or surgery (n=3). The biopsy specimens, obtained after right upper (n=1), left upper (n=1) and right middle (n=1) lobectomy, showed dilated and numerous capillary vessels in the submucosal

region of the right upper lobe bronchus. Pathologic analysis revealed the presence of a submucosal angiomatous lesion at the level of the right middle lobe bronchus (n=1), dilated vessels within the mucosa of the right upper lobe bronchial walls (**Figure 4**), while bronchial wall vessels of the left upper lobe were described as normal in one patient. Apart from smoker's lung lesions and focal filling of alveoli by blood observed in all 3 patients, no associated abnormalities were described in the pathologist's reports.

## **DISCUSSION**

To our knowledge, this is the first study describing the angiographic appearance of bronchial arteries in patients referred for smoking-related hemoptysis. This exclusive diagnosis was assessed by the absence of objective evidence of thoracic disease at the time of examination and no late diagnosis of lung cancer, the clinician's fear when hemoptysis remains of unknown origin (**9**). Although strictly limited to a population referred for endovascular treatment of hemoptysis over a 22-year period, the proportion of smoking-related hemoptysis observed in this population, i.e., 11%, is comparable to those previously reported in the two studies describing cases of hemoptysis in smokers with no other comorbidity and with a detailed history of the patients' smoking history [**10**, **11**]. Over a 5-year period, Hiyama et al observed 51 hospitalized patients with hemoptysis among whom 6 (12%), all smokers, were considered with cryptogenic hemoptysis according to CT and fiberoptic bronchoscopy findings [**11**]. More recently, Savale et al have reported a cohort of 81 patients with cryptogenic hemoptysis, diagnosed from a population of 653 consecutive patients (81/653; 13%) who had been admitted to the respiratory intermediate care ward and intensive care unit for hemoptysis [**11**].

The hallmarks of our population are fourfold. Firstly, there was a male predominance in our cohort of patients, presenting with moderate or massive hemoptysis in 83% of cases (29/35 patients). This finding suggests that smoking-induced inflammatory changes can be

responsible for life-threatening hemoptysis, a situation not previously emphasized especially in smokers with no airflow limitation. In this latter group, usually referred to as “healthy smokers”, it is noticeable that 87.5% of them (14 of 16 patients) were referred for moderate or massive hemoptysis. Secondly, the most frequent sites of bronchial bleeding were the upper lobes (25/35; 71%) with a predominance of right-sided bronchial bleeding (18/25; 72%). These predominant sites of bronchial bleeding in smokers are not unexpected when they are analyzed in the light of the current knowledge of the target areas of tobacco smoke. Whereas the distribution of inflammatory changes in the bronchial tree of smokers remains unknown, there is a well-known predominant or exclusive distribution of smoking-induced inflammatory changes in the upper lung zones [14, 15]. Thirdly, moderate to marked bronchial hypervascularization was found in 80% (28/35 patients) of our study group. When bilateral angiograms were available, the angiographic appearance of bronchial arteries was symmetrical in 76% of cases (19/25 patients). These results suggest the presence of underlying bronchial hyperemia which was confirmed by the pathologic findings available in 4 of our patients. Hypervascularization within the bronchial wall remains an ill-defined process in the pathogenesis of asthma and airway chronic inflammation such as COPD [16, 17]. As previously underlined by several authors, there is a remarkable proliferative capacity of the bronchial vessels in response to a variety of pulmonary diseases [18, 19]. In a recent study, Hashimoto et al reported that submucosal vascularity in patients with COPD was greater than in control subjects, but not significantly, except for the percentage of vascularity in the small airways [20]. These data are consistent with the results of previous studies [21]. The vessel increase in COPD patients might have occurred as the result of simple tobacco-induced inflammation but angiogenic factors might also be secreted as an asthma-specific inflammation. In a minority of patients, more pronounced vascular lesions were observed in the site of bleeding, as suggested by the angiographic findings of more severe hypervascularization in 6 patients

with bilateral angiograms. In the context of angiogenesis in chronic bronchitis, one may also reconsider the endobronchial vascular lesions depicted in 9% of our study group (3/35 patients). Whereas they resemble the endobronchial findings described in Dieulafoy's disease, one may also raise the hypothesis that such lesions could be acquired. Among the 12 cases of Dieulafoy's disease reported in the literature [22-30], 5 cases were described in smokers who had no associated co-morbidity.

