

## CLINICAL VIGNETTE

---

# HEMOPTYSIS- Massive or Not

---

Brian K. Wong, M.D.

### *Case Report*

The patient is an 82-year-old female who presents with acute hemoptysis. She was asymptomatic until the morning of admission, when she coughed up approximately 30 ml of bright red blood. She had no complaints of shortness of breath or chest pain. Several hours later, she developed a second episode of hemoptysis of approximately 300 ml, became dyspneic and presented to the emergency department.

Past medical history was significant for atrial fibrillation treated with aspirin and digoxin. She had a significant smoking history of 40 pack/years, stopping 20 years ago. There was no known history of tuberculosis exposure.

Physical examination revealed an apprehensive female in moderate respiratory distress with an arterial oxygen saturation of 98% with FiO<sub>2</sub> of 1.0 by mask. Vital signs revealed a temperature 37.1C, respirations 22 blood pressure 141/72 mmHg, pulse 122 irregularly irregular. Examination of the nares revealed no evidence of epistaxis. No tongue or oral mucosal lesions were noted. There was no palpable cervical or supraclavicular adenopathy. Auscultation of the lungs revealed harsh rhonchi left upper lung zone posteriorly and bibasilar inspiratory crackles. The abdomen was soft, nontender and without organomegaly. The skin was without rash or visible lesions.

Laboratory revealed a WBC 16.1 thousand, hemoglobin 12.3 g/dl, platelet count 237,000, INR 1.02, and PTT 27 seconds. Electrolytes sodium 148 mmol/L, potassium 3.7 mmol/L chloride 119 mmol/L serum bicarbonate 17 mmol/L. BUN, creatinine, and liver function panel were normal. Arterial blood gas PaO<sub>2</sub> 89 mmHg and PCO<sub>2</sub> 32 mmHg pH 7.38

Chest x-ray revealed increased density in the left hilar area left lower lobe infiltrate. CT scan of the chest revealed left upper lobe mass like infiltrate. There were patchy lower lobe ground glass infiltrates. A large filling defect is seen in the distal aspect of the trachea extending into the left mainstem bronchus and left upper lobe bronchus. Bronchial

wall thickening was seen in the left upper lobe where the infiltrate was present. Transthoracic echocardiogram revealed left ventricular ejection fraction 30-35%. There was moderate tricuspid regurgitation with estimated right ventricular systolic pressure calculated at 42 mmHg

The patient continued to have respiratory distress and developed acute hypercapnic respiratory failure. She was intubated and placed on mechanical ventilation. Diagnostic bronchoscopy was performed which revealed clot in the distal trachea extending to left main stem bronchus and obstructing the left upper lobe bronchus. No evidence of active bleeding was noted. Cultures and cytology studies were negative.

Thoracic surgery consultation was obtained. On the second day of intubation, she underwent repeat fiberoptic bronchoscopy in the operating suite. Significant well-formed organized clot overlying the bifurcation of the left and upper lobe bronchi were lavaged clear. There was no active bleeding. No endobronchial lesions were noted. Repeat cytological studies were negative for malignancy.

On the fourth day of mechanical ventilation, the patient developed recurrent hemoptysis of approximately 150 ml. She was taken back to the operating suite. After removal of clot in the left main stem and upper lobe bronchi, active bleeding was noted emanating distally from the left upper lobe bronchus. Left thoracotomy with left upper lobectomy was performed. Postoperatively the patient did well and was successfully extubated. There was no further hemoptysis. She was subsequently discharged and continues to do well.

Surgical pathology of the left upper lobe revealed extensive intra-alveolar hemorrhage with evidence of pulmonary arteriovenous malformation. There was no evidence of malignancy.

