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RESEARCH ARTICLE

EMBOLIZATION OF NON-BRONCHIAL SYSTEMIC COLLATERALS FOR TREATMENT OF SEVERE HEMOPTYSIS

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ABSTRACT

Massive hemorrhage from bronchial circulation in 90% cases results in respiratory emergency. However, nonbronchial systemic arteries can be responsible for massive hemoptysis especially in patients with pleural involvement caused by an underlying disease. This study includes 174 cases of non-bronchial segmental artery embolization out of a total 400 cases of intervention embolotherapy, performed over a period of 11 years. The immediate control of hemoptysis was achieved in 160/174 (91.95 %). Repeat embolization within one week was done in fourteen patients (8.04%) who had massive recurrent hemoptysis 14/174(8.045%). The incidence of minor complications was in 60 /174 (34.48%) which includes transient dysphagia in 24/174(13.79%) and muscular chest pain in 36/174(20.6%). Major complication was not observed in any patient. Chest radiography was used as a primary tool for the diagnosis and localization of source of hemoptysis.

INTRODUCTION

Bronchial artery embolization (BAE) is an accepted technique worldwide and remains the first line of treatment for massive hemoptysis. It is a minimally invasive procedure without major complications for even the most compromised patient. Endovascular management of hemoptysis has great potential and shown promising results. Embolization of non- bronchial systemic arteries is also required in patients having persistent or recurrent hemoptysis even after successful BAE (David R Spoko, 2011). A thorough hunt of arteries other than bronchial arteries should always be made to achieve complete control of hemoptysis. The incidence of cessation of haemorrhage ranges from 85% to 100%; although recurrence of haemorrhage ranges from 10% to 30% (David R Spoko, 2011). Severe hemoptysis is associated with high mortality rate of 30 to 50% (Conlan *et al*., 1983). Cause of death in these cases is usually due to asphyxia caused by flooding of blood in the airways. The commonest cause of hemoptysis is tuberculosis and its sequel such as cavities, post tubercular bronchiectasis, residual fibrosis and healed cavities with aspergillus (Conlan *et al*., 1983).

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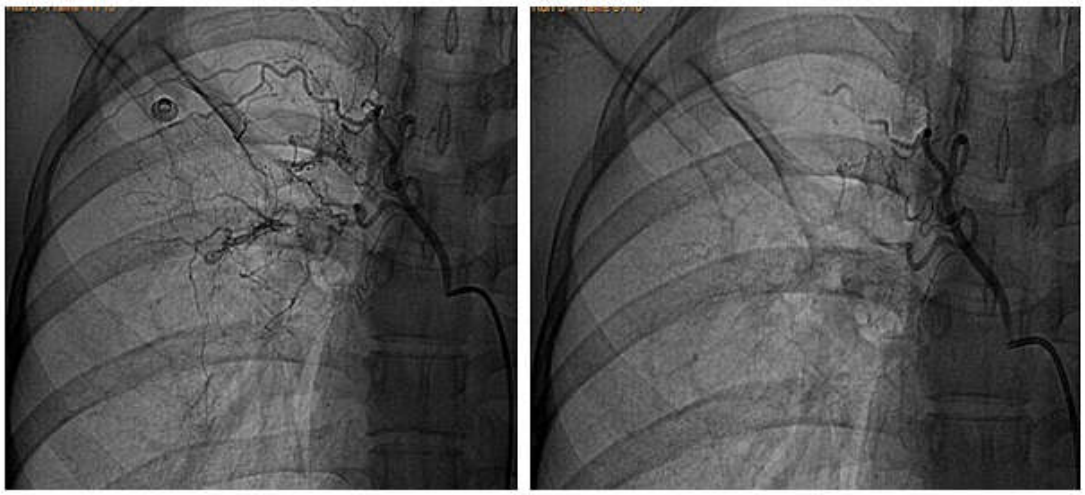
The major source of hemoptysis in these cases is hypervascularity due to feeders arising from bronchial as well as non-bronchial systemic sources (Eckstein *et al*., 1986). Majority of patients with massive hemoptysis usually have lack of respiratory reserve and are therefore, poor candidates for surgery (Eckstein *et al*., 1986).

Objectives: The aim was to study the efficacy of embolization of non-bronchial systemic arteries in the management of severe hemoptysis.