The fourth hallmark of our study group is the lack of relationship between the angiographic findings and the risk of bronchial bleeding and the severity of the underlying bronchopulmonary disease. Whereas most patients had various degrees of bronchial hypervascularization, 20% of our population (7/35 patients) had angiographically normal bronchial arteries on the side of bronchial bleeding. The immediate cessation of bleeding after bronchial embolization confirms the responsibility of angiographically normal bronchial arteries in such cases. With regard to the efficacy of embolization in the overall population, an immediate cessation of bleeding was obtained in 85% of cases (25/34 patients) which is in the range of the immediate control of bleeding reported in the literature in a variety of underlying diseases [31-34]. An early recurrence of bleeding was observed in 5 patients (5/34; 15%) while only 2 patients had a late recurrence of bleeding (3/34; 6%). As in many varieties of chronic disorders, this is likely to reflect the recruitment of blood supply and revascularization by the underlying bronchial inflammation or progression of the underlying disease [34]. Classifying our cohort of smokers according to the revised spirometric classification recently proposed in the GOLD strategy, we failed to demonstrate any statistically significant relationship between the angiographic findings and the COPD severity which is in agreement with previous reports. In a microradiographic study of 17 patients with emphysema and chronic bronchitis, Turner-Warwick had observed that bronchial artery enlargement was not clearly related to the presence of associated bronchitis [35]. In a more recent study [21], Kuwano et al found

no correlation between the degree of vascularity and airflow limitation in patients with COPD. Moreover, we did not find any relationship between the risk of bronchial bleeding recurrence and the angiographic aspect of bronchial arteries at the time of the embolization procedure.

Several limitations have to be acknowledged in this study. The criteria for excluding any specific anatomic lesion potentially responsible for hemoptysis did not include chest CT in 9 patients who were referred prior to the advent of CT technology. However, these patients had been investigated by conventional bronchography and/or pulmonary tomography which did not reveal any abnormality. In addition, the uneventful mean follow-up of 9.72 years reported for these 9 patients reinforces the diagnosis of cryptogenic hemoptysis considered at the time of initial management. The second limitation concerns the exclusion of lung cancer in the 10 patients with a follow-up of less than 2 years. It is important to underline that these patients correspond to the most recently referred to our institution; subsequently, they benefited from fiberoptic bronchoscopy and chest CT not only at the time of diagnosis but also in their follow-up. The absence of endobronchial abnormality and normal chest CT scans in their follow-up reinforce the likelihood of cryptogenic hemoptysis. Lastly, one should underline two additional limitations, dealing with the lack of control group and the length of the inclusion period. Concerning the control group, we did not encounter more than a few cases of nonsmokers presenting with cryptogenic hemoptysis over the inclusion period which precluded any structured comparison with the studied population. The length of the inclusion period, i.e. 22 years, was related to the relative rarity of the target population which shared three similar criteria, namely the symptoms, the underlying cause and the therapeutic option.

In conclusion, our study demonstrates that tobacco smoke and its bronchopulmonary inflammatory consequences do represent an etiology for bronchial bleeding, independent from the severity of the underlying disease.



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**Table 1: Amount of bronchial bleeding according to the spirometric classification of the 35 patients**

|   | <b>Mild<br/>hemoptysis<br/>(n=6)</b> | <b>Moderate<br/>hemoptysis<br/>(n=14)</b> | <b>Massive<br/>hemoptysis<br/>(n=15)</b> |
|---|--------------------------------------|---|--|
| Smokers with no airflow limitation n=16       | 2                                    | 8   | 6  |
| Smokers with “mild” COPD n=12                 | 3                                    | 3   | 6  |
| Smokers with “moderate”, or “severe” COPD n=7 | 1                                    | 3   | 3  |

**Abbreviation:** COPD: chronic obstructive pulmonary disease

**Table 2: Comparison of angiographic findings in the 25 patients who underwent bilateral angiograms**

| <b>Angiographic aspect of BAs on the side of bronchial bleeding</b><br>25 patients | <b>Angiographic aspects of BAs on the controlateral side</b><br>25 patients                                       |
|--|---|
| 11 patients with moderately dilated Bas  | - 8 patients with moderately dilated BAs<br>- 3 patients with normal BAs  |
| 9 patients with severely dilated Bas   | - 6 patients with severely dilated BAs<br>- 2 patients with moderately dilated Bas<br>- 1 patient with normal BAs |
| 5 patients with normal BAs   | - 5 patients with normal Bas  |