## **Introduction**

Hemoptysis, massive or not, is an alarming symptom for both patient and healthcare personnel. Massive hemoptysis has usually been defined by the volume of expectorated blood. Amounts from 200 ml<sup>1</sup> to 1000ml<sup>2</sup> over 24 hours have been advocated without consensus. Newer definitions advocate that abnormal gas exchange and hemodynamic instability should be considered in addition to absolute volume of blood loss<sup>3</sup>. Although it is estimated that less than 5% of patients of patients fulfill the diagnosis of massive hemoptysis<sup>4</sup>, the associated mortality rate ranging from 7% to 30%<sup>4</sup> to as high as 58%<sup>2</sup> illustrates the need for urgent evaluation and management.

## **Pathophysiology**

Blood flow through the lung is accomplished by the low pressure high volume pulmonary arterial circulation and the bronchial artery system, which supplies the structural components of the lung. While the bronchial arterial system has been estimated to represent 1% of pulmonary blood flow<sup>5</sup>, this occurs under systemic pressure, and is responsible for up to 90% of cases with massive hemoptysis<sup>6</sup>.

## **Etiologies**

It must always be carefully considered that the bleeding may come from anatomic sites outside of the lung. Careful history and physical examination or endoscopic evaluation to rule out other sources such as the nasopharyngeal and upper gastrointestinal tract may need to be performed.

The most common causes of hemoptysis has evolved over time. Tuberculosis, lung abscess and bronchiectasis were implicated in earlier reviews<sup>7</sup>. More recent studies suggest pneumonia, lung cancer and bronchiectasis as primary etiologies, although this study included criteria for moderate and massive hemoptysis<sup>8</sup>. Pulmonary arteriovenous malformations, either idiopathic or associated with hereditary hemorrhagic telangiectasia can present with hemoptysis in up to 20% of cases<sup>9</sup>.

With the advent of diagnostic and interventional pulmonary procedures the incidence of iatrogenic massive hemoptysis is increasing. Transbronchial and transthoracic needle biopsies in addition to pulmonary artery rupture from flow directed

pulmonary artery pressure monitoring catheters have been reported<sup>10</sup>. Newer procedures, such as endobronchial ultrasound guided mediastinal lymph node biopsies causing fatal hemoptysis have been documented<sup>11</sup>.

## **Diagnosis and Treatment**

Medical stabilization of hemodynamics and gas exchange is almost always discussed with diagnosis of massive hemoptysis given the rapid clinical deterioration that can be frequently experienced. Basic management includes attempting to identify the affected lung and placing it the dependent position to avoid flooding the unaffected side. Securing a patent airway with a large diameter endotracheal tube to facilitate bronchoscopy, both diagnostic and therapeutic, should be performed if the patient develops respiratory distress or hemodynamic instability. There is some advocacy for placement of double lumen endotracheal tubes for preservation of function in the non-bleeding lung. This requires high proficiency in placement by the operator, and precludes diagnostic and interventional bronchoscopy due to insufficient lumen diameter that also reduces the ability to remove airway obstructing clots<sup>12</sup>.

Bronchoscopy is important in potentially identifying the source of bleeding and may contribute to management in preservation of unaffected lung strategies with balloon tamponade or selective placement of single lumen endotracheal tubes into the mainstem bronchus of the non-bleeding lung<sup>13</sup>.

Computed tomography (CT) is extremely useful in helping localization of bleeding and diagnosing the cause not found on bronchoscopy<sup>14</sup>. Disadvantages are transporting the patient away from the ICU and also potentially misidentification of intraluminal clot as tumor.

Arteriography is performed if there is persistent bleeding despite bronchoscopic maneuvers and medical management, such as correction of coagulopathy or platelet dysfunction. Embolization can be performed in patients deemed too high risk for surgery<sup>15</sup>.

Thoracic surgery consultation should be obtained early, as surgical intervention may need to proceed rapidly if the above interventions are unsuccessful. Surgical mortality for actively bleeding patients was approximately 20%, although these represent older studies and there is a paucity of new data<sup>7</sup>.

### Summary

Massive hemoptysis is a visual and rapidly evolving medical emergency. Our case, although a less common etiology, illustrates the need for rapid control of airway and optimizing gas exchange with concurrent diagnostic procedures to identify the source of bleeding. The selection of interventional bronchoscopy techniques, arterial embolization and surgical resection are often based on the availability of experienced operators in the community hospital practice setting.