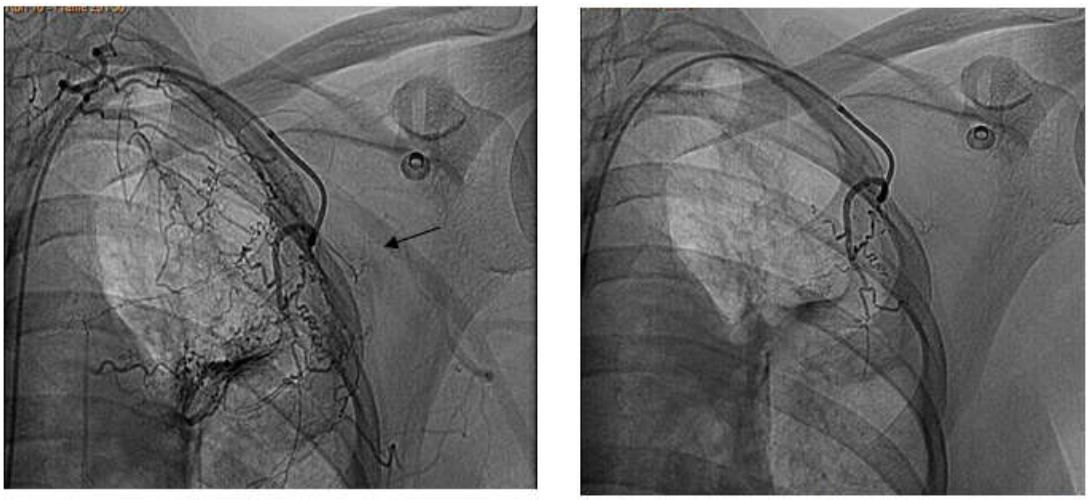
MATERIALS AND METHODS

One hundred and seventy four patients, 136 men (75.2%) and 38(24.8%) women between the age of 15 to 75 years were studied during January 1998 to Dec 2016. In 160 cases, embolization of non-bronchial segmental artery responsible for hypervascularity, was offered. In recurrent hemoptysis, repeat angiography with non-bronchial segmental artery embolization was done in 14 cases (8.04 %): in 10 cases within 24 hours and in 4 cases within 7 days. Chest Radiograph was the only prerequisite for angiography. Bronchoscopy was not done in any of the patients. CECT was done as per the need.



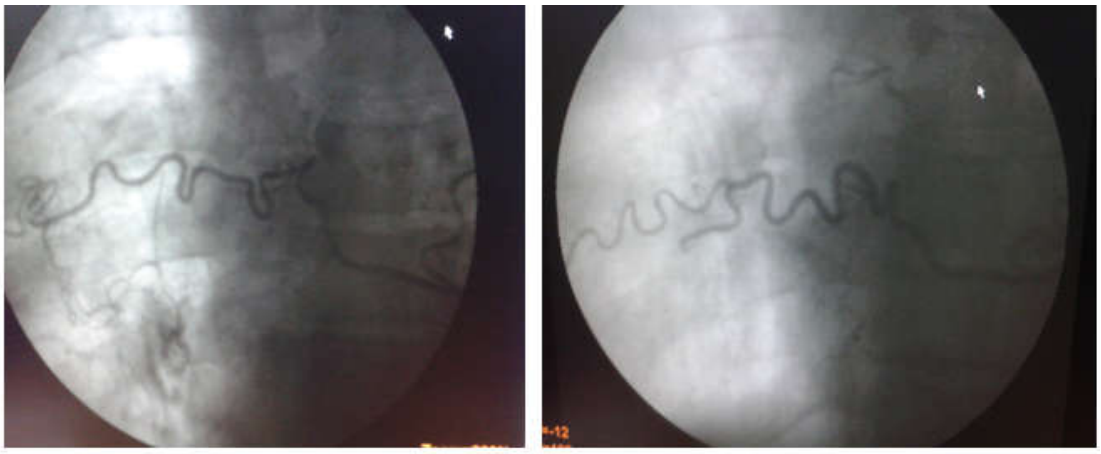
(A) (B)

Fig.1. (A) Right intercostal bronchial artery angiogram (Pre-embolization): selective injection shows hypervascularity from intercostals branch; (B) Post-embolization angiogram shows loss of hypervascularity



(A) (B)

Fig.2. (A) Left lateral thoracic artery pre-embolization angiogram showing hypervascularity at left upper zone with multiple dilated tortuous feeders at peripheral lung field (arrow); (B) post-embolization angiogram showing diminished number and caliber of feeder arteries and reduced vascularity with complete occlusion of feeding artery



(A) (B)

Fig.3. A. Pre-embolization angiogram showing two feeder arteries from right intercostal artery supplying the hypervascular lesion; (B) Post-embolization angiogram showing complete loss of hypervascularity from feeder arteries

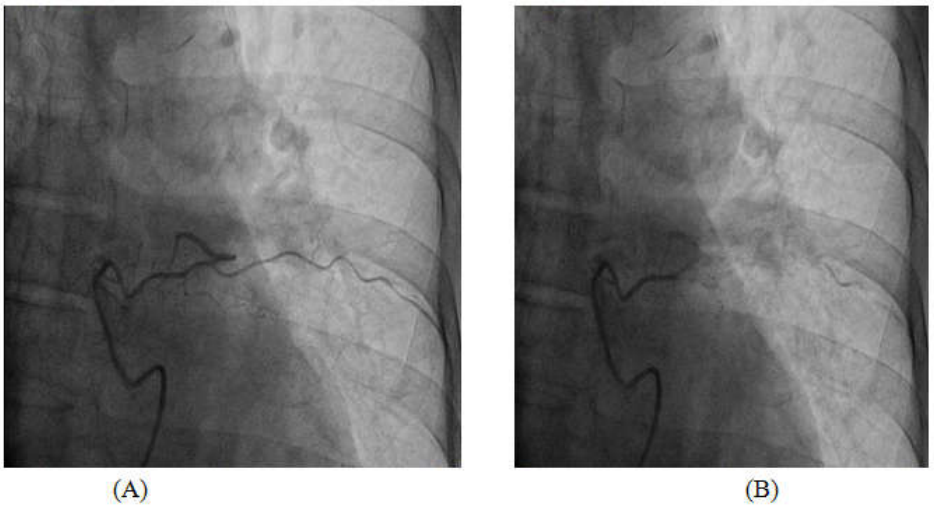


Fig.4. A. Left intercostal artery angiogram showing hypervascularity from a branch of intercostal artery; (B) post-embolization angiogram shows loss of hypervascularity from feeder intercostal vessel

