BRONCHIAL ARTERY EMBOLIZATION: NON BRONCHIAL ARTERIAL SOURCES OF MASSIVE HEMOPTYSIS

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ABSTRACT

Introduction : Bronchial artery embolization (BAE) is a procedure of choice in those patients who have massive hemoptysis and they are poor surgical candidates for lung resection. Source of bleeding in patients with massive hemoptysis is mainly from bronchial artery but non bronchial systemic arteries are also significant contributiors. Knowledge of their anatomy and anatomic variations are important to the operator. In this study we evaluate the contribution of non-bronchial systemic arteries in the causation of massive hemoptysis in patients under going bronchial artery embolization (BAE). **Patients and Methods :** Two years of patients who underwent embolization for massive hemoptysis in our department data was retrospectively evaluated. All patients had physical examination, chest x-ray, chest computed tomography (CT), and fiber optic bronchoscopy before the procedure. Bronchial and non bronchial arterial angiography and embolization was performed with standard protocols. These angiograms were analyzed by two radiologists with over 5 years of experience in angiography. We recorded all feeders which were supplying the lesion e.g. bronchial artery, phrenic artery, intercostals artery, internal mammary artery & its branches, branches of subclavian (other than internal mammary artery) and axillary arteries. **Results :** All 22 patients had bronchial artery contributing (100%) to the hemoptysis. Ten (45.5%) patients had only bronchial artery supply without contribution from non-bronchial systemic arteries. Remaining twelve (54.5%) patients had mixed arterial supply from bronchial and non bronchial arteries. A total of 50 arteries were embolized in twenty-three sessions of embolization in twenty-two patients. Total 28 (56%) non bronchial arteries were contributing the massive hemoptysis. The breakup of non bronchial arterial contribution are 3 (10.7%) phrenic arteries, 11 (39.2%) intercostals arteries, 06 (21.4%) internal mammary arteries and 06 (21.4%) arterial branches of subclavian (other than internal mammary artery) and axillary arteries Except 01 patient, no other recurrence was observed in thirty days follow-up. **Conclusion :** In our study more than half of patients had non bronchial systemic arterial source of their hemoptysis. Therefore it is important to search for non bronchial arterial contribution in cases of massive hemoptysis when performing endovascular embolization.

KEY WORDS : Bronchial artery, angiography, embolization

Introduction

Hemoptysis can be managed by medical, surgical, endobronchial, and endovascular means.[1]

Bronchial artery embolization (BAE) is a procedure of choice in those patients who have massive hemoptysis and are poor surgical candidates for lung resection.[2]

Angiographic operator should know the side of bleeding by bronchoscopy before angiography and embolization.[3]

Interventional radiologist must know the anatomy and anatomic variations of bronchial arteries and non-bronchial systemic arteries that can be a significant source of massive hemoptysis and a cause of recurrence after bronchial artery embolization.[4] Non-bronchial systemic arteries that contribute to massive hemoptysis are internal mammary artery, intercostals arteries, phrenic artery, and other branches of subclavian and axillary arteries.

Purpose of our study is that to evaluate the contribution

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of non-bronchial systemic arteries in the causation of massive hemoptysis in patients under going bronchial artery embolization (BAE).

Patients and Methods

From January 2005 to August 2007, patients who had massive hemoptysis admitted to our hospital and referred to interventional suite for bronchial artery angiography and embolization were included in this study. Twenty two consecutive patients, including 18 men and 4 women; mean age, 54.2 years; age range; 25 – 72 years, were included.

All patients had physical examination, chest x-ray, chest computed tomography (CT), and fiber optic bronchoscopy before procedure. No patient had deranged coagulation profile & raised creatinine level. Most patients had blood transfusion in last 24 hours and were on intravenous fluid therapy before procedure. Right femoral artery was punctured in all patients, followed by placement of vascular access sheath. First descending thoracic aortogram was performed for assessment of vascular supply to already known side, by CT chest and bronchoscopy, of abnormality. Selective catheterization was performed with different catheters e.g. Cobra, Head-hunter, or Simmons for bronchial artery.

For internal mammary artery catheterization dedicated catheter was used, for subclavian or axillary arteries head hunter was used. After detecting the abnormal lung vascularity, micro catheter was used with co-axial method & super selective catheterization was achieved to prevent reflex into aorta or spinal branches of artery. Polyvinyl alcohol particles (350-500µm) were used for embolization.

We intentionally search for non-bronchial arterial contribution from phrenic arteries in case of basal involvement of lung and for upper lobe lesions. Subclavian angiogram was performed to search for supply from branches of the subclavian and axillary arteries. Embolization was achieved with same technique as used in bronchial arterial supply. Polyvinyl alcohol particles were stopped when significant sluggish flow was seen in embolized artery or minimal reflux in proximal artery was observed.

These angiograms were analyzed by two radiologists with over 5 years of experience in angiography. The angiographic finding most commonly present in lesions responsible for hemoptysis was marked hyper-vascularity of the involved area of pulmonary tissue. Frequently the arteries supplying this area were hypertrophied.

We recorded all feeders which were supplying the lesion e.g. bronchial artery, phrenic artery, intercostals artery, internal mammary artery & its branches, branches of subclavian (other than internal mammary artery) and axillary arteries.