**Abbreviations:** BA: bronchial artery

**Table 3: Angiographic findings according to the spirometric severity of COPD**

|   | Diameter of bronchial arteries |                                      |                                | Degree of bronchial hypervascularization |  |  |
|---|--------------------------------|--------------------------------------|--------------------------------|--|--|--|
|   | Normal BAs<br>n=7              | Moderately<br>dilated<br>BAs<br>n=19 | Severely dilated<br>BA<br>n=10 | No<br>hypervascularizat<br>ion<br>n=7    | Moderate<br>hypervasculariza<br>tion<br>n=18 | Marked<br>hypervascularization<br>n=10 |
| Smokers<br>with no<br>airflow<br>limitation<br>n=16   | 3<br>(19%)                     | 5<br>(37%)                           | 7<br>(44%)                     | 4<br>(25%)                               | 5<br>(31%)                                   | 7<br>(44%)                             |
| Smokers with<br>“mild” COPD<br>n=12                   | 2<br>(16%)                     | 9<br>(74%)                           | 1<br>(8%)                      | 2<br>(16%)                               | 9<br>(74%)                                   | 1<br>(8%)                              |
| Smokers with<br>“moderate” or<br>“severe” COPD<br>n=7 | 1<br>(14%)                     | 4<br>(58%)                           | 2<br>(28%)                     | 1<br>(14%)                               | 4<br>(58%)                                   | 2<br>(28%)                             |

**Abbreviation:** COPD: chronic obstructive pulmonary disease

BA: bronchial artery

**Table 4: Amount of hemoptysis in the categories of angiographic findings.**

|                               | Normal Bronchial<br>arteries<br>(n=7) | Moderate bronchial<br>hypervascularization<br>(n=18) | Marked bronchial<br>hypervascularization<br>(n=10) |
|-------------------------------|---------------------------------------|--|--|
| Mild hemoptysis<br>(n=6)      | 1                                     | 3  | 2  |
| Moderate hemoptysis<br>(n=14) | 3                                     | 7  | 4  |
| Massive hemoptysis<br>(n=15)  | 3                                     | 8  | 4  |

**Table 5: Outcome of patients after embolization according to the spirometric classification of COPD severity and angiographic findings**



|                       |  | <b>No<br/>recurrence</b><br>23 patients | <b>Immediate<br/>recurrence</b><br>5 patients | <b>Early<br/>recurrence</b><br>5 patients | <b>Late<br/>recurrence</b><br>2 patients |
|-----------------------|--|---|---|---|--|
| <b>PFT results</b>    | Smokers with no airflow limitation (n=16)      | 11                                      | 3   | 2   | 0  |
|                       | Smokers with “mild” COPD (n=12)                | 8                                       | 2   | 1   | 1  |
|                       | Smokers with “moderate” or “severe” COPD (n=7) | 4                                       | 0   | 2   | 1  |
| <b>Diameter of BA</b> | Normal BAs (n=7)                               | 6                                       | 0   | 1   | 0  |
|                       | Moderately dilated BAs (n=18)                  | 11                                      | 3   | 3   | 1  |
|                       | Severely dilated BAs (n=10)                    | 6                                       | 2   | 1   | 1  |