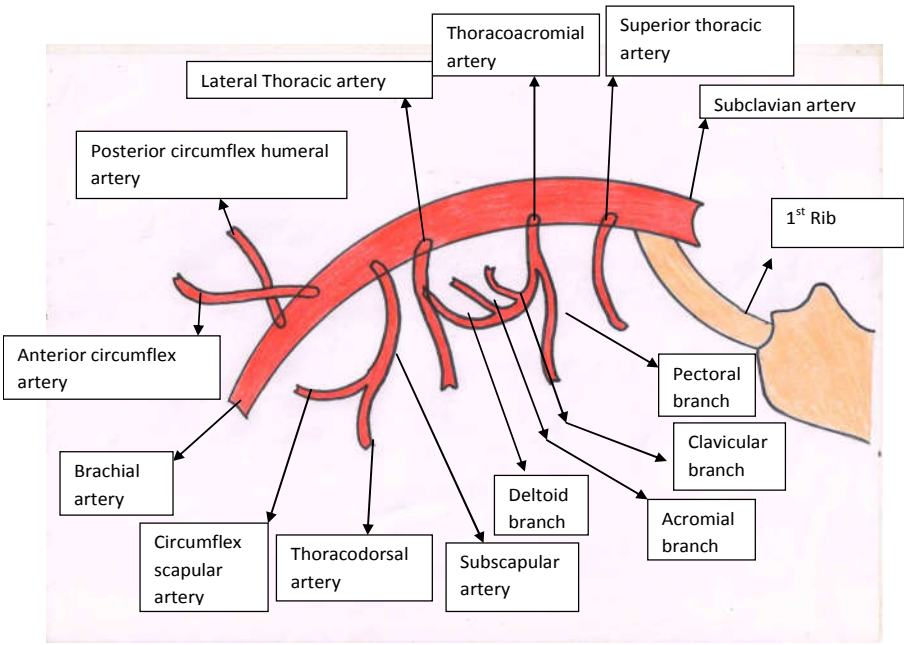


Fig. 5. Branches arising from three segments of axillary artery

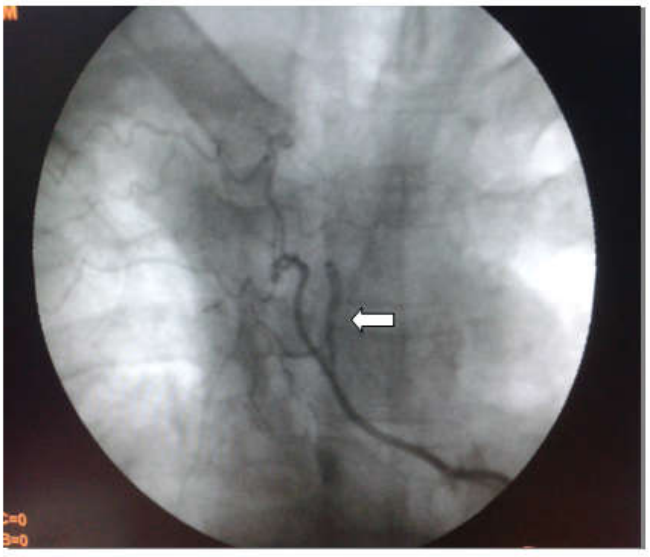


Fig.6: Anterior spinal artery (⇦) taking origin from right costo-cervical trunk

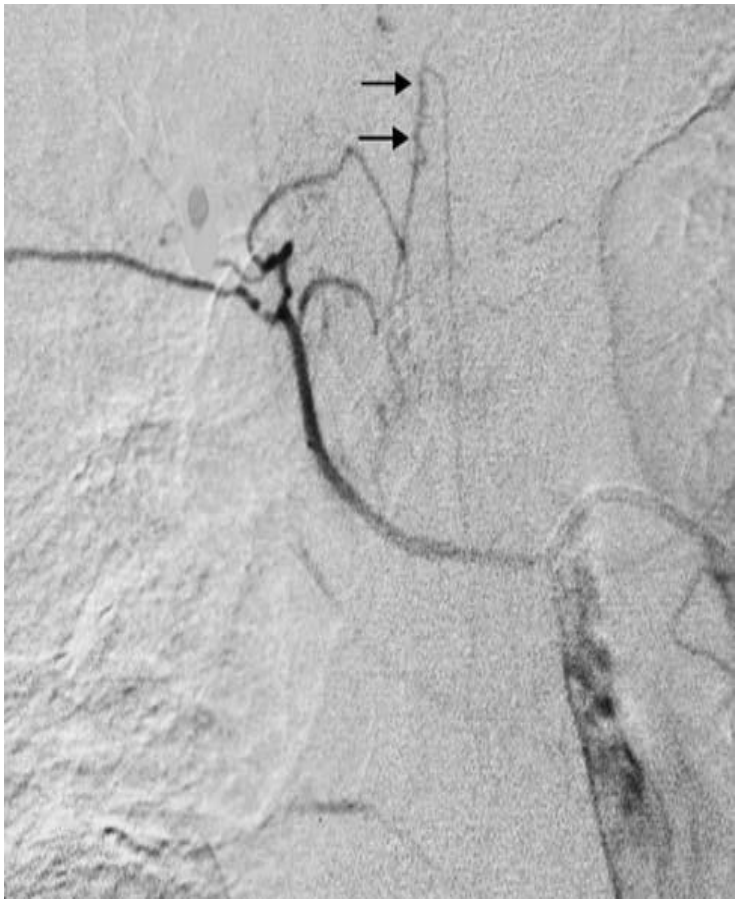


Fig.7. Selective right intercostal artery angiogram showing origin of anterior spinal artery (double arrow) with characteristic hair pin bend appearance. Microcatheter should be advanced beyond the origin of the anterior spinal artery