Results

Of the 22 patients 13 had pulmonary tuberculosis, one had supper added aspergilloma on T.B, 2 patients had cystic fibrosis, 4 patients had chronic bronchitis and emphysema, 1 had bronchogenic carcinoma and 1 had Bronchiactasis. Acute hemoptysis was in 17 patients and chronic hemoptysis was in 5 patients. There degree of hemoptysis prior to embolization was less than 200mls of blood per day in 8 patients and more than 200mls per day in 14 patients. Initially 48 arteries in 22 patients were embolized successfully in 22 embolotherapy sessions. One patient re-bled with in 24 hours; repeat angiography demonstrated contribution to lesion from intercostals arteries, which were embolized successfully. Total 50 arteries were embolized in 22 patients in 23 sessions of embolotherapy.

Right lung involvement seen in 10 (45.4%) patients, left lung was involved in 10 (45.4%) patients and both lung involvements were seen in 2 (9%) patients. On right lung, upper lobe was involved in 7 (31%) patients, middle lobe was in 1 (4.5%) and lower lobe was in 3 (13%) patients. Left lung, upper lobe was involved in 9 (40.9%) patients and lower lobe was in 2 (9%) patients. Most common angiographic finding in all patients was hypertrophic and tortuous bronchial artery and hyper vascularity of lesion.

Acute bleeding was controlled in all patients who had acute hemoptysis. Only one patient as re-bled in twenty four hour, repeat angiography on re-bled patient showed additional arterial supply from 02 intercostals arteries, which were not observed in first angiogram. Embolization was done with polyvinyl alcohol particles. All 22 patients had bronchial artery contributing (100%) to the hemoptysis. Ten (45.5%) patients had only bronchial artery supply without contribution from non-bronchial systemic arteries. Remaining twelve (54.5%) patients had mix arterial supply from bronchial and non bronchial arteries. Total 50 arteries were embolized in twenty-three sessions of embolization in twenty-two patients.

Total 28 (56%) non bronchial arteries were contributing the massive hemoptysis.

The breakup of non bronchial arterial contribution are 3 (10.7%) phrenic arteries, 11 (39.2%) intercostals arteries, 06 (21.4%) internal mammary arteries (Fig.1) and 06 (21.4%) arterial branches of subclavian (other than internal mammary artery) and axillary arteries. Except 01 patient, no other recurrence was observed in thirty days follow-up.

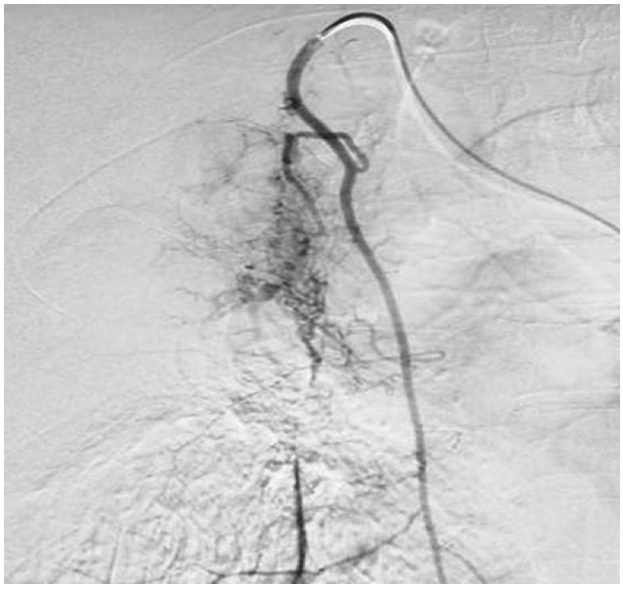

Figure 1: 56 year male known case of right upper lobe tuberculosis presented in emergency room with massive hemoptysis. Selective angiogram shows abnormal increase vascularity from branch of right internal mammary artery.

Discussion

During the past three decades percutaneous embolization of the bronchial arteries has become an accepted procedure to control massive or recurrent hemoptysis, especially in patients with chronic pulmonary diseases who are poor candidates for lung resection.[5,6] Hemoptysis in the vast majority of the patients originates from systemic rather than pulmonary arteries; the bronchial arteries are almost universally involved.[7,8] In addition to the bronchial arteries other systemic arteries may contribute to the perfusion of the lesions responsible for the hemoptysis and occasionally may be their major or only arterial blood supply. These non-bronchial systemic collaterals can originate from phrenic, intercostals, internal mammary, thyro-cervical and other branches of the subclavian and axillary arteries. Common angiographic finding in our study was hypertrophic and tortuous bronchial arteries and hyper vascularity with tortuous and hypertrophic non-bronchial arterial supply if involved. Active extravasations of contrast were not seen in a single patient. Fredrick S et al[8] had found only three cases out of 20 patients of acute bleeding, Ferris [9] did not had a single case with extravasations while Uflacker et al [10] observed it only in 03 out of 75 patients. In our group of patients, 56% (28 of 50) of the embolized arteries were non-bronchial systemic collateral. In this study 56% of patients had non-bronchial systemic collaterals which were considered to be contributing to the hemoptysis. In the only case of re-bleeding in this study non –embolization of these non-bronchial systemic collaterals was the cause. It is therefore an important cause of re-bleeding and treatment failure. Non bronchial supply should always be looked for and embolized when found.

Conclusion

In our study more than half of patients had non bronchial systemic arterial source of their hemoptysis. Therefore it is important to search for non bronchial arterial contribution in cases of massive hemoptysis when performing endovascular embolization.

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