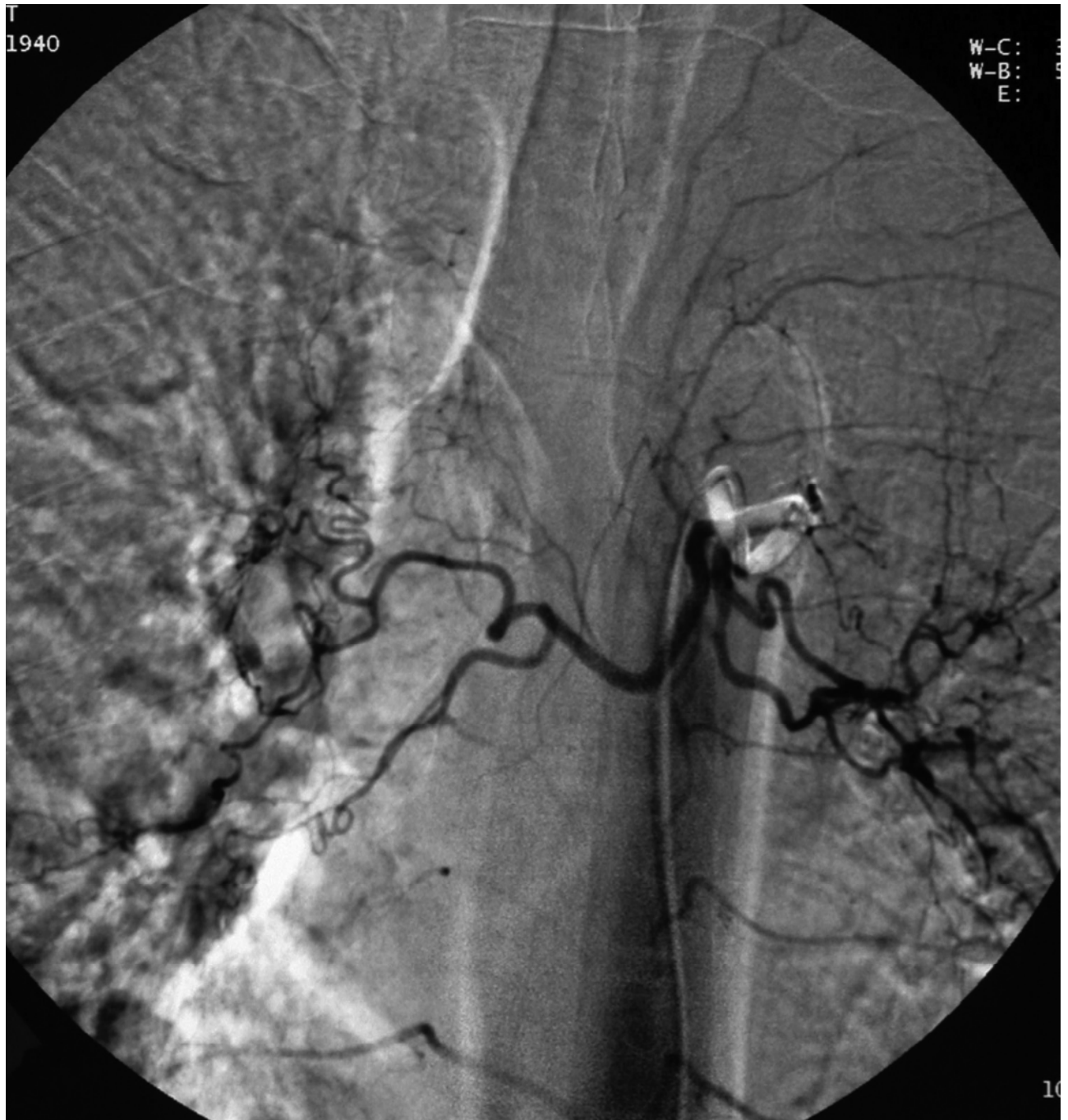
**Abbreviation:** COPD: chronic obstructive pulmonary disease

\ PFT: pulmonary function test

BA: bronchial artery

**FIGURE TEXT**

**Figure 1:** 58 year-old male with a cigarette consumption of 12 pack-years and no airflow limitation on pulmonary function tests. This patient was referred for management of moderate hemoptysis originating from the mediobasal segment of the right lower lobe. Selective catheterization of a common trunk for the right and left bronchial arteries showing moderately dilated bronchial arteries on both sides. Embolization of this artery led to an immediate cessation of hemoptysis which recurred 1 month later; a second embolization procedure enabled definitive cessation of bronchial bleeding (9-year follow-up).