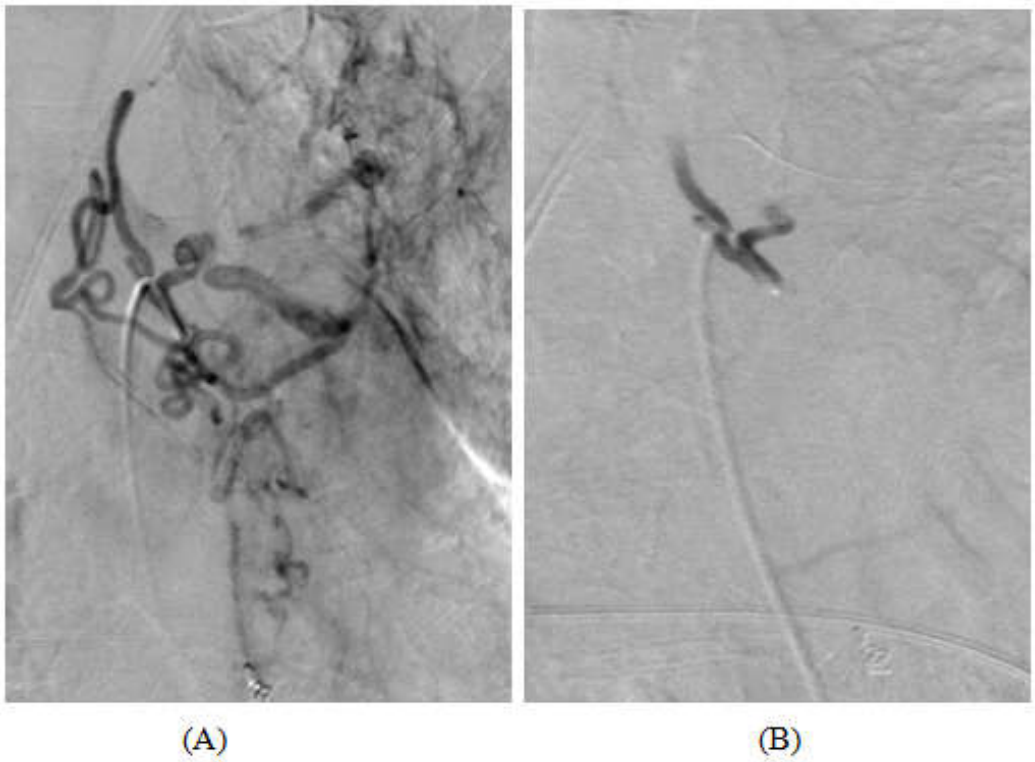


Fig.8. A. 24 year female with tuberculosis. Superselective pre-embolization angiogram using co-axial technique shows hypervascularity from left bronchial artery with arterio-venous shunting at left upper lobe. (B) Post-embolization angiogram shows loss of peripheral vascularity

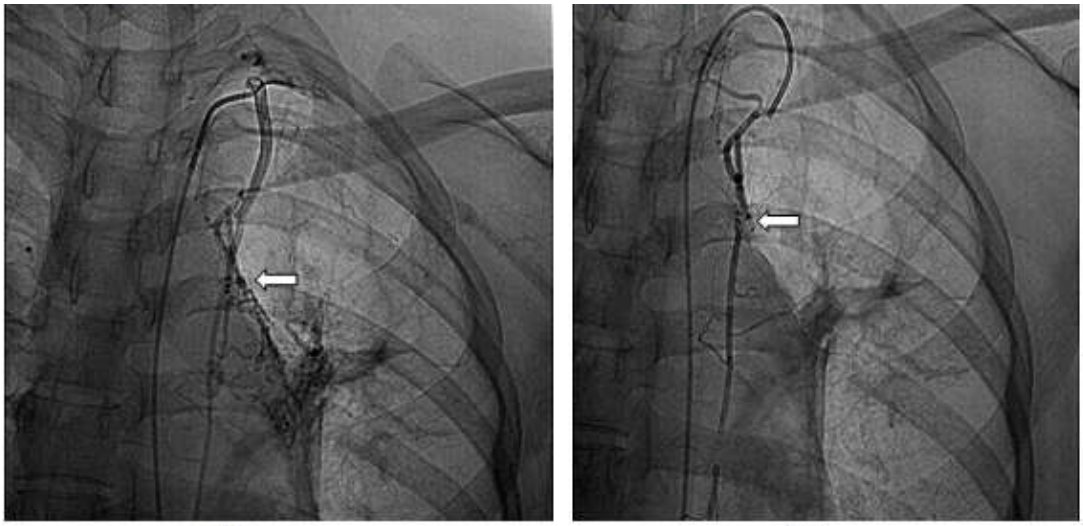


Fig.9: A. Pre-embolization angiogram showing hypervascularity at left upper zone with multiple dilated tortuous feeders from left internal mammary artery (arrow); B: post-embolization angiogram showing markedly reduced vascularity (arrow).