### REFERENCES

1. **Knott-Craig CJ, Oostuizen JG, Rossouw G, Joubert JR, Barnard PM.** Management and prognosis of massive hemoptysis. Recent experience with 120 patients. *J Thorac Cardiovasc Surg.* 1993 Mar;105(3):394-7. PubMed PMID: 8445918.
2. **Corey R, Hla KM.** Major and massive hemoptysis: reassessment of conservative management. *Am J Med Sci.* 1987 Nov;294(5):301-9. PubMed PMID: 3425580.
3. **Ibrahim WH.** Massive haemoptysis: the definition should be revised. *Eur Respir J.* 2008 Oct;32(4):1131-2. doi: 10.1183/09031936.00080108. PubMed PMID: 18827169.
4. **Lordan JL, Gascoigne A, Corris PA.** The pulmonary physician in critical care \* Illustrative case 7: Assessment and management of massive haemoptysis. *Thorax.* 2003 Sep;58(9):814-9. Review. PubMed PMID: 12947147; PubMed Central PMCID:PMC1746797.
5. **Yoon W, Kim JK, Kim YH, Chung TW, Kang HK.** Bronchial and nonbronchial systemic artery embolization for life-threatening hemoptysis: a comprehensive review. *Radiographics.* 2002 Nov-Dec;22(6):1395-409. Review. PubMed PMID: 12432111.
6. **Thompson AB, Teschler H, Rennard SI.** Pathogenesis, evaluation, and therapy for massive hemoptysis. *Clin Chest Med.* 1992 Mar;13(1):69-82. Review. PubMed PMID:1582150.
7. **Cahill BC, Ingbar DH.** Massive hemoptysis. Assessment and management. *Clin Chest Med.* 1994 Mar;15(1):147-67. Review. PubMed PMID: 8200191.
8. **Hirshberg B, Biran I, Glazer M, Kramer MR.** Hemoptysis: etiology, evaluation, and outcome in a tertiary referral hospital. *Chest.* 1997 Aug;112(2):440-4. PubMed PMID: 9266882.
9. **Wong HH, Chan RP, Klatt R, Faughnan ME.** Idiopathic pulmonary arteriovenous malformations: clinical and imaging characteristics. *Eur Respir J.* 2011 Aug;38(2):368-75. doi: 10.1183/09031936.00075110. Epub 2010 Dec 22. PubMed PMID: 21177836.
10. **Flores R, Sunder S.** Massive hemoptysis. *Hospital Phy.* 2006; pp. 37-43.
11. **Aguilar-Lopez CA, Weir I, Winter S.** Fatal hemoptysis after endobronchial ultrasound guided mediastinal biopsies. *Am J Resp Crit Care Med.* 2013;187:A5809.
12. **Santana-Cabrera L, Arroyo MF, Rodriguez AU, Sanchez-Palacios M.** Double-lumen endobronchial tube in the emergency management of massive hemoptysis. *J Emerg Trauma Shock.* 2010 Jul;3(3):305. doi: 10.4103/0974-2700.66527. PubMed PMID:20930994; PubMed Central PMCID: PMC2938515.
13. **Haponik EF, Fein A, Chin R.** Managing life-threatening hemoptysis: has anything really changed? *Chest.* 2000 Nov;118(5):1431-5. PubMed PMID: 11083697.
14. **Revel MP, Fournier LS, Hennebicque AS, Cuenod CA, Meyer G, Reynaud P, Frija G.** Can CT replace bronchoscopy in the detection of the site and cause of bleeding in patients with large or massive hemoptysis? *AJR Am J Roentgenol.* 2002 Nov;179(5):1217-24. PubMed PMID: 12388502.
15. **Ching K, Medsinghe A, Santos E, Bandi R.** Arterial embolization for massive hemoptysis in patients with chronic lung consolidations. *J Vasc Interven Radiology.* 2013; 24:S112.

Submitted on August 11, 2014