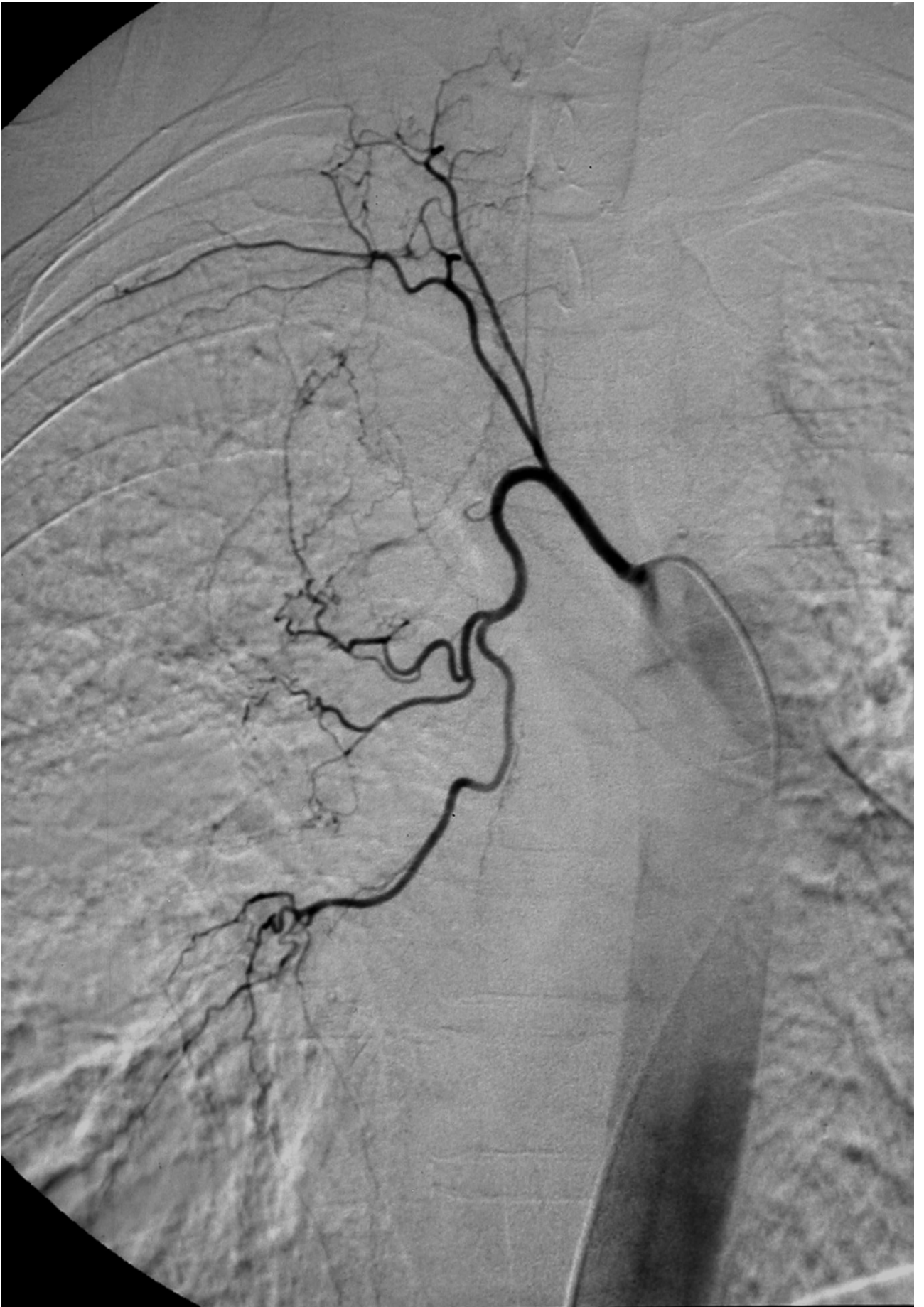
**Figure 2:** 70 year-old patient with a cigarette consumption of 69 pack-years and no airflow limitation on pulmonary function tests. This patient was referred for management of moderate hemoptysis originating from the right upper lobe.

Selective catheterization of the right intercostobronchial trunk showing features of marked hypervascularization on the side of bleeding including a tortuous and dilated right bronchial artery (arrows) and bronchial-to-pulmonary retrograde shunts in the right apex (star). Note the presence of tortuous but minimally dilated left bronchial branches (arrowheads), opacified because of the vicinity between the ostium of the left bronchial artery and that of the right intercostobronchial trunk. Immediate cessation of hemoptysis was obtained after embolization of the right intercostobronchial trunk with no recurrence of bronchial bleeding over the 5-year follow-up period.



**Figure 3:** 45 year-old patient with a cigarette consumption of 25 pack-years and moderate COPD (stage II). This patient was referred for management of massive hemoptysis originating from the right upper lobe.

Selective catheterization of the right intercostobronchial trunk showing normal bronchial arteries on the side of bleeding. Immediate cessation of hemoptysis was obtained after embolization of this artery. No late recurrence during the 4-year follow-up period.



**Figure 4:** 58 year-old patient with a cigarette consumption of 25 pack-years and mild COPD (stage I). This patient was referred for moderate hemoptysis originating from the right upper lobe which required surgery as definitive treatment.

**Figure 4a:** Selective catheterization of the right bronchial artery showing mild hypervascularization from its superior division (arrowhead) and more dilated vessels from its inferior division (arrows). Hemoptysis recurrence within 24 hours after embolization of this artery led to right upper lobectomy. No recurrence of hemoptysis was observed over the 4-year follow-up.





**Figure 4b:** Lung tissue section (hematoxylin and eosin-safran, x200) demonstrating numerous and dilated capillary vessels in a cartilaginous bronchial wall of the right upper lobe.