Technique: Diagnostic angiography was performed through the right femoral artery using 4f visceral hook catheter which was properly hooked into the feeder artery. Initial thoracic flush aortogram was done as per the need of the situation. Many advocate initial thoracic aortography to delineate the number, size and position of the bronchial arteries (Marshal *et al*., 1997; Yoon *et al*., 2002; Philips *et al*., 2000) particularly in cases of aberrant or ectopic bronchial arteries. A preliminary angiogram was obtained to localize the area of hypervascularity. Subsequently, the lesion was embolized with small gel foam pallets (3-8mm) suspended in non-ionic contrast media. Sometimes, gel foam mixed with polyvinyl alcohol particles of 350-500 micron size was used. Co-axial system using microcatheter with controlled hand injection with small volume syringes was used when primary catheter could not be stabilized for delivery of embolic material. Angiogram after embolization showed complete occlusion of the feeder artery. Patients were followed-up for six months as out-patient. Angiography should be performed with either low-osmolar or iso-osmolar nonionic contrast material, as high osmolar contrast has resulted in transverse myelitis (Fiegelson, 1965; Khalil *et al*., 2010),

Criteria for hemoptysis and success of Embolization

- Criterion for inclusion of patients in this study was massive hemoptysis i.e. more than 400 ml of blood in 24 hours.
- The criterion for successful embolization was disappearance of hypervascularity resulting in expectoration of less than 100 ml of blood in 24 hours post-embolization.
- Complete control of hemoptysis within one week was considered as successful embolization.
- Hemoptysis of more than 100 ml in 24 hours was considered as treatment failure.

RESULTS

Non-bronchial segmental artery as a feeder was found in 174/400 (43.5% cases). Additional feeders were detected in 132 cases (75.8%): right intercostal artery in 72 cases(54.54%) and left intercostal artery in 60 cases (45.46%).

The lateral thoracic artery as a feeder vessel was seen in 2 cases (1.14%). Details of feeder arteries and additional feeders are shown in Table1. Therepeutic embolization of non-bronchial segmental artery was done in 174 patients. 160/174 (91.95%) had immediate control of hemoptysis. Residual hemoptysis 5 to 20 ml for 2 to 7 days was observed in 14/174 (8.04 %): recurrence within 24 hours was observed in 10/174 (5.74%) and within one week in 4 cases (2.29%). In 2 patients (1.14%), with spinal artery was found as a feeder, the embolization was performed by placing the catheter tip beyond the origin of the anterior spinal artery (Table 2). CECT was done in cases who had recurrent hemoptysis after embolization (14/174= 8.04%).

Complications: Incidence of Minor complications was 34.48% (60/174): transient dysphagia lasting for > 24 hours in 24/174(13.79%) and muscular chest pain for < 15 minutes in 36 (20.68%). 6 patients even after a successful embolization, died due to various other causes. None of the patients died due to inadvertent embolization.

DISCUSSION

Historically, surgery is considered as definitive therapy in severe hemoptysis. A mortality of 18% in electively used surgical intervention may rise to 40% when performed in emergency (Fernando *et al*., 1998). Massive hemoptysis is a life threatening condition which requires emergency intervention (Rabkin *et al*., 1987). The bronchial arteries are most commonly responsible (Jardin M Remy, 1988; Ramakantan *et al*., 1996), however involvement of non bronchial systemic collaterals should be kept in mind especially when bronchial arteries are normal. BAE was introduced in 1973 by Remy *et al*. (Remy *et al*., 1973), is considered now-a-days as the first-line therapy (6). Hapnoic *et al*. (2000) reported in a survey that 50% clinicians prefer an interventional radiologic procedure of arterial embolization for the treatment of massive hemoptysis.

Etiology of Hemoptysis: Active tuberculosis remains the most common cause of hemoptysis all over the world (Marshal *et al*., 1997; Jean-Baptiste, 2000). Cystic fibrosis, bronchiectasis,

bronchogenic carcinoma and congenital heart disease are the non-inflammatory causes of hemoptysis. Amongst the smoking population, it is accounting for 42% of hempotysis complaints (Hirschberg, 1997; Hiyama, 2002).

Brief anatomy of bronchial arteries and nonbronchial segmental collaterals: The bronchial and pulmonary circulations are two distinct circulatory systems that supply blood to the lungs. The bronchial circulation terminates at the level of the terminal bronchioles where it joins with pulmonary capillaries and venules (Carlos Jose Suarez, 2012).The pulmonary artery which originates from the right ventricle gives rise to pulmonary circulation. Pulmonary capillaries empty into pulmonary veins within the pulmonary parenchyma coursing along the interlobular septa (Carlos Jose Suarez, 2012). Bronchial circulation is the source of massive hemoptysis in 90% cases (Yoon *et al*., 2002).Bronchial arteries vary significantly in their numbers and sites of origin: more than 70% from the descending aorta between the levels of the fifth and sixth thoracic vertebrae (Stroll, 1988), 20% may have an aberrant origin(from other systemic arteries); 10% from anterior surface of the aortic arch or the descending aorta, and in 5% a spinal artery can originate from a bronchial artery; right side being more common than the left(5, 20). The variant anatomy includes its origin from aortic arch, internal thoracic artery, thyro-cervical trunk, subclavian artery and coronary arteries. The accessory/ nonbronchial arterial supply may originate from thoracic or abdominal vascular distributions (Walker *et al*., 2015). The nonbronchial systemic arteries can be distinguished from the bronchial arteries by careful observation of the course of the vessel. The ectopic or orthopic bronchial arteries have a more vertical course while nonbronchial systemic arteries follow a transpleural course, but do not join the bronchial tree. This should be identified at the time of initial arteriogram (Kellar *et al*., 1987) and should be investigated to avoid failure of the procedure resulting in recurrence of hemoptysis (Kellar *et al*., 1987). In the presence of pleural thickening, non-bronchial systemic feeder vessels may originate from intercostals arteries (Fig.1& 2), branches of the subclavian (Fig.3) and axillary arteries, internal mammary artery (Fig.4), inferior phrenic artery (Marshal *et al*., 1997; Kellar *et al*., 1987; Hashimoto, 1990; Katoh, 1990; Do, 2001) and develop along the pleural surface to become enlarged as a result of the inflammatory process and can be a major source of massive hemoptysis (Yoon *et al*., 2002). In chronic tuberculosis repeat hemoptysis is due mainly to hypertrophy of the collateral nonbronchial systemic arteries (Goh *et al*., 2002). Failure to recognize non–bronchial feeders, leads to persistence of hemoptysis (Yoon *et al*., 2002). Many investigators have documented that a concerted search for non-bronchial systemic arterial supply should be made (Marshal, 1997; Kellar *et al*., 1987; Goh *et al*., 2002; Wong *et al*., 2002) to avoid early recurrent bleeding after successful embolization. The branches arising from three segments of axillary artery are depicted in Fig. 5. The anterior spinal artery assumes the classic "hairpin" configuration in angiography (Fig. 6 & 7). Embolization of the medullary artery resulting in transverse myelitis, can be avoided by placing the coaxial microcatheter tip beyond the origin of the artery (Fiegelson, 1965). All angiograms, including intercostal arterogram, must be carefully scrutinized for opacification of spinal arteries to avoid inadvertent embolization (Fiegelson, 1965). In chronically inflamed lungs apart from hypervascularity, abnormal broncho-pulmonary shunts develop, which get blood supply through the bronchial as well as non- bronchial arterial feeders (Fig.8). On angiography, the major source of hemorrhage is the bronchial as well as systemic non – bronchial arteries; and hypervascularity is the single most common pathological abnormality on the affected side (Ramakantan *et al*., 1996; Remy, 1973; Kellar *et al*., 1987). Trans-pleural angiogenesis occurs in the setting of chronic inflammatory or neoplastic conditions which may result in recurrent hemoptysis (Marshal *et al*., 1997; Yoon *et al*., 2002; Kellar *et al*., 1987; Katoh *et al*., 1990). The presence pleural thickening in chest radiograph, negatively influences the long term success rate of bronchial artery embolization (Tamura *et al*., 1993). In many acute and chronic lung diseases, pulmonary circulation is reduced or occluded at the level of the pulmonary arterioles because of hypoxic vasoconstriction, intravascular thrombosis and vasculitis (Deffebach *et al*., 1987).The convenience and portability of chest radiography, make it a useful diagnostic tool in the evaluation of hemoptysis. The chest radiograph can be normal in up to 30% of patients (Marshal *et al*., 1997; Hirschberg *et al*., 1997). The episode of massive hemoptysis or the presence of bilateral pleural disease makes localization difficult due to opacification of both lungs. CT and CECT are helpful for rapid examination in critically ill patients allowing detection of a feeder vessel supplying a parenchymal lesion and in identification of bronchial or non-bronchial systemic artery. It is also helpful in localizing the source of bleeding and in selection of interventional procedure (Yoon *et al*., 2002) (Fig.10). In a prospective study of 40 patients with massive hemoptysis, 27 patients (67.5%) had a nonbronchial systemic arterial supply had an overall accuracy of 84% in identifying this finding on CT (Yoon *et al*., 2002) (unpublished data). Yoon *et al*. (2002) have described that pleural thickening of more than 3 mm and tortuous enhancing vascular structures within hypertrophic extrapleural fat seen in CECT, are signs of non-bronchial systemic arterial supply in patients with massive hemoptysis. In addition to the parenchymal pathology, CECT is also helpful in detecting the enlarged bronchial and non-bronchial arteries (Abul *et al*., 2001).

Multidetector computed tomography (MDCT) is a developing imaging technique for both the localization of hemorrhage and identification of the causative etiology of hemoptysis (Do *et al*., 2001). Bronchocopy, once considered as the primary method to localize the bleeding source in hemoptysis, has been shown to have a less chance of localizing the source as compared to CECT. Generally accepted rates of cessation of hemoptysis following bronchial artery embolization approach 90% (Yoon *et al*., 2002; Stroll *et al*., 1988). Guidelines for massive hemoptysis includes 200 to 1000 ml over a 24 hour interval, but the volume documentated as > 300 ml appears to be most frequently accepted (Yoon *et al*., 2002; Marshal *et al*., 1997; Crocco, 1968). Patients with chronic hemoptysis may have an average of >100 ml per day for 3 or more days (Kalva, 2009).

Safety and Efficacy: Embolization therapy is very effective in acute control of hemoptysis. Super-selective catheterization and complete embolization improve the initial success rate. The recurrence rate depends on several factors including the primary disease condition, adequacy of the procedure and presence of non-bronchial systemic collaterals. The causes of delayed recurrence include incomplete embolization, re-canalization of the embolized vessel, development of other

systemic collaterals and progression of the underlying disease (Marshal, 1997; Katoh *et al*., 1990). In several reports, the major conditions in which recurrence has been noted include tuberculosis, aspergilloma and lung cancer (Katoh *et al*., 1990; Goh, 2002).

Complications: BAE is a safe procedure. Transient chest pain and dysphagia are the common minor complications.

Minor Complications: Chest pain occurring in 24-91% is self-limiting in majority of cases (12, 33).Chest pain lasting for 15 minutes was seen in 15 patients (8.62%). were treated with narcotic analgesics. Transient dysphagia is also self-limiting and may result in 18% of interventions (Ramakantan *et al*., 1996; Ramakantan *et al*., 1999).

Major Complications: The occurrence of transverse myelitis because of spinal cord ischemia may vary from 1.4-6.5% (Ramakantan *et al*., 1996; Wong *et al*., 2002; Tanaka *et al*., 1997; Mal *et al*., 1999). This most serious complication was not observed in any patient in this series. Pain in the orbit or temporal region ipsilateral to the side of embolization is considered to be referred pain (Ramakantan *et al*., 1999). Cortical blindness represents an extraordinary rare neurologic complication (Liu *et al*., 1998). The other rare complications include bronchial stenosis, necrosis, bronchoesophageal fistula (Girard *et al*., 1990). Pulmonay infarction and ischemic colitis have been described as isolated case reports (Lemoigne *et al*., 1983). Active extravasation of contrast reported by Ramakantan *et al*. (1996) in 10.7% of examinations, was found in two cases in this study (1.14%). (Fig.11 & Fig.12).Stroke as a complication can occur due to particle size smaller than the size of the small vascular communications between the bronchial arterioles and pulmonary venous branches. Stroke can result from traumatic catheterization of contributing vessels such as systemic and anomalous bronchial arteries (Park *et al*., 2012; Fitz Gerald *et al*., 2005). Use of different embolic material, success rate, incidence of recurrence and complications by various authors is shown in Table 3. Compared to gelfoam, PVA particles have less chance of recanalization and hence recurrence of hemoptysis. PVA particles, therefore are permanent embolizing material. Absolute alcohol and N-butyl cyanoacrylate glue are other liquid embolizing materials which are not routinely used because of the extensive tissue necrosis associated with them (Marshal *et al*., 1997; Hashimoto *et al*., 1999). Metallic coils are not the preferred embolizing agents as they lead to more proximal occlusion and loss of subsequent access to the bleeding vessel (David R Spoko, 2011).

Conclusion

The embolization of nonbronchial systemic artery is a safe and highly effective nonsurgical treatment for immediate control of massive hemoptysis with less significant complications. It can be repeated in cases of recurrent hemoptysis. Surgery, however, remains the definitive treatment of hemoptysis.

REFERENCES

Abul AT, Nair PC, Cherian J. 2001. Hemoptysis: etiology, evaluation and outcome: a prospective study in a third-world country. *Resp Med.,* 95: 548-52.

Carlos Jose Suarez, Charles W. 2012. Frevert, In Comparative Anatomy and Histology.

Cohen AM, Doershuk CF, Stern RC. 1990. Bronchial artery embolization to control hemoptysis in cystic fibrosis. Radiology 175(2): 401-405. (PubMed)

Conlan AA, Therwitz SS, Krige SS, Jhonnesburg L *et al*. 1983. Massive Hemoptysis, *J Thorac. cardio vas. Surg.* 85:120-124.

Crocco JA, Rooney JJ, Fankushen DS *et al*. 1968. Massive hemoptysis. *Arch Intern Med.* 121(6): 495-498. (PubMed)

David R Spoko, Tony P Smith. 2011. Bronchial artery embolisation for hemoptysis. Semin intervent. *Radiology,* 28, 48-62.

Deffebach ME, Charan NB, Lakshminarayan S *et al*. 1987. The bronchial circulation. Small, but a vital attribute of the lung. *Am Rev Dis.,* 135(2): 463-481.(PubMed)

Do KH, Goo JM, Im JG *et al*. 2001. Systemic arterial supply to the lungs in adults: spiral CT findings. *Radio Graphics*., 21: 387-402. Link

Eckstein MR, Wallman AC, Athanaulig CA. 1986. The management of massive hemoptysis, control by angiographic methods: Current controversies in thoracic surgery, Philadelphia WB Saunder; 255-260.

Fernando HC, Stein M, Benfield JR *et al*. 1998. Role of bronchial artery embolization in the management of hemoptysis. *Arch Surg.,* 133(8):862-68. (PubMed)

Fiegelson HH, Ravin HA. 1965. Transverse myelitis following selective bronchial arteriography. *Radiology.,* 85(4): 663-65(PubMed)

FitzGerald DB, Suran EL, Sargent J. 2005. Posterior circulation infarct after bronchial artery embolization and coiling. *Neurology.,* 65(8): 1312 (PubMed)

Girard P, Baldeyrou P, Lemoine G *et al*. 1990. Left mainstem bronchial stenosis complicating bronchial artery embolization. *Chest*. 97(5): 1246-1248. (PubMed)

Goh PYT, Lin M, Teo N *et al*. 2002. Embolization for hemoptysis : a six-year review. Cardiovas Intervent Radiol 25: 17-25. *Cross Ref; Medline*

Haponic EF, Fein A, Chin R. 2000. Managing life-threatening hemoptysis: has anything really changed? Chest 2000; 118(5): 1431-35. (PubMed)

Hashimoto M, Helanna J, Okane K *et al*. 1999. Internal mammary artery embolization for hemoptysis. *Acta Radiol.,* 40 : 187-190. CrossRef ; Medline

Hirschberg B, Biran I, Glazer M *et al*. 1997. Hemoptysis : etiology and outcome in a tertiary hospital. *Chest*, 112(2): 440-44.(PubMed)

Hiyama J, Horita N, Shoita Y *et al*. 2002. Cryptogenic hemoptysis and smoking. *Chest*, 12 (4):1375-76.author reply 1376.(PubMed)

Jardin M Remy J. 1988. Control of hemoptysis: systemic angiography and anastomoses of the internal mammary artery. *Radiology.* 168:377-383.

Jean-Baptiste E. 2000. Clinical assessment and management of massive hemoptysis. *Crit Care Med..,* 28 (5) : 1642-47.(PubMed)

Kalva SP. 2009. Bronchial artery embolization. *Tech Vasc Interv Radiol.,* 12(2): 130-38

Katoh O, Kishikawa T, Yamada H *et al*. 1990. Recurrent bleeding after arterial embolization in patients with hemoptysis. Chest., 97: 541-546.*Cross Ref; Medline*

Kellar F.S. Rosch F, Loffin, T.G etal. 1987. Non bronchial systemic collateral arteries: significance in percutaneous

embolothereapy for hemoptysis. *Radiology* 164:687-692.Link

Khalil A, Fartoukh M, BazotM *et al*. 2010. Systemic arterial embolization in patients with hemoptysis: initial experience with ethylene vinyl alcohol copolymer in 15 cases. *AJR Am J Roentgenol.* 194(1): W104-W110.(PubMed)

Lemoigne F, Rampal P, Petersen R. 1983. Fatal ischemic colitis after bronchial artery embolization.Presse Med.; 12(33): 2056-2057. (PubMed)

Liu SF, Lee TY, Wong SL *et al*. 1998. Transient cortical blindness: a complication of bronchial artery embolization. *Respir Med.,* 92(7): 983-986.(PubMed)

Mal H, Rullon I, Mellot F *et al*. 1999. Immediate and long-term results of bronchial artery embolization for life-threatening hemoptysis. *Chest*., 115(4): 996-1001. (PubMed)

Marshal TJ, Jackson JE. 1997. Vascular intervention in the thorax: bronchial artery embolization for hemoptysis. Eur Radiol 7: 1221-27. *Cross Ref; Medline*

Park JH, Kim DS, Kwon JS *et al*. 2012. Posterior circulation stroke after bronchial artery embolization and coiling. Neurol Sci 2012; 33(4): 923-26 (PubMed)

Philips S, Ruttley MS. 2000. Bronchial artery embolization: the importance of preliminary thoracic aortography. *Clin Radiol.,* 55(4):317-19.(PubMed)

Rabkin JE, Astafjev VI, Gothman LN *et al*. 1987. Transcatheter embolization in the management of pulmonary hemorrhage. *Radiology* 163(2): 361365 (PubMed)

Ramakantan R, Bandekar VG Gandhi MS, Aulakh BG, Deshmukh H.L. 1996. Massive hemoptysis due to pulmonary tuberculosis: Control with bronchial artery embolization. *Radiology,* 200 (93):691-694.(PubMed)

Ramakantan R, Ketkar M, Maddali K *et al*. 1999. Referred pain to the ipsilateral forehead and orbit: An unusual phenomenon during bronchial artery embolization. *Cardiovasc Intervent Radiol.* 22(4): 275-277. (PubMed)

Remy J, Voisin C, Ribet M *et al*. 1973. Treatment by embolization of severe or repeated hemoptysis associated with systemic hypervascularization. *Nouv Presse Med.,* 2:2060-68.(Frech).

Stroll J F, Bettman MA. 1988. Bronchial artery embolization to control hemoptysis: a review. Cardiovasc Intervent Radiology; 11:262-269

Swanson K. L, Johnson C. M, Prakash U. B. *et al*. 2002. Bronchial artery embolization: experience with 54 patients. Chest, vol. 121, no. 3, pp. 789–795.View at Publisher · View at Google Scholar · View at Scopus

Tamura S.Kodama T, Otsuka N *et al*. 1993. Embolotherapy for persistent hemoptysis: the significance of pleural thickening. *Cardiovas Intervent Radiol.,* 16: 85-88. CrossRef ; Medline

Tanaka N, Yamakado K, Murashima S *et al*. 1997. Superselective bronchial artery embolization for hemoptysis with a coaxial microcatheter system. *J Vasc Interv Radiol.* 8(1 Pt 1): 65-70. (PubMed)

Walker CM, Rosado-de-Christenson ML, Martinez-Jimenez S *et al*. 2015. Bronchial arteries: anatomy, function, hypertrophy and anomalies. *Radiographics,* 35(1): 32-49. Doi: 10,1148/rg,351140089-Pubmed citation

Wong ML Szkup P, Hopley MJ. 2002. Percutaneous embolo-therapy for life threatening hemoptysis. Chest; 95-102

Yoon, W, Kim JK, KimYH Chung TW, Kang HK. 2002. Bronchial and non bronchial systemic artery embolisation for life threatening hemoptysis; a comprehensive review *Radiographic,* 22(6);1395-1409.(PubMed)

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